4. Uluslararası Türk Dünyası Multipl Skleroz Kongresi

4. Международный
КОНГРЕЕС СТРАН ТЮРКСКОГО МИРА ПО РАССЕЯННОМУ СКЛЕРОЗУ

06-10 Haziran 2022, Semerkant - Özbekistan

06-10 июня 2022 г., Самарканд - Узбекистан

BİLDİRİLER ve TAM METİNLER KİTABI
ABSTRACTS and FULL TEXTS

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İÇİNDEKİLER / *Индекс*

ÖNSÖZ

Değerli Türk ve Türki Cumhuriyetlerindeki Meslektaşlarımız

Türk ve Türki cumhuriyetlerde multipl skleroz alanında çalışan meslektaşlarımız arasında bilgi birikimini paylaşmak ve ortak çalışma ve araştırmalar ile aramızdaki işbirliğini güçlendirmek için ilk kez 2019 yılında Ankara'da düzenlediğimiz Uluslararası Türk Dünyası Multipl Skleroz Kongresi'nin hem ülkemizdeki hem de dost ve kardeş ülkelerdeki paydaşlarımız tarafından çok ilgi gösterilmesi ve önemli bir açığı kapatması nedeniyle daha sonraki yıllarda da geleneksel olarak her yıl bir ülkede yapılması kararı alınmış ve her yıl artan ve binlere yaklaşan katılımcı ile 4'ncüsü 6-10 Haziran 2022'de Özbekistan'da gerçekleştirilecektir.

Özbekistan'daki paydaşlarımızın çalıştıkları akademik kurumlarının bu önemli organizasyona katılmak ve ev sahipliği yapmak istemeleri nedeniyle kongremiz 4 gün Semerkant'ta yapılacaktır. Kongrede klinik ve tanı ile tedaviler tartışılacaktır.

Kongremizde; multipl skleroz, NMO, MOG ilişkili hastalıkların güncel tanısı, tedavisi ve bu alandaki güncel gelişmeler alanında uzman konuşmacılar eşliğinde tartışılacaktır.

Sözel ve poster bildiriler ile ülkelerimizde yapılan çalışmalar sunulacak ve yapılan çalışmalar ile ülkelerimiz arasında güç birliği oluşturularak ortak çalışmalar için planlamalar yapılabilecektir.

Kongre sürecinde verilen aralarda ülkelerimize ait sosyal etkinlikler de olacaktır.

Ülkemizdeki ve Tüm Türk cumhuriyetlerinde çalışan nöroloji uzmanları ve asistanlarımızın geçen yıllarda olduğu gibi bu yıl da kongreye geniş bir katılım göstermesini beklemekteyiz.

6-10 Haziran 2022 tarihinde Özbekistan Semerkant'ta gerçekleştireceğimiz Uluslararası Türk Dünyası Multipl Skleroz Kongresinin hem ülkemiz hem de Türk Dünyasındaki nörologlara önemli katkılar sağlayacağını umuyoruz. Tüm meslektaşlarımızla bu özel etkinlikte birlikte olmaktan onur ve mutluluk duyacağız.

Saygılarımızla,

KONGRE EŞ BAŞKANLARI

Prof Dr. Şeref Demirkaya	Prof. Dr. Hüsnü Efendi	Prof. Dr. Yokhutkhon Madzhidova	Prof. Dr. Aziza Jurabekova

ПРЕДИСЛОВИЕ

Уважаемые коллеги в Турции и тюркских странах!

Международный конгресс стран тюркского мира по рассеянному склерозу, который мы впервые провели в Анкаре в 2019 году с целью обмена знаниями и укрепления нашего сотрудничества совместными исследованиями и исследованиями среди наших коллег, работающих в области рассеянного склероза в Турции и Тюркских странах, была высоко оценена нашими заинтересованными сторонами как в нашей стране, так и в дружественных и братских странах. В связи с проявленным интересом и устранением важного пробела, традиционно было принято решение проводить его ежегодно в одной из стран. Четвертый конгресс пройдет в Узбекистане с 6 по 10 июня 2022 года, с каждым годом увеличивающимся и приближающимся к тысячам количеством участников.

Поскольку академические учреждения наших заинтересованных сторон в Узбекистане готовы принять участие и поддержать эту важную организацию Наш конгресс будет проходить в течение 2 дней в Ташкенте и 2 дней в Самарканде. Клиника и диагностика будут обсуждаться в разделе в Ташкенте, а методы лечения – в разделе в Самарканде.

На нашем конгрессе будут обсуждаться в присутствии экспертов-докладчиков методы современной диагностики и лечение рассеянного склероза, оптического нейромиелита (NMO), заболеваний связанных с гликопротеинами миелиновых олигодендроцитов (MOG).

На конгрессе будут представлены устные и стендовые презентации об исследованиях и работах проводимых в наших странах, по проделанной работе будут составлены планы совместной работы по созданию союза сил между нашими странами.

Во время пеперыров будут проводится социальные мероприятия.

Мы ожидаем, что наши специалисты и ассистенты-неврологи, работающие в нашей стране и во всех тюркских странах, примут широкое участие в конгрессе в этом году, как и в предыдущие годы.

Мы надеемся, что Международный конгресс стран тюркского мира по рассеянному склерозу, который состоится в Ташкенте и Самарканде, в городах Узбекистана с 6 по 10 июня 2022 года, внесет значительный вклад в работу неврологов, как в нашей стране, так и в странах тюркского мира. Для нас будет честью и радостью принять участие в этом особенном мероприятии вместе с нашими коллегамии.

С уважением,

СОПРЕДСЕДАТЕЛИ КОНГРЕССА

Проф. др. Демиркая Шереф	Проф. Др. Эфенди Хюсню	проф. Др. Маджидова Йохутхон	Проф. Др. Джурабекова Азиза

KOMİTELER */ КОМИТЕТ*

Kongre Eş Başkanları
Şeref Demirkaya
Hüsnü Efendi
Yakuthon Madjiyeva
Jurakebova Aziza Tahirovna

Kongre Sekreterleri
Aynur Feyzioğlu
Gencer Genç
Sedat Şen

Kongre Düzenleme Kurulu
Şeref Demirkaya
Hüsnü Efendi
Ceyla İrkeç
Canan Yücesan
Cavit Boz
Yakuthon Madjiyeva
Jurakebova Aziza Tahirovna
Rana Kazim Shiraliyeva
Aynur Feyzioğlu
Egemen İdiman
Saule Turuspekova
Yerkin Smaguuloviç Nurgujaev
Saltanat Kamenov
Kunduz Karbozova
Manija Ganieva
Sıla Usar
Yusuf Tamam

BİLİMSEL PROGRAM / *ВИДЕО ПРЕЗЕНТАЦИЙ*

6 Haziran 2022 Pazartesi	
10:00 - 12:00	**Açılış Töreni** *Yokuthan Madjidova* *Şeref Demirkaya* *Hüsnü Efendi* *Aziza Jurabekova* *Jasur Rizaev*
12:0 - 13:00	**ÖĞLE YEMEĞİ**
13:00 - 14:15	**1. OTURUM**
	Oturum Başkanları: **Hüsnü Efendi, Yokuthan Madjidova**
13:00 - 13:20	Multipl Skleroz'un İmmunpatogenezinde enflamasyon? / **Canan Yücesan**
13:20 - 13:40	Multipl Skleroz'un İmmunpatogenezinde dejenerasyon? / **Ceyla irkeç**
13:40 - 14:00	MS'te genetik ve epigenetik faktörler / **Belgin Koçer**
14:00 - 14:15	Tartışma
14:15 – 15:30	**2. OTURUM**
	Oturum Başkanları: **Ceyla İrkeç, Aziza Jurabekova**
14:15 – 14:35	Multipl Skleroz'da Değiştirilebilir Çevresel Faktörlerin Rolü ve Etkisi **/ Nur Yüceyar**
14:35 – 14:55	Multipl skleroz ortaya çıkışında ve seyrinde D vitamini ne kadar önemli? / **Şeref Demirkaya**
14:55 – 15:15	Multipl Skleroz'un Tanı ve Tedavisindeki Algoritma / **Rena Shiraliyeva**
15:15 – 15:30	Tartışma
15:30 – 15:45	**KAHVE MOLASI**
15:45 – 17:00	**3. OTURUM**
	Oturum Başkanları: **Tahir Kurtuluş Yoldaş, Saule Turuspekova**
15:45 – 16:05	MS tanı kriterleri ve McDonald 2017 kriterlerinin klinikte kullanımı **/ Gülnur Uzuner**
16:05 – 16:25	Multipl Skleroz'un Klinik fenotipleri,MS kliniğinde Tipik ve Atipik Özellikler **Levent Sinan Bir**
16:25 – 16:45	RR MS'den sekonder progresyona geçiş ve hastaların izlemi **/ Cavit Boz**
16:45 – 17:00	Tartışma
17:00 – 17:15	**KAHVE MOLASI**
17:15 – 19:15	**4. OTURUM - Ülkelerimizde MS'in Günümüzde Tanı ve Tedavi İmkanları**
	Oturum Başkanları: **Şeref Demirkaya, Rana Kazim Shiraliyeva**
	Azerbaycan'da MS'in Günümüzde Tanı ve Tedavi İmkanları / **Ayten Mamedbeyli**
	Kazakistan'ın Kuzey ve Güney bölgelerinde multipl sklerozun karşılaştırmalı klinik ve epidemiyolojik özellikleri. **Klara Kanatovna Almahanova**
	KKTC'de MS'in Günümüzde Tanı ve Tedavi İmkanları **/ Sıla Usar**
	Kırgızistan'da MS'in Günümüzde Tanı ve Tedavi İmkanları / **Kunduz Karbazova**
	Özbekistan'da MS'in Günümüzde Tanı ve Tedavi İmkanları / **Yokuthan Madjiova**
	Tajikistan'da MS'in Günümüzde Tanı ve Tedavi İmkanları / **Manija Ganiyeva**
	Türkiye'de MS'in Günümüzde Tanı ve Tedavi İmkanları **/ Erdem Toğrol**

7 Haziran 2022 Salı	
09:00 – 10:15	**5. OTURUM**
	Oturum Başkanları: **Canan Yücesan, Markhamat Yakubova**
09:00 – 09:20	RİS Tanı, İzlem ve Tedavi? **/ Aksel Siva**
09:20 – 09:40	Multipl Skleroz tanısında/ayırıcı tanıda ve izleminde MRG'nin önemi? **/ Rana Karabudak**
09:40 – 10:00	Progressif MS' de MRG belirteçleri nelerdir? **Hüsnü Efendi**
10:00 – 10:15	Tartışma
10:15 – 10:30	**KAHVE MOLASI**
10:30 – 11:45	**6. OTURUM**
	Oturum Başkanları: **Taşkın Duman, Şuhrat Toşmirovic Niyazov**
10:30 – 10:50	MS tedavisinde I. Basamak enjektabl tedaviler ve tedavideki yeri? **/ Alev Leventoğlu**
10:50 – 11:10	MS tedavisinde I. Basamak oral tedaviler (teriflunomid ve dimetilfumarat) ve tedavideki yeri? Atak tedavisi? / **Serhan Sevim**
11:10 – 11:30	MS tedavisinde 2. Basamak oral tedaviler (fingolimod ve kladribin) ve tedavideki yeri? **Semra Mungan**
11:30 – 11:45	Tartışma
11:45 – 13:00	**7. OTURUM**
	Oturum Başkanları: **Nebahat Taşdemir, Gülnara Rahimbeyava**
11:45 – 12:05	MS tedavisinde monoklonal antikorlar tedaviler ve tedavideki yeri? / **Murat Kürtüncü**
12:05 – 12:25	MS tedavisinde BTK inhibitörleri ve faz çalışmaları, tedavide olası yeri? / **Uğur Uygunoğlu**
12:25 – 12:45	Ms'da atak tanımı? Atak Tedavisi; hangi hastaya ne zaman? Nasıl yapılmalıdır? **/ Taşkın Duman**
12:45 – 13:00	Tartışma
13:00 – 14:00	**ÖĞLE YEMEĞİ**
14:00 – 15:15	**8. OTURUM**
	Oturum Başkanları: **Caner Feyzi Demir, Manija Ganiyeva**
14:00 – 14:20	MS tedavi seçimi ve değiştirmedeki genel yaklaşım nedir? / **Ömer Faruk Turan**
14:20 – 14:40	MS tedavisinde eskalasyon veya indüksiyon? **Aslı Tuncer**
14:40 – 15:00	MS in progresif formlarını ne zaman, nasıl ve ne kadar tedavi etmeli? / **Münire Kılınç**
15:00-15:15	Tartışma
15:15 – 15:30	**KAHVE MOLASI**
15:30 – 16:45	**9. OTURUM**
	Oturum Başkanları: **Belgin Koçer, Nazira Asanovna Jarkinbekova**
15:30 – 15:50	MS tedavisinde güvenlik ve risk yönetimi? **Serpil Bulut**
15:50 – 16:10	İmmun rekonstrüksiyon: Multipl Skleroz Tedavisinde Yeni Bir Yaklaşım / **Saule Turuspekova**
16:10 – 16:30	Multipl sklerozun modern tanı ve tedavi ilkeleri. **Erkin Smaguloviç Nurgujayev - L.E. Yesjanova - K.K. Almahanova**
16:30 – 16:45	Tartışma
16:45 – 17:00	**KAHVE MOLASI**

17:00 – 18:15	**10. OTURUM**
	***Oturum Başkanları*: Dürdane Aksoy / Gulnoz Şerzamonovna Davlatmirova**
17:00 – 17:20	Multipl sklerozda uyku bozuklukları ve tedavisi **Semai Bek**
17:20 – 17:40	Multipl sklerozda paroksismal bozukluklar ve tedavisi / **Gülnihal Kutlu Günergin**
17:40 – 18:00	Multipl sklerozda sifinkter ve cinsel işlev bozuklukları ve tedavisi / **Gencer Genç**
18:00 – 18:20	Multipl sklerozlu hastalarda ağrı sendromu **Liliya Bareevna Novikova**
18:20 – 18:30	Tarışma
18:30 – 19:20	**11. OTURUM**
	***Oturum Başkanları*: ..**
18:30 – 18:50	Multipl skleroz tanısında Beyin MR Algoritması **Taisiya Vladimirovna Kulneva**
18:50 – 19:10	Multipl sklerozda Meddulla spinalisteki fokal değişikliklerin ayrıcı tanısında MR algoritmaları **Aleksey Yurieviç Popov**
19:10 – 19:20	Tartışma
8 Haziran 2022 Çarşamba	
09:00 – 10:45	**12. OTURUM: TARTIŞMALI OLGULAR – Bölge Ülkelerinden Gelebilecek Atipik Olguların Tartışılması**
	Tartışmacılar: Ömer Faruk Turan, Cavit Boz, Canan Yücesan, Ceyla İrkeç, Funda Uysal Tan, Ufuk Aluçlu, Tahir Kurtuluş Yoldaş, Münire Kılıç, Aytan Kamal Mamadbayli, Yokuthan Madjidova
10:45 – 11:00	**KAHVE MOLASI**
11:00 – 12:15	**13. OTURUM**
	***Oturum Başkanları:* Belgin Petek Balcı, Yerkin Smaguuloviç Nurgujaev**
11:00 – 11:20	Kadınlarda MS Tedavisi Yönetimi / **Aylin Akçalı**
11:20 – 11:40	MS ve NMO'da gebelik / **Ayşe Altıntaş**
11:40 – 12:00	Nörooptikomyelit spektrumunun hastalıkları: soruna modern bakış **Nazira Asanovna Jarkinbekova**
12:00 – 12:15	Tartışma
12:15 – 13:00	**ÖĞLE YEMEĞİ**
13:00 – 14:15	**14. OTURUM**
	***Oturum Başkanları* Mehmet Tecellioğlu, Aytan Kamal Mamadbayli**
13:00 – 13:20	MS ve Aşılar / **Bilge Piri Çınar**
13:20 – 13:40	Ailesel MS ve Özellikleri / **Sibel Güler**
13:40 – 14:00	Multipl Skleroz ve EBV / **Haluk Gümüş**
14:00 – 14:15	Tartışma
14:15 – 14:30	**KAHVE MOLASI**
14:30 – 16:25	**15. OTURUM**
	***Oturum Başkanları:* Yaşar Altun, Marhamat Mirakramovna Yakubova**
14:30 – 14:50	Covid sürecinde MS Tedavisi / **Aziza Jurabekova**
14:50 – 15:10	COVID-19'u geçiren multipl sklerozlu hastalarda bilişsel işlevlerin özellikleri **Matlyuba Jakhonkulovna Sanoyeva**
15:10 – 15:30	Multipl Skleroz ve Koronavirüs Enfeksiyonu: seyir özellikleri ve komorbidite **Gülnara Rahimbayeva**
15:30 – 15:50	Covid 19 sürecinde multipl skleroz **Roza Baltabayevna Nurjanova - Kosbolovna Demesinova**
15:50 – 16:10	Covid - 19 pandemisinde MS ve NMO'da ve izlem/tedavi yaklaşımı? / **Sedat Şen**
16:10 – 16:25	Tartışma

9 Haziran 2022 Perşembe	
09:00 – 10:15	**16. OTURUM**
	Oturum Başkanları: **Cavit Boz, Matluba Sanayeva**
09:00 – 09:20	NMO ve NMO spektrum hastalıklarında immun patogenez ve tanı kriterleri **Sibel Canbaz Kabay**
09:20 – 09:40	NMO ve NMO spektrum hastalıklarında tedavi yaklaşımı / **Egemen İdiman**
09:40 – 10:00	MOG ilişkili hastalıklar ve tedavisi **Kadriye Ağan**
10:00 – 10:15	Tartışma
10:15 – 10:30	**KAHVE MOLASI**
10:30 – 11:45	**17. OTURUM**
	Oturum Başkanları: **Semra Mungan / Bakhtiyar Gafurov**
10:30 – 10:50	MS'de BOS İncelemesi ve Biyobelirteçler **Murat Terzi**
10:50 – 11:10	Kanda Biyobelirteçler **/ Birsen Can Demirdöğen**
11:10 – 11:30	İmmunite ve Mikrobiyata Regülasyonunda Diyetin Rolü **/ Semir Beyaz**
11:30 – 11:45	Tartışma
11:45 – 12:45	**18. OTURUM**
	Oturum Başkanları: **Ufuk Aluçlu, Aysun Soysal**
	Tartışmacılar: **Mesrure Köseoğlu, Şeyda Figül Gökçe, Özlem Ethemoğlu, Fatih Yetkin, Cihat Uzunköprü, Destan Sena Bünül**
12:45 – 13:30	**ÖĞLE YEMEĞİ**
13:30 – 14:45	**19. OTURUM**
	Oturum Başkanları: **Gençer Genç, Matlyuba Sanoeva**
13:30 – 13:50	Nörolojik Hastalıklarda Intravenöz Immunglobulinin yeri ve kullanım alanları **Abdulkadir Tunç**
13:50 – 14:10	Tacikistan Cumhuriyeti'nde MS'in klinik belirtileri / **Gulnoz Şerzamonovna Davlatmirova**
14:10 – 14:30	Özbekistan koşullarında multipl sklerozun bazı klinik ve epidemiyolojik özellikleri **Marhamat Mirakramovna Yakubova**
14:30 – 14:50	MS'da klinik fenotipler, radyolojik izole sendromdan sekonder progressif MS'a klinik seyir / **Münife Neyal**
14:50 – 15:05	Tartışma
15:05 – 15:20	**KAHVE MOLASI**
15:20 – 16:55	**20. OTURUM (Bölgelerden gelen çalışmalar)**
	Oturum Başkanları: **Murat Terzi, Aynur Feyzioğlu**
15:20 – 15:40	Multipl sklerozlu hastaların tedavisinde ozonlanmış serebralizin kullanımı. **Şuhrat Toşmiroviç Niyazov**
15:40 – 16:00	Longitudinal transvers miyelit: klinik bir vaka **Nikolay Valeryeviç Stepnuk -** **Togjan Jaylıkereevna Mukaşeva**
16:00 – 16:20	Kazakistan'ın kuzey bölgelerindeki İkincil ilerleyici tip multipl skleroz tanısı için MS Pro Tartışma anketini kullanma deneyimi **Laura Yerkenovna Yesjanova**
16:20 – 16:40	Kazakistan Cumhuriyeti Akmola bölgesinde multipl sklerozlu hastalarda natalizumab tedavisine erken geçişin sonuçları / **Makşarip Bamatgereyeviç Martazanov**
16:20 – 16:55	Tartışma

10 Haziran 2022 Cuma	
08:00 – 11:00	**21. OTURUM (Sözel Oturum)**
	Oturum Başkanları: **Serpil Bulut, Saltanat Kamenov, Funda Uysal Tan**
11:00 – 11:30	***KAHVE MOLASI***
11:30 – 12:30	**22. OTURUM (Poster Oturumu)**
	Oturum Başkanları: **Musa Temel, Antiqa Mammadova, Alev Leventoglu**
12:30 – 13:30	**ÖĞLE YEMEĞİ**
13:30 – 14:30	**23. OTURUM (Kongrenin Genel Değerlendirmesi ve Gelecek Kongre için planlamalar)**
	Şeref Demirkaya Hüsnü Efendi Yakuthon Madjiyeva Jurakebova Aziza Tahirovna Rana Kazim Shiraliyeva Saltanat Kamenov Kunduz Karbozova Manija Ganieva Sıla Usar Saule Turuspekova Aynur Feyzioğlu
14:30 - 14:45	**KAPANIŞ & 2023 BAKÜ KONGRESİNE DAVET OTURUMU**
	Konuşmacılar: **Yakuthon Madjiyeva, Rana Kazim Shiraliyeva**

POSTERLER / *ПОСТЕРЫ*

PS-01	Transvers Miyelit Taklitçi Lezyonlar Esra Eruyar, Ceyla İrkeç
PS-02	Tümefaktif Multipl Skleroz Olgularinda Tedavi Yaklaşimi : Olgu Serisi Cansu Kostakoğlu, Cemre Güçlü, Fatma Seda Bingöl, Berna Arli, Z. Neşe Öztekin, Semra Ö. Mungan
PS-03	Nummular Headache associated with Multiple Sclerosis Ceyla İrkeç, Esra Eruyar, Doğa Vurallı

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4. Международный
КОНГРЕЕС СТРАН ТЮРКСКОГО МИРА ПО РАССЕЯННОМУ СКЛЕРОЗУ

06-10 Haziran 2022, Semerkant - Özbekistan

06-10 июня 2022 г., Самарканд - Узбекистан

SÖZLÜ BİLDİRİLER / УСТНЫЕ ПРЕЗЕНТАЦИИ

SS-01 / 1892

MULTİPLE SKLEROZ HASTALARININ TESTMYBRAIN DİJİTAL NÖROPSİKOLOJİK TEST BATARYASI PERFORMANSLARI

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Amaç: Pandemi koşullarında deneyimlenen hastaneye ulaşma zorluklarının bazı durumlarda dijital değerlendirmeler yardımıyla aşılabileceği düşünülmektedir. McLean Hastanesi ve Harvard Üniversitesi Tıp Fakültesi Beyin ve Kognitif Sağlık Teknolojileri Laboratuvarı tarafından geliştirilen TestMyBrain (TMB), bir dijital nöropsikolojik test bataryasıdır. Bu çalışmada, Multiple Skleroz (MS) hastalarının kognitif işlevlerinin bilgisayar kullanılarak TMB Dijital Nöropsikolojik Test Bataryası ile değerlendirilmesi amaçlanmıştır.

Gereç ve Yöntem: Araştırmamız Ocak 2021-Mayıs 2021 tarihlerinde pandemi sırasında gerçekleştirilmiştir. İstanbul Haseki Eğitim ve Araştırma Hastanesi Nöroloji Kliniği MS polikliniğinde takip ve tedavi gören, 18-55 yaşları arasında 40 MS hastası (24 kadın, 16 erkek) ve 40 sağlıklı birey (24 kadın, 16 erkek) araştırmaya dahil edilmiştir. Tüm katılımcılara Standardize Mini Mental Test (SMMT), Beck Depresyon Ölçeği (BDÖ) ve TMB'den seçilen beş alt test uygulanmıştır. SMMT'den 24 ve üzeri puan alan ve BDÖ'den 10 ve altı puan alan bireyler çalışmaya dahil edilmiştir.

Bulgular: Gruplararasında cinsiyet, yaş ve eğitim açısından anlamlı bir fark bulunmamıştır (sırasıyla p = 1.000, p = .655, p = .712). TMB İleri Doğru Sayı Menzili Testi ve TMB Geriye Doğru Sayı Menzili Testinde sağlıklı kontroller, MS grubundan anlamlı olarak daha yüksek puan almıştır (p = .025, p = .023). TMB Seçici Tepki Zamanı Testinde tepki zamanı sağlıklı kontrollerde MS grubuna göre anlamlı olarak daha kısa olmakla birlikte (p < .001), doğruluk puanı açısından gruplararası anlamlı bir fark bulunamamıştır (p = .424). TMB Görsel Çağrışım Çiftleri Testi, TMB Matris Akıl Yürütme Testi ve TMB Rakam Sembol Eşleştirme Testi puanları sağlıklı kontrollerde MS grubuna göre anlamlı olarak daha yüksektir (tümü p <.001).

Sonuç: TMB Dijital Nöropsikolojik Test Bataryası ile MS hastalarının kısa süreli bellek, dikkat, çalışma belleği, görsel bellek, epizodik bellek, algısal muhakeme ve prosesleme hızı gibi kognitif işlevlerinde bozulma saptanmıştır. Çalışmamız, TMB Dijital Nöropsikolojik Test Bataryasının MS hastalarında kognitif bozulmaları saptayabildiğini ve MS hastalarında kullanılabileceğini düşündürmektedir. Ayrıca bu çalışma, TMB'nin MS hastalarında geçerlilik çalışmaları için bir temel sunmaktadır.

Anahtar Sözcükler: Dijital Nöropsikoloji, Multiple Skleroz (MS), Kognitif İşlevler

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PRİMER SANTRAL SİNİR SİSTEMİ VASKÜLİTİNDE AYIRICI TANI: OLGU SUNUMU

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GİRİŞ: Primer santral sinir sistemi vasküliti (PSSV), yüksek morbidite ve mortaliteye sahip nadir görülen inflamatuar bir hastalıktır. Başlangıç kliniği heterojendir. Kesin tanı için çoğu hastada biopsy gerekmektedir. Klinik ve nörogörüntüleme bulgularındaki çeşitlilik nedeniyle birçok hastalığın ayırıcı tanısında yer almaktadır. İlk başvurusu nöbet ve baş ağrısı olan ve beyin biyopsi sonucu ile tanı konulan bir olgu temel alınarak PSSV ayırıcı tanısı sunulmaktadır.

OLGU: 39 yaşında erkek hasta sol tarafta güçsüzlük ve gövde ataksisi şikayetleri ile Haziran 2021'de SANKO Üniversitesi Nöroloji polikliniğine başvurdu. Hikayesinde Ocak 2021'de şiddetli başağrısı ve nöbet ile başka bir hastaneye başvurduğu serebral venöz enfarkt tanısıyla varfarin tedavisi başlandığı öğrenildi. Son 6 aylık takibinde klinik ve radyolojik bulgularının kötüleştiği, varfarin kullanmaya devam ettiği öğrenildi. Yatış günü yapılan nörolojik muayenede sol santral fasial paralizi, hafif sol hemiparezi (4/5) ve solda DTR artışı saptandı. Beyin MRG'de multifokal vazojenik ödem ve hemorajik alanlar içeren, periferal kontrastlanan birden fazla lezyon izlendi. JCV PCR, vaskülit tetkikleri, periferik yayma normal olarak raporlandı. Pulse prednol tedavisi başlandı. Hastanın klinik ve görüntüleme bulguları kötüleştiği için siklofosfamid tedavisi eklendi, ancak fayda sağlamadı. Beyin biopsisi patolojik değerlendirme sonucu vaskülit ile uyumlu geldi.

SONUÇ: PSSV heterojen klinik bulguları ve atipik görüntüleme özellikleri olması sebebiyle tanısı halen zor olan

hastalıklardandır. Tedavisi de tanısı kadar zor olabilir. Özellikle gecikilen olgularda etkin immünsüpresif tedaviye rağmen hastalığın kötü seyri durdurulamayabilir.

DIFFERENTIAL DIAGNOSIS IN PRIMARY CENTRAL NERVOUS SYSTEM VASCULITIS: CASE REPORT

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INTRODUCTION: Primary central nervous system vasculitis (PCNSV) is a rare inflammatory disease with high morbidity and mortality risk. The initial clinical presentation is heterogeneous. Biopsy is required in most patients for definitive diagnosis. Due to the diversity in clinical and neuroimaging findings, it is included in the differential diagnosis of a number of diseases. The differential diagnosis of PCNSV is presented here, on the base of a case whose initial presentation was seizure and headache and diagnosis could be done due to interpretation of the brain biopsy findings.

CASE: A 39-year-old male patient addmitted to SANKO University neurology outpatient clinic in June 2021 with complaints of truncal ataxia and weakness on the left side. He had a seizure and severe headache attacks in January 2021, and warfarin was started with the diagnosis of cerebral venous infarction in an other hospital. During the follow-up in the previous 6 months, his clinical and radiological finding got worse. Investigations for brain tumor were done with negative results and he went on using warfarin. On the admission day, left central facial paralysis, mild left hemiparesis (4/5), and increased DTR on the left side were noted in the neurological examination. Brain MRI revealed multiple peripherally enhanced lesions with multifocal vasogenic edema and hemorrhagic areas. JCV PCR, blood tests for vasculitis, peripheral blood smear were reported as normal. Prednisolon treatment was started. However, clinical and imaging findings deteriorated, cyclophosphamide was added without any benefits. A brain biopsy was performed, that interpreted as pathological evaluation was compatible with vasculitis.

CONCLUSION: PCNSV is one of the diseases whose diagnosis is still difficult due to heterogeneous clinical findings and atypical imaging features. Treatment can be as difficult as diagnosis. Especially in delayed cases, the poor course of the disease may not be interrupted despite effective immunosuppressive therapy.

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MULTİPL SKLEROZ TEDAVİSİNDE KLADRİBİN: TEK MERKEZ TECRÜBESİ

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AMAÇ: Kladribin adeonozin deaminazı inhibe ederek etki eden sentetik bir pürin analoğudur. Yüksek hastalık aktivitesi gösteren, ataklarla seyreden multipl skleroz (MS) hastalarında kullanım onayı bulunmaktadır. Bu çalışmada kliniğimizde kladribin tedavisi kullanan MS hastalarımızın etkinlik ve güvenlik profillerinin değerlendirilmesi amaçlanmıştır.

GEREÇ VE YÖNTEM: Erciyes Üniversitesi Tıp Fakültesi Multiple Skleroz ve Demiyelinizan Hastalıklar Polikliniği'nde Kladribin tedavisi başlanan 30 hastanın yaş, cinsiyet, hastalık süresi, önceki tedavileri, tedavi değişiklik nedeni, tedavi öncesi ve sonrası EDSS skorları kaydedilmiştir.

BULGULAR: Çalışmaya dahil edilen 30 hastanın 20'si kadın, 10'u erkek cinsiyetteydi. Yaş ortalamaları 32,03±7,9, ortalama hastalık süresi 6,86 ise yıldı. Hastaların tedavi öncesi ortalama EDSS 1,83±1,3 (0- 5) olup, tedavi sonrası anlamlı değişiklik saptanmadı. Hastalarımızın tedavi değişiklik nedeni en sık önceki tedaviye yanıtsızlık (%60) olup bunu önceki ilacın yan etkisi (%27) izlemekteydi. %10 hastada JCV pozitifliği, %3 hastada ise tedavi uyumsuzluğu dolayısıyla önceki tedavilerden kladribine geçiş yapıldı. Kladribin öncesi 12 (%40) hasta fingolimod, 8 (%26,6) hasta natalizumab, 5 (%16,6) hasta dimetilfumarat, 3 (%10) hasta okrelizumab, 1 (%3,3) hasta alemtuzumab, 1 (%3,3) hasta interferon kullanıyordu. Tedavi sırasında hiçbir hastada alerjik reaksiyon, dirençli enfeksiyon ve malignite gelişmedi.

SONUÇ: MS hastalarında kladribin tedavisi iyi tolere edilmektedir. Hastalık aktivitesi yüksek olan MS hastalarında erken ve etkin tedavi seçenekleri ile takip önemlidir. Uzun dönem etkinlik için çok merkezli gerçek yaşam verilerine ihtiyaç vardır.

CLADRIBINE FOR THE TREATMENT OF MULTIPLE SCLEROSIS: A SİNGLE-CENTER EXPERIENCE

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OBJECTIVE: Cladribine is a synthetic purine analog that acts by inhibiting adeonosine deaminase. It is approved for use in patients with multiple sclerosis (MS), which has a high disease activity and is accompanied by attacks. In this study, it was aimed to evaluate the efficacy and safety profiles of MS patients using cladribine therapy in our clinic.

MATERIALS AND METHODS: The age, sex, duration of illness, previous treatments, cause of treatment change, pre- and post-treatment EDSS scores of 30 patients who started Cladribine treatment at Erciyes University Faculty of Medicine Multiple Sclerosis and Demyelinating Diseases Outpatient Clinic were recorded.

RESULTS: Of the 30 patients included in the study, 20 were female and 10 were male. The mean age was 32.03±7.9 years and the mean disease duration was 6.86 years. The mean EDSS of the patients was 1.83±1.3 (0-5) before treatment and no significant changes were detected after treatment. The most common cause of treatment changes in our patients was unresponsiveness to previous treatment (60%), followed by a side effect of the previous drug (27%). JCV positivity was found in 10% of the patients and cladribine was transferred from previous treatments due to treatment incompatibility in 3% of the patients. 12 (40%) patients were using fingolimod before cladribine, 8 (26.6%) patients were using natalizumab, 5 (16.6%) patients were using dimethylfumarate, 3 (10%) patients were using okrelizumab, 1 (3.3%) patient was using alemtuzumab, 1 (3.3%) patient was using interferon. During treatment, no patients developed allergic reactions, resistant infections and malignancies.

CONCLUSION: Cladribine therapy is well tolerated in MS patients. It is important to follow up with early and effective treatment options in MS patients with high disease activity. For long-term activity, multicenter real-life data is needed.

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YUTMA SORUNU OLAN VE OLMAYAN MULTİPL SKLEROZ HASTALARININ SİYALORE PROBLEMLERİNİN VE YAŞAM KALİTELERİNİN İNCELENMESİ

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Giriş: Multipl sklerozun (MS) seyrinde hastalığın bir parçası olduğu fark edilemeyen çeşitli derecelerde yutma sorunları eşlik edebilir. Bu sorunların hastaların yaşam kalitesini ne derecede etkilediği ve siyalore ile ilişkisi net olarak bilinmemektedir. Biz bu çalışmamızda genellikle göz ardı edilen yutma sorunlarının MS hastalarındaki yaşam kalitesine etkisini ve siyalore ile ilişkisini araştırmayı amaçladık.

Metod: Çalışmaya 56 MS hastası dahil edilmiştir. Hastaların yutma taraması Eating Assessment Tool (EAT-10) aracı ile yapılmıştır. Bu tarama aracından 3 ve üzeri puan alanlar yutma güçlüğü açısından riskli olarak belirlenmiştir. Katılımcıların yaşam kaliteleri EuroQol-5 Dimension Questionnaire (EQ-5D) ile değerlendirilirken siyalore problemleri Siyalore Klinik Ölçeği (SCS-TR) ve Salya Kontrol Problemi Şiddet ve Sıklık Skalası (DSFS) ile değerlendirilmiştir.

Bulgular: Çalışmaya alınan 56 hastanın 32'si kadın 24'ü erkekti. Hastalar yutma sorunu olan (21 hasta) ve olmayan (35 hasta) şeklinde iki gruba ayrıldı. Yutma sorunu olan MS hastalarının EQ-5D Hareket, EQ-5D Özbakım, EQ-5D Aktivite, EQ-5D Ağrı/rahatsızlık, EQ-5D Anksiyete skorları istatistiksel olarak anlamlı derecede yaşam kalitesinin daha fazla bozulduğunu gösterecek şekilde daha yüksekti. EQ-5D Visual Analog Scale (VAS) yutma sorunu olmayan hastaların skoru daha yüksekti (74,91±21,57 vs 49,04±18,27). Yutma sorunu olan MS hastaların siyaloreyi yansıtan SCS-TR toplam skorları anlamlı derecede daha yüksekti (1,34±2,50 vs 5,14±4,59). Ayrıca bir diğer siyalore ölçeği olan DSFS1 ve DSFS2 skoru yutma sorunu olan MS hastalarında olmayanlara göre anlamlı derecede daha yüksek bulunmuştur (sırasıyla 1,76±0,70 vs 1,14±0,35; 1,85±1,10 vs 1,22±0,42).

Sonuç: Bildiğimiz kadarıyla çalışmamız MS hastalarındaki yutma bozukluğu ile yaşam kalitesi ve siyalore parametreleri arasındaki ilişkiyi inceleyen ilk çalışmadır. Yutma sorunu olan MS hastaları olmayanlara göre daha şiddetli siyalore yaşamakta ve yaşam kalitesinde daha belirgin bozulma göstermektedir. MS'te siyaloreye artan tükürük üretiminden ziyade zayıflamış orofasiyal kaslar ve disfaji neden oluyor gibi gözükmektedir. MS hastalarındaki disfajinin ve siyalorenin doğal seyrini ve altta yatan patofizyolojisini daha iyi anlamak için ileri çalışmaların yapılması gerekmektedir.

Anahtar Kelimeler: Multipl skleroz, disfaji, siyalore, yaşam kalitesi

COMPARISON OF SIALORRHEA PROBLEMS AND QUALITY OF LIFE IN MULTIPLE SCLEROSIS PATIENTS WITH AND WITHOUT SWALLOWING PROBLEMS

Background: Various degrees of swallowing problems may accompany the course of multiple sclerosis (MS), which may not be recognized as a part of the disease. The extent to which these problems affect the quality of life of patients and their relationship with sialorrhea is not clearly known. In this study, we aimed to investigate the effect of swallowing problems, which are often overlooked, on quality of life in MS patients and its relationship with sialorrhea.

Methods: 56 MS patients were included in the study. Swallowing screening of the patients was performed with the Eating Assessment Tool (EAT-10). Those who score 3 and above on this screening tool are determined to be at risk in terms of swallowing difficulties. While the quality of life of the participants was evaluated with the EuroQol-5 Dimension Questionnaire (EQ-5D), sialorrhea problems were evaluated with the Sialorrhea Clinical Scale (SCS-TR) and the Drooling Severity and Frequency Scale (DSFS).

Results: Of the 56 patients included in the study, 32 were female and 24 were male. The patients were divided into two groups as those with swallowing problems (21 patients) and those without (35 patients). EQ-5D Movement, EQ-5D Self-Care, EQ-5D Usual activity, EQ-5D Pain/discomfort, EQ-5D Anxiety/depression scores of MS patients with swallowing problems were statistically significantly higher, indicating that the quality of life was more impaired. EQ-5D Visual Analog Scale (VAS) patients without swallowing problems had a higher score (74.91±21.57 vs 49.04±18.27). SCS-TR total scores reflecting sialorrhea were significantly higher in MS patients with swallowing problems (1.34±2.50 vs 5.14±4.59). In addition, DSFS1 and DSFS2 scores, which are another sialorrhea scales, were found to be significantly higher in MS patients with swallowing problems compared to those without (1.76±0.70 vs 1.14±0.35; 1.85±1.10 vs 1.22±0.42; respectively).

Conclusion: To the best of our knowledge, our study is the first to examine the relationship between swallowing disorders and quality of life and sialorrhea parameters in MS patients. MS patients with swallowing problems experience more severe sialorrhea and show a more significant deterioration in their quality of life than those without. In MS, sialorrhea appears to be caused by weakened orofacial muscles and dysphagia rather than increased saliva production. Further studies are needed to better understand the natural history and underlying pathophysiology of dysphagia and sialorrhea in MS patients.

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INVESTIGATION OF THE RELATIONSHIP BETWEEN SMOKING AND CADMIUM IN MULTIPLE SCLEROSIS

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Background: Multiple sclerosis (MS) is among the most prevalent neurological disorders among the young adults. Environmental factors are considered to play a role in MS. Metals are important for homeostatic regulation and dysregulated metal homeostasis causes various diseases including neurodegeneration. Smoking is common among people living in Turkey. Cadmium (Cd) is commonly found in the environment and it is found toxic for human health. Reports show the etiology of various diseases related with metals. Studies investigating relationships between MS, and Cadmium are limited. We, therefore, investigated the possible relationship of cadmium and smoking in MS patients.

Method: Fifty subjects including 20 healthy subjects and 30 MS patients were included in this study. Blood Cd levels and demography of the patients were determined.

Results: Cd levels were found higher in the control subjects, Cd levels were lower in smoking subjects in MS group ($p < 0.05$).

Conclusion: Our results did not show a direct relationship of cadmium and smoking in MS. Randomized, double-blinded, multi-centred, clinical studies with larger cohorts might help to investigate the relevance of cadmium and other metals in MS.

Keywords: Cadmium, Heavy metals, Smoking, Multiple Sclerosis

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Background

While Multiple Sclerosis (MS) is a common neurological disease of the young population, it is still a mystery to understand the causes of the disease (1). Especially after the industrial revolution, the dispersion of metals pollutants like cadmium (Cd) in the environment has been increased (2).

It is more common in patients age between 20 to 50. There are different theories of considerations based on genetic and environmental factors that might be related to the disease development and progression. There are now effective diagnostic tools for the diagnosis of MS but the therapy is a challenge (3).

MS might have many risk factors such as genetics, autoimmunity, environmental factors or immune system activity. There estimated to be around 2 million people with MS globally. And four main types are clinically isolated syndrome (CIS), relapsing remitting MS (RRMS), secondary progressive MS (SPMS) and primary progressive MS (PPMS) as described and widely accepted.

There are two different aspects of the disease; neuroinflamation and neurodegeneration there is an abnormal activation of immune system against CNS antigens and autoreactive CNS-an-

tigen(s)-directed T lymphocytes (CD4+, CD8+) play significant roles in the development of CNS demyelinating lesions. And loss of myelin/oligodendrocyte complex as well as neurodegeneration take place and the disease might be diagnosed at a level when clinical symptoms are apparent.

The diagnosis can be done using Mc Donald Criteria in which laboratory tests and magnetic Resonance Imaging is needed.

Various studies showed a relation with smoking and MS but the relation of heavy metals and metalloids in the environment such as cadmium are to be investigated in our region and patient group.

Heavy metals are the metallic or the semi-metallic elements that occur naturally in the environment and have a density higher than water (4).

Although the toxicity and the heaviness of a metal are indicated to be interrelated, heavy metals are also toxic at low levels (5, 6). Besides cadmium (Cd) is not utilized in any physiological processes (7).

Cd is utilized in Ni-Cd batteries (8), as pigment in the paints (9), for electroplating (10) and polyvinyl chloride plastics (11).

Cd is found in the food stuffs as the Cd is accumulated in soil and water after releasing by several factors including natural processes, mining, refining and product manufacturing, fertilizers, manure and sludge, and is transferred to the humans through the plant and animal foods (12).

Smokers had around 4 to 5 times higher Cd levels in their blood when compared to non-smokers (13). However, blood Cd levels in smokers are indicated to be altered due to lifestyle including dietary habits (14).

In drinking water World Health Organization (WHO) guidelines accept maximum 3 μg/L Cd (15). Cd may affect both central nervous system and peripheral nervous system(16). Clinical finding may include fatigue, headache, muscle weakness, dizziness, syncope (17-19), There are decreased motor speed, attention and memory vision (20). Besides patients can show learning disabilities (21). Patients may have peripheral polyneuropathy that is caused by Cd (22, 23).

Various studies have conflicting results for increased Parkinson Disease risk and Cd exposure (24). Plasma (25) and liver (26) Cd levels were found significantly higher in the Alzheimer disease (27)

Besides a relationship between the Cd and Amyotrophic lateral sclerosis (ALS) were determined (28).

Our region (Kocaeli) is an industrial zone in Turkey and some toxicants like cadmium (Cd) might be found in the nature. The unwanted neurological effects of such toxicants might have an impact on MS risk as well.

Because there was not such a study in our region we wanted to investigate the possible relationship between smoking and Cadmium (Cd) in Multiple Sclerosis (MS) in MS patients in Kocaeli Turkey.

Materials and Methods

20 healthy individuals as a control group and 30 patients with relapsing remitting multiple sclerosis (RRMS) are enrolled to our study. All subjects were aged between18 to 55. The study was approved by the ethical committee of Kocaeli University. All MS patients were under regular control of neurology clinic at outpatient clinic between July-November 2018. 20 healthy controls all agreed to be included to the study among patient companions and hospital staff are enrolled. They had a similar characteristics of age and gender with the patient group. All patients and controls signed the informed consent forms prior to the study.

All 30 patients were RRMS and followed by outpatient clinic in Kocaeli university hospital. They did not experience any attack during the last 30 days prior to the enrollment to the study. Detailed history of the patients were taken and no patients were included to the study if they were working in metal industry or with a history of systemic, metabolic disease or any diagnosed malignancy.

We used 5 ml ethylenediaminetetraacetic acid (EDTA) tubes of BD diagnostics (BD Vacutainer® Plus Plastic K_2EDTA Tubes; 367899) for collecting blood samples from all 30 patients and 20 controls. Then tubes were centrifuged (4,400 RPM for 7-10 mins) and pellet is taken and adding 0.9% NaCl to the tube that was then rotated gently to mix and then re centrifuged taking the same steps two more times. Finally the pellet were added with 0.9% NaCl and taking only 1 ml of the final suspension, it is transferred to a clean tube and mixed with 4 mL cold double-distilled water. All tubes vortexed and the erythrocytes were then lysed. The lysates in tubes were then stored at – 80°C for Cd determination.

A Thermo Scientific™ X SERIES 2, inductively coupled plasma – mass spectrometer (ICP-MS) is used to analyze Cd levels. Forward power = 1450 watts, nebulizer flow = 0.9 mL/min, auxiliary gas flow = 0.8 mL/min, cool gas flow = 13 mL/min, collision cell technology gas flow = 0.6 mL/min. were the parameters for running the samples on the device. Intensity counts per second ICPS was the measurement units of each standard and analyte. Cd levels were determined according to the standard curves (0 – 100 ppb).

Statistical Analysis

GraphPad Prism® 7.0 (GraphPad Software Inc., USA) was used to conduct statistical analyses. All findings were specified as mean ± standard deviation (S.D.).

Standard curve was found by linear regression of optical density (O.D.) values of respective concentrations of standards. Shapiro-Wilk normality test was used for determining the data distribution.

Normally distributed data was analyzed by using Unpaired two-tailed Student's t-test was used for analyzing the normally distributed data while Mann-Whitney U test was used for analyzing non-normally distributed data. Pearson's correlation coefficient test were used for Correlations between parameters.

Chi-square test determined the relation between MS risk and smoking status. Relative risk was and 95% confidence interval (95% CI) was investigated by using Koopman asymptotic score. A p value lower than 0.05 was accepted as statistically significant.

Results

The Study Demographics

There were 50 subjects of which 30 were MS patients (10 male, 20 female) that fits the natural sex distribution of MS patients in general. And 20 controls in the same ratio. Mean age was 33.80 ± 9.64 in male MS group, and it was 28.71 ± 3.77 in male control group. In the female MS group, Mean age was 36.4 ± 10.75 and the female control group mean age was 29.77 ± 4.09 .

	MS	Control
Male	**10**	**7**
	Mean Age 33.80 ± 9.64	Mean Age 28.71 ± 3.77
Female	**20**	**13**
	Mean Age 36.4 ± 10.75	Mean Age 29.77 ± 4.09

Table:1 The Study Demographic Characteristics

Blood Cd Levels

Mean blood Cd levels were significantly lower in MS patients (0.682 ± 0.803 ppb) than control group (1.337 ± 2.839); p<0.0001 .Five MS patients are excluded as they had negative values.

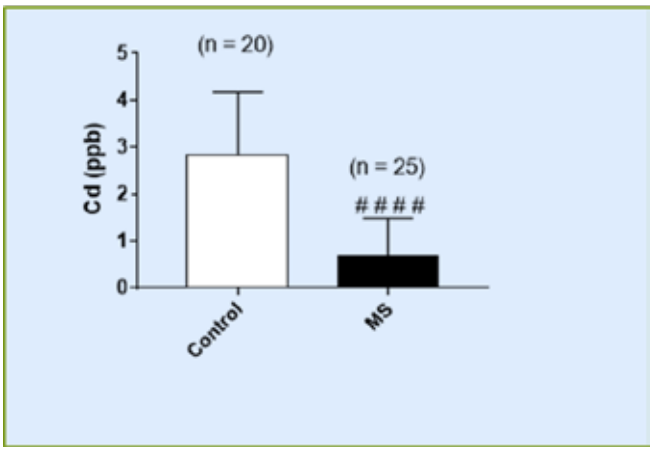

Figure 1. Blood Cd levels of control subjects and MS patients.

(Data is presented as mean ± S.D. Statistical analysis: Mann-Whitney U test. # # # #p < 0.0001).

As for sex values, Blood Cd levels were significantly higher in both male and female control groups. Male controls (2.21± 0.464) than male MS patients (0.3490.117±) ; p<0.0001 and female control group (3.178± 1.5389 than female MS patients (0.811± 0.918 ppb); p<0.0001 . Blood Cd levels of 3 male MS patients and 2 female MS patients could not be measured.

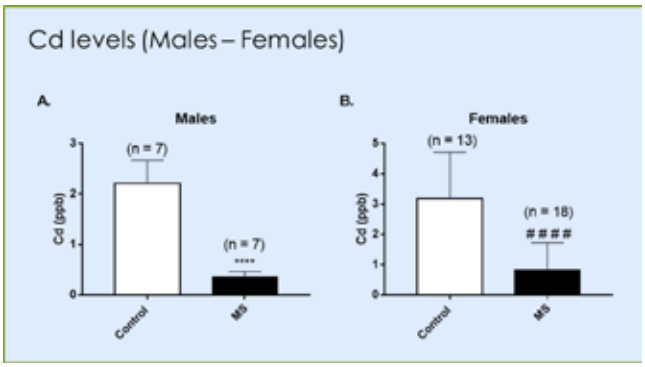

Figure 2. Blood Cd levels in control subjects and MS patients. Data include (A) male and (B) female subjects. (Data is presented as mean ± S.D. Statistical analysis: Unpaired Student's t-test, ****p < 0.0001; Mann-Whitney U test, # # # #p < 0.0001).

Blood Cd Levels and Smoking

Smoking group blood Cd (1.413 ± 1.943 ppb) did not differ from non-smoker group (1.754 ± 1.274 ppb; p > 0.05 figure A). In the same way, Smoking group of control group (3.548 ± 2.155 ppb) did not differ from non-smokers of control group (2.603 ± 0.927 ppb; p > 0.05 figure B). Somehow Blood Cd levels of smokers in MS group (0.3454 ± 0.113 ppb) were significantly lower than non-smokers in MS group (0.906 ± 0.981 ppb; p < 0.05 figure C). Blood Cd levels of two smokers MS patients and 3 non-smokers MS patients could not be determined.

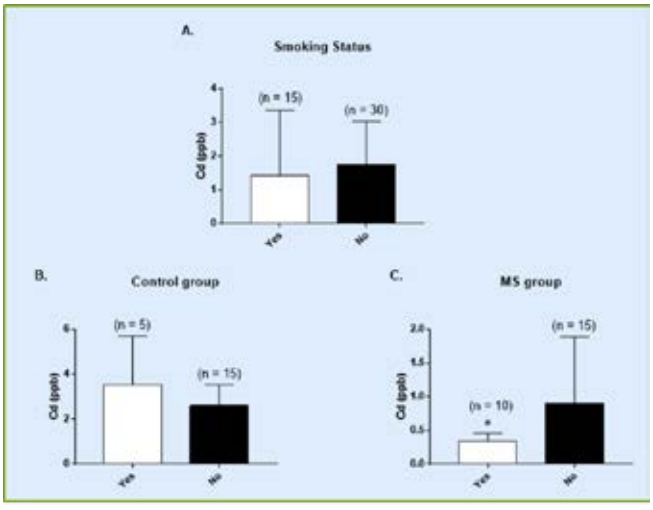

Figure 3. Blood Cd levels in smoker and non-smoker subjects.

Data include (A) both healthy individuals and MS patients, (B) only control subjects and (C) only MS patients. (Data is presented as mean ± S.D. Statistical analysis: Mann-Whitney U test for panels A and C; Unpaired Student's t-test for panel C, $p > 0.05$ for panels A and B and #$p < 0.05$ for panel C).

Relation Between Smoking and MS

	Smoking	Non-smoking
MS patients	12	18
Control	5	15

Table 2 : Relation between smoking and MS

There was not a significant association between smoking status and MS incidence (Chi-square test, $p = 0.2727$). But there was a relative risk associated with smoking 1.294 (95% CI = 0.7961 - 1.999).

Discussion

In our study MS patiens had lower Cd blood concentrations. Cd levels have controversial results in different studies (31, 32). When it was deeply investigated it was seen that Cd also accumulated in different tissues in body. But as there was no exact method to prove tissue concentrations for Cd, we could not speculate on that.

Gadolinium and gadolinium-based reagents have been used as a contrast agent for detection in MS for a while (33-35).

Interference of gadolinium with some metal has been indicated (36, 37) and was suggested to collect the blood sample for metal analyses at least 96 hours after gadolinium administration (38). In our study blood collections are done during the yearly hospital checks ups.

Cigarette smoking has been indicated as a risk factor for MS (39, 40, 41). In our study, we did not reveal any significant associations between smoking status and MS incidence. But, relative risk associated with smoking was higher than 1.0. This indicates that smoker subjects are more likely to have MS.

On the other hand, relative risk associated with smoking was more pronounced in males than whole population (Relative risk > 2.0). MS revealed that smoking is associated with worse prognosis of MS and is an accelerator of the conversion of RRMS to a progressive MS (42).

Cadmium levels were significantly lower in MS patients ($p < 0.0001$). Smoking is a risk factor for multiple sclerosis. Cadmium was not found in relation with multiple sclerosis.

Patient diets and health recommendations done by their doctors can make lifestyle changes and all these should be taken into consideration in randomized double-blinded multi-centered clinical studies with larger cohorts and more markers to further understand the relation of cadmium (Cd) and smoking in multiple sclerosis

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SS-06 / 4402

YÜKSEK VE DÜŞÜK DÜŞME KORKU DÜZEYİNE SAHİP MULTİPL SKLEROZ HASTALARINDA DENGE VE YÜRÜME PERFORMANSLARININ KARŞILAŞTIRILMASI

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Özet

Amaç: Düşme korkusu, denge ve yürüme bozukluğunun bir sonucu olarak ortaya çıkmaktadır. Ancak multipl skleroz (MS) hastalarında denge ve yürüme performanslarının düşme korkusu düzeyine göre karşılaştırılmadığı görülmektedir. Bu nedenle yüksek ve düşük düşme korku düzeyine sahip MS hastalarında denge ve yürüme performanslarının karşılaştırılması amacıyla bu çalışma planlanmıştır.

Gereç ve Yöntem: Çalışmaya toplam 25 MS hastası hasta dahil edilmiştir. Düşme korkusu Uluslararası Düşme Etkinlik Ölçeği (UDEÖ) ile değerlendirilmiştir. UDEÖ'den 23 ve üzeri puan alan 13 MS hastası yüksek düşme korku düzeyi grubunu (yaş:38.84±8,72, EDSS:3.30±0.96), 23 puan altında alan 12 MS hastası ise düşük düşme korku düzeyi grubunu (yaş:31.58±8,62, EDSS:1.45±1.07) oluşturmuştur. Denge; Berg Denge Ölçeği (BDÖ), Dört Adım Kare Testi (DAKT), Tek Ayak Üzerinde Durma Testi (TAÜDT) ve Aktiviteye Özgü Denge Güven Ölçeği (AÖDGÖ) kullanılarak değerlendirilmiştir. Yürüyüş; 10 Metre Yürüme Testi (10MYT), Sekiz Şekilli Yürüme Testi (8ŞYT), 3 Metre Geri Yürüme Testi (3MGYT) ve Süreli Kalk Yürü Testi (SKYT) kullanılarak incelenmiştir.

Bulgular: Hastaların EDSS skorları dışında demografik ve klinik özellikleri arasında anlamlı fark olmadığı gözlenmiştir. Çalışma sonucunda yüksek düşme korku düzeyi grubunun BDÖ, DAKT, TAÜDT, AÖDGÖ, 8ŞYT, 3MGYT ve SKYT skorlarının düşük düşme korku düzeyi grubuna göre anlamlı derede daha kötü olduğu bulunmuştur ($p<0.001$, $p<0.001$, $p=0.035$, $p<0.001$, $p<0.001$, $p<0.001$ ve $p<0.001$, sırasıyla). Ancak, iki grup arasında 10MYT sonuçlarında anlamlı bir fark yoktu ($p>0,05$).

Sonuç: Bu sonuçlar, özellikle yüksek düşme korku düzeyine sahip olan MS hastalarının rehabilitasyon programlarında denge ve kompleks yürüme becerilerine yönelik egzersizlere daha çok yer verilmesi gerektiğini göstermektedir.

Anahtar Sözcükler: Multiple skleroz, Düşme korkusu, Denge, Yürüme

COMPARISON OF BALANCE AND WALKING PERFORMANCES IN MULTİPLE SCLEROSIS PATIENTS WITH HIGH AND LOW FEAR OF FALLING

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Abstract

Objectives: Fear of falling (FoF) occurs as a result of balance and walking disorders. However, balance and walking performances of multiple sclerosis (MS) patients are not compared according to the level of FoF. Therefore, this study was planned to compare balance and walking performances in MS patients with high and low FoF.

Methods: A total of 25 MS patients were included in the study. FoF was evaluated with the Falls Efficacy Scale-International (FES-I). Thirteen MS patients with scores of 23 and above on the FES-I constituted the high FoF group (age:38.84±8,72, EDSS:3.30±0.96), and 12 MS patients with a score below 23 constituted the low FoF group (age: 31.58±8,62, EDSS:1.45±1.07). Balance was assessed using the Berg Balance Scale (BBS), Four-Step Square Test (FSST), One-leg Stance Test (OLST), and Activity-Specific Balance Confidence Scale (ABC). Walking was examined using the 10-Meter Walk Test (10MWT), Figure of Eight Walk Test (F8WT), 3-meter Backward Walk Test (3MBWT) and Timed Up and Go Test (TUG).

Results: There were no significant differences between groups in any demographic and clinical characteristics except EDSS scores. As a result of the study, it was found that the BBS, FSST, OLST, ABC, F8WT, 3MBWT, and TUG scores of the high FoF group were significantly worse than the low FoF group ($p<0.001$, $p<0.001$, $p=0.035$, $p<0.001$, $p<0.001$, $p<0.001$, and $p<0.001$, respectively). However, there was no significant difference in 10MWT results between the two groups ($p>0.05$).

Conclusion: These results demonstrate that exercises for balance and complex walking skills should be included more in the rehabilitation programs of MS patients, especially those with a high level of FoF.

Keywords: Multiple sclerosis, Fear of falling, Balance, Walking skills

SS07 / 6505

EFFECTS OF COMORBIDITY ON DISABILITY, DEPRESSION AND QUALITY OF LIFE IN MULTIPLE SCLEROSIS

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Objectives: The heterogeneity of the disease course in Multiple Sclerosis (MS) has been associated with various factors, one of them is the presence of comorbidity. Several studies

suggest that, increasing frequency of comorbidities with aging and longer disease duration, may affect the disease course in MS. The aim of this study is to investigate the relationship between the presence of comorbidities and disability, depression and quality of life (QOL) in people with MS (PwMS).

Methods: Patients who were followed in our MS outpatient clinic were included in the study. Patients' demographics, clinical characteristics, comorbidities and Expanded Disability Status Scale (EDSS) scores were recorded. Beck Depression Invantery (BDI) and The Multiple Sclerosis Quality of Life-54 (MSQOL-54) scale were applied. In subgroup analysis, the effects of comorbidity on disability, depression and QOL in patients who have a diagnosis of MS for more than 10 years were evaluated.

Results: Of 90 patients (mean age, 40.1 ± 9.3 years, 61.1% female), 47.7% (n=43) had comorbidities and the rate of depression in this group was found to be higher than those without any comorbidities (p=0.03). Patients with depression (n=18) had higher EDSS scores (3.1 vs 1.7, p=0.01) and lower MSQOL-54 scores (162.6 vs 177.6, p=0.02) than those without depression. In patients with a diagnosis of MS over 10 years (n=38), the EDSS scores of those with comorbidity (50%) were higher than those without (3.5 vs 1.8, p=0.03).

Conclusion: Comorbidity in MS increases the rates of depression and negatively affects the EDSS scores and QOL. Especially in PwMS with a disease duration of 10 years or more, the presence of comorbidity is associated with poor EDSS scores. Early diagnosis and proper management of diseases accompanying MS can potentially improve the disease course.

Keywords: Multiple Sclerosis; comorbidity; depression; quality of life

SS-08 / 7441

Effects of Therapy On the Serum Levels of Apelin and Sirtuin 1 in Responder and Non-responder Multiple Sclerosis Patients

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Abstract:

Some patients fail to achieve adequate response at therapeutic doses of IFN-β and GA in multiple sclerosis. This variability in response has prompted the search for prognostic markers in order to personalize therapy. Our goal was to investigate apelin and sirtuin1 levels as a possible predictor of response to IFN-β and GA.

IFN-β, GA treated responder and non- responder patients serum levels of apelin and SIRT1 were assessed by ELISA before and after treatment.

GA responder group had higher SIRT1 levels than all other groups. There wasn't any difference for apelin. A significant decrease was found in serum SIRT1 levels in non-responder GA group.

In previous studies, NLRP3 levels were found to be high in patients who responded to IFN-β and SIRT1 levels were higher in GA responder patients as in our study. Our data suggest that SIRT1 could a possible biomarker to evaluate patients' responsiveness to GA therapy.

Keywords: Multiple Sclerosis, Interferon beta, Glatiramer acetate, apelin, sirtuin1

1. Introduction:

Multiple sclerosis (MS) is a neuroinflammatory and neurodegenerative disease of the central nervous system (CNS) with considerable heterogenity including response to therapy (1,2). MS patients commonly present with the relapsing-remitting form of the disease (RRMS). RRMS usually managed with first and second line immunomodulatory drug treatments, such as interferon beta (IFN- β), glatiramer acetate (GA), fingolimod (FTY), teriflunamide and dimetilfumarate(3). RRMS has a heterogeneo-

us natre, so it is difficult anticipate patients prognosis or response to treatment(1,3). There is significant need for the validation of biomarker for the prediction of disease ectivity and treatment response. IFN- β regulate the expression of pro and anti inflammatory genes(3,4,5). GA act as an altered peptide ligands to inhibit myelin basic protein specific T helper cells. Some of the patients fall to achieve adequate response at the repetitive doses of IFN- β and GA(6,7). This variability in response to treatment has prompted the search for prognostic markers in order to personalize the therapy so as to treat MS more effectively(8).

Apelin is a peptide that encoded by the APLN gene. It is a key regulator in a normal glucolipid metabolism, glucose uptake, lipolysis, fatty acid oxidation and mitochondrial biogenesis. Sirtuin 1 (SIRT1) is a member of the class III histon deacetylases (HDACs) family of proteins (9,10). It regulates possitively the function of Th17 cells. It was shown that SIRT1 expression in MS patients is significantly decreased during relapses (11).

The objective of this study is to investigate the roles of apelin and SIRT1 serum levels as a possible predictor of response to IFN- β and GA treatment in MS.

2. Materials and Methods:

2.1. Study Design and Data Collection

Patients who were diagnosed with multiple sclerosis in Gazi University Faculty of Medicine, Neurology Department included in this study. We include 34 RRMS patients and 20 healthy controls. The criteria for inclusion of MS patients in this study were:

1. Fullfilment of McDonald criteria for definite MS
2. RRMS course
3. Treated with IFN- β (n=18), GA (n=16) for 3 year period
4. No treatment with any other drugs
5. EDSS between 0,5 and 5

The patients were divided into four groups. Responders to IFN- β or GA treatments were defined as patient who exhibited no relapse during the 3 year period following the initiation of IFN- β and GA.

1. Responder IFN- β group
2. Non-responder IFN- β group
3. Responder GA group
4. Non-responder GA group

2.2. Experimental Procedure

Enzyme-linked immunosorbent assay (ELISA) was used for the quantitative measurement of apelin (Bioassay Technology Laboratory® Human Apelin (AP) ELISA Kit, Shanghai, China) and sirtuin 1 (Bioassay Technology Laboratory® Human SIRT1 Kit, Shanghai, China)in serum samples with in-vitro technique. Peripheral blood samples (10ml) were collected from the patients and healthy controls and centrifugated at 3000 rpm for 5 minutes for separating the serum. The serum samples were stored at -20° C until analysis. After standard mediums that containing sirtuin 1 and apelin antibodies were diluted, serum samples were added into the wells and incubated at room temperature for 2.5 hours. Soon after, washed and then incubated with a biotin antibody for 1 hour. After washing, streptavidin was added, left for 45 hours, washed again and one step reagent was added and incubated again for 30 min. The concentration of sirtuin 1 and apelin was determined spectrophotometrically. Absorbance values at 450 nm were determined using an ELISA reader.

2.3. Statistically Analysis

Analysis of the data was done using the IBM SPSS 21.0 statistical package program. Pearson Chi-square test was used to compare qualitative data as well as descriptive statistical methods (frequency, percentage, mean, standard deviation, median, min-max) while evaluating study data. The relationship between variables according to age was examined by independent samples t-test. The statistical significance level was set at $p<0.05$.

1. Results

It was found that 68% of the participants were female and 32% were male. The mean age of the participants was found to be 37, the youngest age was 20 and the oldest age was 56.

In this study, we investigated the relationship between apelin and SIRT1 serum levels and responsiveness to treatment with IFN- β and GA. Responders to IFN- β or GA treatments were defined as patient who exhibited no relapse during the 3 year period following the initiation of IFN- β and GA, whereas two groups of nonresponders exhibited two or more relapses. We found a significant decrease in SIRT1 serum levels in GA non-responders when compared with all other groups. There wasn't any statistical difference in apelin serum levels between IFN- β and GA responder, non responder and control groups (Table 1,2).

Table 1: Serum apelin and SIRT1 levels in IFN- β group

IFN- β Group	Responder	Non-responder	Controls
Apelin (pg/ml)	591.4±272.6	586.9±283.7	587.5±225.2
SIRT1 (ng/ml)	2.6±0.4	2.1±0.9	2.4±0.7

Table 2: Serum apelin and SIRT1 levels in GA group

GA Group	Responder	Non-responder	Controls
Apelin (pg/ml)	594.7±267.5	589.6±254.6	587.5±225.2
SIRT1 (ng/ml)	4.2±1.3	0.9±0.3	2.4±0.7

2. Conclusion

Multiple sclerosis is a disease that involves all natural, acquired immune cells, cytokines, chemokines and various molecules with progressive neurodegeneration additionally to inflammation (1,2, 11). In this compound immunopathogenesis, we aimed to evaluate the serum levels of apelin and SIRT1 as a biomarker for IFN- β and GA responsiveness.

Only one current data have demonstrated that the levels of SIRT1 mRNA are significantly decreased in GA non responder RRMS group (12-14). Our study confirm this prior results (14).

In recent years, new studies have just been conducted on the role of apelin in MS, but sufficient data have not yet been generated about its role in neuroimmunological conditions. Mainly, neuroprotective effects are emphasized in conditions such as neurodegenerative processes and ischemic brain damage in previous studies(9). For this reason, we were interested in whether the apelin has a role in evaluating the response to treatment in both an inflammatory and degenerative process such as MS.

SIRT1, which has a protein deacetylase and adenosine diphosphate (ADP)-ribosyl transferase activity, is a molecule that has been studied and focused on its epigenetic effect in both MS and other neurological diseases, especially neurodegenerative processes, in recent years. SIRT1 expression levels are positively correlated with histon acetylation and methylation; compared the levels in GA responder and non responders(14,15). Histon H3K9 levels are decreased in GA non responders. It may indicate transcriptional activation of proinflammatory genes. In addition, other studies have implicate H3K9 as a supressor of IFN type 1 and IFN response genes which are key regulators of innate antiviral immunity (16,17).

There have been some limitations in our study. The most important limitation of our study was the small sample size. Also we didn't take into account the number of demyelinating lesions on neuroimaging if there was no clinical relapse. In addition, in our study, we aimed to evaluate the 3-year relapse rate rather than the annual relaps rate as in many studies.

Eventhough the study has limiting features, it has been one of the few studies to search biomarkers that might be use to evaluate IFN and GA response in MS patients. Our results were suggested that SIRT1 may have a predicting value for therapy response of GA. Particularly, decreased levels of SIRT1 in GA non-responder group has been drawn our attention. According to our and other studies, SIRT1 may be an effective biomarker response to GA therapy than response to IFN- β therapy in RRMS patients (12,14). The results of this study may allow to find either biomarkers for follow-up or new treatment options for this complex MS pathogenesis.

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SS-09 / 4945

EVALUATION OF THE RETINAL VASCULAR NETWORK WITH OPTICAL COHERENS TOMOGRAPHY ANGIOGRAPHY IN PATIENTS WITH MULTIPLE SCLEROSIS AND ITS RELATIONSHIP WITH CLINICAL

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Objective: In patients with multiple sclerosis (MS); with the aid of optical coherence tomography angiography (OCTA) to reveal the relationship of vascular involvement in the retina and optic nerve with disease-related clinical parameters.

Methods: Eye measurements of 49 MS patients and 28 healthy controls, most of whom were diagnosed with relapsing remitting multiple sclerosis (RRMS), were included. After the ophthalmological examination, vascular measurements of the retina and optic nerve head were performed using OCTA. In the patient group, 23 (ON- MS) patients without a history of optic neuritis (ON) and 26 (ON + MS) patients with a history of optic neuritis were compared among themselves and with the healthy group in terms of retinal and optic nerve head blood supply parameters. Patients were divided into subgroups according to parameters such as EDSS value, number of attacks, year of disease, visual acuity, CSF results and visual evoked potential (VEP) findings. Detailed optic nerve head vascular measurements using OCTA were compared with MS-related clinical data in the participants.

Results: The parameters we examined for optic nerve head vascular evaluation were TI-PPDD (48.2 ± 3.4 p=0.02), ID-DD (45.8 ± 5, p=0.015), PPDD (50.9±4, 3, p<0.001), UTI-PPDD (51.3 ± 4.3, p<0.001) and AF-PPDD (50.5 (40.1-60.1), p<0.001) measurements were found to be statistically significantly lower in the patient group. When the ON+ group was compared with the ON- group, no significant difference was observed between these two groups; When the ON+ group and the healthy group were compared, among the optic nerve blood supply parameters, except ID-DD (TI-PPDD (48.2 ± 3.5a, p=0.008), ID-DD (45.9 ± 4.9, p=0.050), PPDD (50.6 (41.1- 59.5)a, p=0.001), UTI-PPDD 50.8 (42.1-59.9)a, p=0.005), MD-PPDD (50.2 ± 4.6a, p<0.001)) significant reduction was found. When compared in terms of other MS-related clinical parameters and OCTA findings, there was no correlation between EDSS value and CSF findings and OCTA data, but it was observed that vascular involvement was significantly higher in the group with more attacks and years of disease, decreased visual acuity, and impaired VEP measurement.

Conclusion: In our study, it was observed that vascular involvement of the optic nerve head was correlated with many disease-related parameters, especially the history of ON. More studies are needed to emphasize the importance of OCTA data in determining prognosis and determining the degree of clinical involvement in patients.

Keywords: multiple sclerosis, optical coherens tomography angiography, optic neurit

SS-10 / 8505

DECREASED LEVELS OF SOLUBLE FRACTALKİNE AS AN EARLY BIOMARKER DISTINGUISHING SECONDARY PROGRESSIVE FROM RELAPSİNG REMITTING MULTIPLE SCLEROSİS

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Abstract

Secondary Progressive Multiple Sclerosis (SPMS) differs from Relapsing Remitting Multiple Sclerosis (RRMS) in clinical, immunological, pathological, radiological charecteristics and response to treatment. A spesific biomarker for the transition from RRMS to SPMS have not yet been reported, so our aim in this study is to evaluate the role of soluble fractalkine (sFKN) as an early biomarker for disease progression in distinguishing between SPMS and RRMS.Fractalkine is a unique chemokine with direct relationship with its receptor to reduce expression of pro-inflammatory genes in activated microglia.In our study,serum levels of sFKN levels were found to be decreased in SPMS compared to RRMS and healty controls.As a result,the decrease in serum sFKN levels support the development of neurodegeneration and promote the transition from RRMS to SPMS.thus sFKN can be used as an early biomarker and in future it will be a promising new therapeutic target.

Introduction

In generally, MS starts relapsing remitting course (RRMS), at later times,may transform into SPMS.

Two types of inflammation have been reported in MS patients. The first is the focal invasion of T and B lymphocytes which affect the white matter and causes active demyelinated lesions.The other type is associated with the formation of subpial demyelinated lesions, a slow accumulation of T and B cells and with diffuse neurodegeneration in the white or gray matter. Neurodegeneration is associated with progressive clinical disease(1).Cortical lesions in SPMS are charecterised by activated microglia and inflammatory infiltrates. Meningeal lymphocytic aggregates have been found in patiennts with SPMS.Risk factors associated with progression to SPMS inlude longer disease duration,male sex,higher baseline EDSS score, lower brain volume, spinal cord involvement. There is no definitive laboratory test indicative of progressive disease, however, measures of disability progression widely used in clinical practise(2). Barbour C et al, reported CSF biomarkers are able to separate RRMS from progressive forms, CSF biomarker-based approaches are not yet integrated into neurological examination(3). On the other hand,in our previous study, in acomparison of the MS patients and controls, we found that the median values of the EDSS scores among genotype of the V2941 polymorphism in the fractalkine gene receptor were statistically higher in genotype II than genotype VI. Also RRMS was statistically higher in genotypeVI than in genotypeII,where as the frequency of SPMS was statistically higher in genotypeVV than in the genotypeVI for the same polymorphism.(4).In another study,it was shown that the CX3CR1 I(249) T(280) haplotype has a protective effect when transforming into the SPMS(5). To date,no spesific clicnical,immunologic,patholodi,radiologic marker to determine when RRMS transform into SPMS. There is a need for biomarkers to support early detection of SPMS.For this reason,we searced previously unstudied sFKN for its ability to be an early biomarker distinguishing SPMS from RRMS.

Materials and Methods

Our study consisted of 24 SPMS, 38 RRMS and 30 healty controls. SPMS and RRMS were diagnosed according to Mc Donald 2017 criteria(6) and were not taken any drug at the time of the study. The degree of neurologicaldeficits were assessed by Expanded Disability Status Scale (EDSS). All individuals in the patient and control group were informed about the study and consent of the patients was obtained. Serum levels of sFKN were tested by Enzyme Linked Immunosorbent Assay(ELISA) method. 5cc peripheral blood samples were taken from the patient and healty controls, centrifuged at 5000 rpm for 10 minutes, and the serum was separated and stored at -20 degrees until analysis. Fractalkine concentration was determined spectrophotometrically.

Absorbances read at 450 nm.Statistical analysis were performed using the IBM SPSS 21.0 Differences among SPMS and RRMS patients groups were assessed with Chi-square,Mann-Whitney and Fisher exact test.

Results

Clincal charecteristics of the paeticipants of this study is presented in Table 1. According to this table,in the SPMS group, there were 15 women with a mean age of 38.02, a total of 24, in the RRMS group, there were a total of 38 patients, 28 of whom were women with a mean age of 31.06. Disease duration 12.21 in the SPMS group, 4.67 in the RRMS group, EDSS was 5.5±1.7 in the SPMS group and 1.5±1.6 in the RRMS group. The patients and healty control groups were similar in terms of age and gender. No statistically significant differences was found in terms of the mean age,gender distribution and disease duration between the groups. As assessed by the chi-square test serum sfractalkine mean values did not show a statistically significant and difference in terms of genders(p=0.253). Although there was a weak negative correlation between serum fractalkine levels and age and disease duration,but this relationship was not statistically significant (p=0.614,p=0.086). Serum fractalkine levels of patients and healty controls are shown in Table 2. Serum Fractalkine levels

of SPMS group were significantly lower than RRMS group and controls ($p<0.05$).

Table 1.Clinical charecteristics of the participants

	SPMS	RRMS	CONTROL
Age	37.02±3.72	31.06±2.68	34.18±4.0
Sex			
a.Female	15	28	20
b.Male	9	10	10
Duration of disease (year)	12.21	4.67	---
EDSS	5.5±1.7	1.5±1.6	---

Table 2: Serum Fractalkine levels of patients and healty controls.

	Fractalkine (pg/ml)
SPMS (n=24)	0.52±2.14
RRMS (n=38)	
a:During the attack	1.2±0.32
b:After the attack	0.98±0.16
Controls (n=30)	0.74±3.32

Discussion

Cytokines and chemokines are konown to play important role in the immunopathogenesis of MS. Among chemokines serum levels of FKN was reported to be elevated in RRMS patients (7). However, it has not been observed in patients with SPMS. FKN is a chemokine that can exist i a soluble form, as chemotactie cytokine, or in a membrane-attached form that arts a binding molecule. FKN dose dependently suppressed the production of nitric oxide (NO), interleukine-6 (IL-6), tumor negrosis factor alpfa (TNF- α) with activated microglias. It is also significantly suppressed neuronal cell death induced by microglia activated with lipopolisaccarides (LPS) and interferon- gamma (INF- γ) in dose dependent manner. These possible functions of fractalkine as on intrinsic inhibitor against neurotoxicity by activated microglia and may be an intrinsic neuroprotective chemocine in the CNS. However, due to its dual effects, FKN exerts numerous effects on pathophysiological conditions that have both negative and positive consequences on immunopathogenesis (8-13).

FKN is a unique chemokine with direct relationship with its receptor (CX3CRI) to reduce expression of proinflammatory genes in activated microglia (14). According to the results of our study, the decrease in sFKN level supports the development of neurodegeneration by increasing neuroinflammation in CNS. It may contribute to the neurodegenerative mecanisms of progression and promote the transition from RRMS to SPMS. As a result our research shows that sFKN can be used as an early biomarker associated with disease progression in distinguishing between SPMS and RRMS, Additionally in future it will be a promising new therapeutic target.

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SS-11 / 9518

MULTİPL SKLEROZDA AKDENİZ DİYETİNE UYUM DURUMU, YÜRÜYÜŞ ETKİLENİMLERİ VE YAŞAM KALİTESİ ETKİLENİMLERİNİN BELİRLENMESİ

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Amaç: Multipl Skleroz'lu (MS) bireylere özel diyet konusunda kanıtlar halen yetersizdir. Akdeniz diyeti sağlığı olumlu etkileyen bir beslenme modelidir. MS hastalarında akdeniz diyetine uyum, yürüyüş etkilenimleri ve yaşam kalitesinin arasındaki ilişkinin değerlendirilmesi amaçlanmıştır.

Gereç ve Yöntem: Çalışmaya Sanko Üniversitesi Hastanesi Nöroloji polikliniğinde takip edilen, 19-65 yaş arasında, 81 hasta (13 erkek, 68 kadın) dahil edildi. Dahil edilen bireylerin demografik bilgileri, MS alt tipleri ve Genişletilmiş Özürlülük Durum Ölçeği (EDSS) kaydedildi. Olguların akdeniz diyetine uyumları Akdeniz Diyeti Uyum Ölçeği (MEDAS), yürüyüyüş etkilenimleri 12 maddeli MS Yürüyüş Skalası (MSWS-12), yaşam kaliteleri Multipl Skleroz Uluslararası Yaşam Kalitesi Ölçeği (MusiQoL) ile değerlendirildi.

Bulgular: Dahil edilen bireylerin yaş ortalamaları 35,9±10,3, vücut kütle indeksi 25,5±5,3 kg/cm^2, EDSS 1,7±1,5 idi. Bireylerin 68'i RRMS, 8'i SPMS, 4'ü PPMS, 1'i KİS idi. Bireylerin akdeniz diyetine uyumları incelendiğinde 19'u düşük uyum, 54'ü orta uyum, 8'i yüksek uyum düzeyindeydi. MSWS-12 ortalaması 27,56±14,23, MusiQoL toplam skoru ortalaması 70,63±14,93 idi. MEDAS ile MusiQoL toplam skoru ve MSWS-12 arasında anlamlı ilişki bulunmadı. MEDAS ile EDSS arasında negatif yönde zayıf ilişki bulundu (p=0,007, r=-0,297). MusiQoL toplam skoru ile MSWS-12 (p<0,001, r=-0,398) ve EDSS (p=0.042, r=-0,227) arasında negatif yönde zayıf ilişki bulundu.

Sonuç: Çalışmamızda EDSS puanı düşük olan hastalarının akdeniz diyetine uyumları anlamlı olarak daha yüksek oranda bulundu. MS hastalarının yaşam kalitesi değerlendirmesinde yürüyüş bozukluklarının etken olabileceği gözlendi.

Anahtar Kelimeler: Multipl Skleroz, Akdeniz Diyeti, Yaşam Kalitesi, Yürüyüş

GİRİŞ ve AMAÇ

Multipl Skleroz (MS) inflamasyon, demiyelinizasyon ve akson hasarı ile karakterize otoimmün bir hastalıktır (1). Dünyada yaklaşık 2,5 milyon bireyi etkileyen ve daha çok genç erişkinlerde görülen MS, bireylerde fonksiyonel aktivitelerde farklı düzeylerde özürlülüğe yol açabilir, sosyal ve mesleki hayatta kısıtlamalara neden olabilir ve yaşam kalitesini bozabilir (2, 3). MS'in genetik yatkınlık ve çevresel faktörler zemininde geliştiği düşünülür. Beslenme alışkanlıkları ve fiziksel aktivite önemli çevresel faktörlerdir. Bunlar MS'in seyrinde etkili olabilirler ve yaşam kalitesini değiştirebilirler (1, 4).

Çeşitli beslenme modellerinin MS üzerine etkisini inceleyen klinik araştırmalar devam etmekte olup, MS'li bireyler için herhangi bir özel diyet tedavisi kullanımı konusunda kanıtlar halen yetersizdir (5). Hastalığın daha yaygın olduğu gelir düzeyi yüksek batı ülkelerinde yaşayan bireyler genel olarak; sedanter yaşam sürmekte, güneş ışınlarına daha az maruz kalmakta hayvansal kaynaklı doymuş yağlardan ve rafine şekerden zengin, yüksek enerjili diyetle beslenmektedir (2). Akdeniz diyeti evrensel olarak sağlığı geliştirdiği düşünülen bir beslenme modelidir ve bu diyetin kronik hastalıkları önlemede ve tedavide yararlı etkileri olduğu bilinmektedir (6). Akdeniz diyeti ile batı tarzı diyet arasındaki en büyük fark diyetsel yağ ve karbonhidratların kaynağı ve miktarıdır. Akdeniz diyeti, MS'in önlenmesine ve seyrinde olumlu etkileri olabileceği düşünülen, tekli doymamış yağ (özellikle omega-3) ile antioksidan vitamin ve mineraller (A, C, E vitaminleri, folik asit, selenyum, çinko, kalsiyum) içeren besinlerden zengindir (6-8). Akdeniz tipi beslenmede hayvansal yağlar yerine bitkisel yağların tercih edilmesi, kırmızı et tüketimi yerine balık tercih edilmesi, süt ve süt ürünlerinin az yağlı / yağsız olarak tercih edilmesi, basit şeker ve rafine karbonhidrat alımının sınırlandırılması, posa ve antioksidanlardan zengin meyve sebze tüketiminin arttırılması gibi özellikler olması nedeniyle bu beslenme tipinin inflamasyonu azaltarak immün hastalıkların seyrini ve tedavisini olumlu etkileyebileceği düşünülmektedir (9-11).

MS'in en yaygın semptomları kas güçsüzlüğü olduğundan MS'li bireyler genellikle yaş uyumlu sağlıklı yetişkinlere göre fiziksel olarak daha az aktiftir. Bu durum fonksiyonel aktivitelerde azalma, mobilite kaybı ve denge problemleri ile sonuçlanmaktadır (12). Yürüme ve daha geniş anlamda mobilite, MS'de sıkça etkilenen fonksiyonlardan olup, yaşam kalitesi üzerine olumsuz etkileri fazladır. MS'de yürüyüş problemleri sıktır ve bu gruptaki bireylerin önemli bir kısmında günlük yaşam aktivitelerinin olumsuz etkilendiği rapor edilmektedir (13).

Ülkemizde MS hastalarının günlük hayatlarında süregiden beslenme alışkanlıklarının nasıl olduğu konusunda yeterli bilgi yoktur ve yaşam kalitesi çalışmaları sınırlıdır. Bu konularda bilgimizin sınırlı olması MS hastalarında düzeltilebilecek ve bu sayede yaşam kalitesine olumlu etki sağlayabilecek bir alanda hareket imkânını daraltmaktadır. Beslenmenin Akdeniz diyeti ile uyumlu olması genel olarak fiziksel ve ruhsal iyilik halinin, dolayısıyla kişinin yaşam kalitesinin artmasında faydalı olabilir.

Bu araştırmanın amacı MS hastalarında Akdeniz diyetine uyum, özürlülük durumu, yürüyüş etkilenimleri ve yaşam kalitesi arasındaki ilişkinin kesitsel olarak değerlendirilmesidir.

GEREÇ ve YÖNTEM

Bu çalışma SANKO Üniversitesi Sani Konukoğlu Uygulama

ve Araştırma Hastanesi Nöroloji Polikliniği'nde takip edilen 18-65 yaş arası yetişkin MS hastaları üzerinde yürütüldü. Anket formundaki sorular sorulmaya başlanmadan önce hastalara; bu çalışmada Akdeniz diyetine uyum, yürüyüş ve yaşam kalitesi ile ilgili sorular yöneltileceği açıklandı. Bu aşamada çalışmaya katılmak zorunda olmadıkları, çalışmaya katılmayı ya da katılmamayı tercih edebilecekleri ve her iki durumda da takiplerinin her zamanki gibi yapılacağı belirtilerek, çalışmaya katılmak isteyip istemedikleri soruldu. Çalışma amacının açıklanması üzerine çalışmaya katılmayı kabul eden 81 (13 erkek, 68 kadın) MS hastası çalışmaya dahil edildi. Araştırma esnasında gebe veya postpartum dönemde olan, araştırma sırasında atak geçiriyor olan, aktif enfeksiyon olan, fizikel aktivite yapmasına engel başka bir hastalığı olan, özel bir diyet yapması gerekecek hastalığı olan bireyler dahil edilmedi. Dahil edilen bireylerin demografik bilgileri ve MS alt tipleri (RRMS, SPMS, PPMS, KİS) kaydedildi. Hastaların özürlülük durumlarının tespiti Genişletilmiş Özürlülük Durum Ölçeği (Expanded Disability Status Scale- EDSS) kullanılarak belirlendi (14). Bireylerin Akdeniz diyetine uyum durumlarının belirlenmesinde 14 soruluk Akdeniz Diyeti Uyum Ölçeği (MEDAS) kullanıldı (15). Ölçeğin Türkçe geçerlilik ve güvenilirlik çalışması Pehlivanoğlu ve arkadaşları tarafından yapılmıştır (16). Bireylerin yaşam kalitesi, Multipl Skleroz Uluslararası Yaşam Kalitesi Ölçeği (Multiple Sclerosis International Quality of Life - MusiQoL) ile belirlendi (17,18). Türk Toplumu için geçerlilik ve güvenilirlik çalışması Türkiye'nin de içinde olduğu 20 ülkede ve 14 ayrı dilde eşzamanlı olarak yapılmış ve tamamlanmıştır (19). Yürüyüş etkileniminin belirlenmesinde 12 maddeli MS Yürüyüş Skalası (MSWS-12) kullanıldı (20). Türkçe geçerlilik ve güvenilirlik çalışması Pehlivanoğlu ve arkadaşları tarafından yapılmıştır (21).

Verilerin istatistiksel analizinde SPSS 20.0 kullanıldı. Sürekli değişkenler için ortalama (X̄) ve standart sapma (ss) değerleri, kategorik değişkenler için sayı (n) değerleri verildi. Veriler normal dağılıma uymadığı için ilişkiler Spearman Korelasyon testi ile belirlendi. Sonuçlar $p<0.05$ düzeyinde anlamlı kabul edildi.

BULGULAR

Dahil edilen 81 bireyin (13 erkek, 68 kadın); 68'i RRMS alt tipinde olup, 54 birey Akdeniz diyetine orta uyum göstermekteydi. Bireylerin cinsiyet, MS alt tipi ve Akdeniz diyetine uyum sınıfı özellikleri Tablo 1'de verilmiştir.

Tablo 1. Bireylerin Cinsiyet, Ms Alt Tipi ve Akdeniz Diyetine Uyum Sınıfı Özellikleri

Özellik (n=81)		**n**
Cinsiyet	Kadın	68
	Erkek	13
MS alt tipi	RRMS	68
	SPMS	8
	PPMS	4
	KİS	1
Akdeniz diyetine uyum durumu	Düşük uyum	19
	Orta uyum	54
	Yüksek uyum	8

Bireylerin yaş, VKİ, EDSS, MEDAS, MSWS-12 ve MusiQoL ortalamaları Tablo 2'de verilmiştir.

Tablo 2. Bireylerin Yaş, VKİ, EDSS, MEDAS, MSWS-12 ve MUSİQoL Ortalamaları

Özellik (n=81)	**X̄±ss**
Yaş	35,9±10,3
VKİ (kg/m²)	25,5±5,3
EDSS	1,7±1,5
MEDAS	6.97±1.99
MSWS-12	27,56±14,23
MusiQoL	70,63±14,93

Bireylerin MEDAS ortalaması ile EDSS, MSWS-12 ve MusiQoL ortalamaları arasındaki ilişkiler değerlendirildiğinde; MEDAS ile MusiQoL toplam skoru ve MSWS-12 arasında anlamlı ilişki bulunmadı. MEDAS ile EDSS arasında negatif yönde zayıf ilişki bulundu ($p=0,007$, $r=-0,297$) (Tablo 3).

Tablo 3. Bireylerin MEDAS Ortalaması ile EDSS, MSWS-12 ve Musiqol Ortalamaları Arasındaki İlişki

Özellik (n=81)	**EDSS**	**MSWS-12**	**MusiQoL**	
MEDAS	**0,007***	0,085	0,204	**p**
	-0,297	-0,193	0,143	**r**

Bireylerin MusiQoL ortalaması ile EDSS ve MSWS-12 ortalamaları arasındaki ilişkiler değerlendirildiğinde MusiQoL toplam skoru ile EDSS ($p=0.042$, $r=-0,227$) ve MSWS-12 ($p<0,001$, $r=-0,398$) arasında negatif yönde zayıf ilişki bulundu (Tablo 4).

Tablo 4. Bireylerin MusiQoL Ortalaması ile EDSS ve MSWS-12 Ortalamaları Arasındaki İlişki

Özellik (n=81)	**EDSS**	**MSWS-12**	
MusiQoL	**0,042***	**0,000***	**p**
	-0,227	**-0,398**	**r**

TARTIŞMA ve SONUÇ

Çalışmamıza katılan bireylerin büyük çoğunluğu Akdeniz diyetine orta-yüksek uyum göstermekteydi. Ortalama MEDAS skoru orta uyum düzeyine denk gelmekteydi. MS hastaları üzerinde Akdeniz diyetine bağlılık ile yorgunluk şiddeti arasındaki ilişkiyi değerlendirmek amacıyla yürütülen başka bir çalışmada da MS hastalarının ortalama MEDAS skoru çalışmamıza benzer şekilde orta uyum düzeyinde bulunmuştur (22). MS hastalarının genel olarak yüksek sosyokültürel düzeyde olması sağlıklı beslenme davranışları göstermelerinde etkili olabilir.

EDSS puanı düşük olan hastalarının Akdeniz diyetine uyumları anlamlı olarak daha yüksek oranda bulundu. Akdeniz diyet müdahalesinin MS üzerine etkisini araştıran randomize kontrollü bir çalışmada 128 MS hastası kontrol ve müdahale gruplarına ay-

rılarak, müdahale grubuna Akdeniz diyetine uyumu teşvik emek amacıyla 6 ay boyunca beslenme eğitimi verilmiştir. Araştırma sonucunda müdahale grubunun EDSS puanlarında istatistiksel olarak anlamlı bir düşüş olduğu gözlenmiştir (4). MS hastaları üzerinde yürütülen Güney İtalya hasta kohortundan 435 MS hastasının verilerinin alındığı kesitsel bir çalışmada da Akdeniz diyetine uyumun hastaların özürlülük durumlarını azalttığı belirlenmiştir (23). Benzer bulguların daha büyük örneklemli ileri çalışmalarla desteklenmesi durumunda Akdeniz diyeti MS hastalarında özürlülük durumunun azaltılmasında önerilebilecek bir beslenme modeli olabilir.

Çalışmamızda MS hastalarının yaşam kalitesi değerlendirmesinde yürüyüş bozukluklarının etken olabileceği gözlendi. Ayrıca çalışmamızda MS hastalarının yaşam kalitesi ile özürlülük durumu arasında ters yönde ilişki olduğu gözlendi. Hastaların yaşam kalitesinin EDSS skoru ve yürüyüş etkilenim düzeyi ile ters ilişkili olması beklenen ve çeşitli çalışmalarla da desteklenen (13) bir sonuçtur. Öte yandan bir çalışmada MS hastalarının yaşam kalitesi durumlarında hastanın mizacının EDSS skorundan daha yüksek oranda etkili olduğu sonucuna ulaşılmıştır (24). Hastaların yaşam kalitesi ile yürüyüş etkilenimi ve özürlülük durumları arasındaki ilişkilerin değerlendirildiği ileri çalışmalara ihtiyaç duyulmaktadır.

MS hastalarının beslenme, özürlülük durumu, yürüyüş etkilenimi ve yaşam kalitesi durumlarının belirlenmesi; hem hastaların mevcut durumlarının belirlenmesi hem de beslenme durumunun MS üzerine etkisinin gözlemleneceği ileriye yönelik çalışmalara veri oluşturulması açısından önemlidir.

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MULTİPL SKLEROZLU HASTALARDA YEME DAVRANIŞ BOZUKLUĞUNUN DEĞERLENDİRİLMESİ

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Amaç: Bu çalışmada genellikle genç erişkin yaşlarda başlayan ve kronik bir hastalık olan multipl skleroz (MS) hastalarında beslenme durumu ve yeme bozukluklarının değerlendirilmesi amacı ile yapılmıştır. Yeme bozuklukları da MS'de olduğu gibi sıklıkla genç erişkin ve adölesan yaşlarda görülebilmektedir.

Materyal-Metod: Çalışmaya Mc Donald kriterlerine göre kesin MS tanısı almış 35 hasta alındı. Hastaların Expanded Disability Status Scala (EDSS) skoru, MS alt tipi, hastalık süresi, vücut kitle indeksi (VKİ) gibi demografik veriler kayıt edildi. Hastalara yeme bozukluklarının değerlendirilmesi amacıyla Yeme Bozukluğu Değerlendirme Ölçeği (YEDÖ), REZZY Yeme Bozuklukları Ölçeği uygulanmıştır. YEDÖ ve REZZY ortalamaları hesaplandı.

Bulgular: MS hastalarının yaş ortalaması 37.8±12.3/yıl ve ortalama EDSS skorları 1.9'du. Hasta grupları 30 (%85.7) kadınlardan, 5(%14.3) erkeklerden oluşuyordu. Ortalama 7,5 yıl önce MS tanısı konmuştu. Hastaların VKİ ortalaması 26.7±5.7 idi. Hastaların YEDÖ ortalama puanı 1.36 (cut off değeri 4 ve üzeri) ve REZZY ortalama puanı ise 1.5 (cut off değeri 2 ve üzeri) olarak değerlendirildi istatiksel anlamlılığa ulaşmadı. VKİ 25 üstü olan kişilerde REZZY testinde değerlendirildiğinde anlamlı farklılık ortaya çıkmıştır (p=0.003). Hastalık süresi ve hastalık tipi ile YEDÖ ve REZZY arasında anlamlı ilişki saptanamamıştır.

Sonuç: Çalışma sonucunda MS'li hastalarda özellikle VKİ 25'in üzerinde olan hastalarda yeme bozukluğu riskinin arttığı gözlenmiştir. YEDÖ ve REZZY testleri ile değerlendirilen yeme bozukluğunun MS hastalık süresi, alt tipi ve EDSS ile ilişkisi saptanmamış olup sadece VKİ ile ilişkilisi saptanmıştır. MS hastalarında özellikle VKİ yüksek olan hastalarda yeme bozukluklarına yönelik taramalar yapılabilir ve böylece gerekiyorsa takip edilip tedavi imkanı sağlanmalıdır.

EVALUATION OF EATING BEHAVIOR DISORDER IN PATIENTS WITH MULTIPLE SCLEROSIS

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Aim: The aim of this study was to evaluate the nutritional status and eating disorders in patients with multiple sclerosis (MS), a chronic disease that usually starts in young adults. Eating disorders can also be seen frequently in young adults and adolescents, as in MS.

Material-Method: Thirty-five patients with a definite diagnosis of MS according to McDonald criteria were included in the study. Demographic data of the patients such as Expanded Disability Status Scala (EDSS) score, MS subtype, disease duration, and body mass index (BMI) were recorded. The Eating Disorder Examination Questionnaire (EDE-Q) and REZZY Eating Disorders Scale were applied to the patients in order to evaluate their eating disorders. The mean of EDE-Q and REZZY were calculated.

Results: The mean age of MS patients was 37.8±12.3/year and the mean EDSS score was 1.9. The patient groups consisted of 30 (85.7%) women and 5 (14.3%) men. MS was diagnosed an average of 7.5 years ago. The mean BMI of the patients was 26.7±5.7. The patients' mean EDE-Q score was 1.36 (cut-off value 4 and above) and REZZY mean score was 1.5 (cut-off value 2 and above) did not reach statistical significance. When the REZZY test was evaluated in individuals with a BMI above 25, a significant difference emerged (p=0.003). No significant relationship was found between disease duration and disease type, and between EDE-Q and REZZY.

Conclusion: As a result of the study, it was observed that the risk of eating disorders increased in patients with MS, especially in patients with a BMI above 25. Eating disorder, which was evaluated with the EDE-Q and REZZY tests, was not associated

with MS disease duration, subtype, and EDSS, but only with BMI. In MS patients, especially in patients with high BMI, screening for eating disorders can be done, so that if necessary, follow-up and treatment should be provided.

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LESIONS MIMICKING TRANSVERSE MYELITIS

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Abstract

Transverse myelitis (TM) is a rare, acquired focal inflammatory disorder often presenting with rapid onset weakness, sensory deficits and bowel/bladder dysfunction. Generally occurring independently as a complication of infection; however, it may also exist as part of a continuum of other neuro-inflammatory disorders. The list of differential diagnoses of TM is long; hence, a meticulous history and detailed physical examination are indispensible. Despite the improvement of ancillary diagnostic techniques, including neuroimaging and serological testing, the correct diagnosis of acute and subacute myelopathy is challenging. A systematic, careful history and may help exclude other mimics of TM. In this article, we aimed to evaluate different etiological diagnosis in patients who presented with TM clinic.

Introduction

Transverse myelitis (TM) includes a pathobiologically heterogeneous syndrome characterized by acute or subacute spinal cord dysfunction resulting in paresis, a sensory level and autonomic (bladder, bowel, and sexual) impairment below the level of the lesion. Etiologies for TM can be broadly classified as parainfectious, paraneoplastic, drug/toxin-induced, systemic autoimmune disorders (SAIDs) and acquired demyelinating diseases like multiple sclerosis (MS) or neuromyelitis optica (NMO) (1). Patients with isolated TM present a diagnostic dilemma, as it is common in both MS and NMO, but may also be the initial manifestation of SAIDs. Also, there are noninflammatory etiologies (eg, vascular, metabolic) that may mimic the clinical and radiologic appearance of TM. The cause of TM remains unknown despite an extensive workup in about 15% to 30% of patients and is therefore referred to as "idiopathic" according to set criteria (2,3).

The annual incidence of TM ranges from 1.34 to 4.60 cases per million, but increases to 24.6 cases per million if acquired demyelinating diseases like MS are included. TM can occur at any age, although a bimodal peak in incidence occurs in the second and fourth decades of life (4). Half of patients have an antecedent infection. The list of differential diagnoses of TM is long; hence, a meticulous history and detailed physical examination are indispensible. A systematic and careful history may help exclude other mimics of TM. Magnetic resonance imaging (MRI) of the complete spinal axis is mandatory in any patient with myelopathic features to exclude structural lesions. Treatment is directed towards the cause.

Methods

We retrospectively studied 9 consecutive patients with clinical evidence of TM. All patients underwent spinal MRI, 4 underwent cerebrospinal fluid analysis. Solid lesions were diagnosed with biopsy. Various other studies were performed to assess for connective tissue disease and causes of TM other than demyelinating disease. The inclusion criteria were as follows: patients with sensory defect giving level or deep sensory loss. Exlusion criteria included patients aged<18 years, pregnant women, who cannot have MRI. The lesion was visualized by MRI in all patients. The variables of interest were patient demographic characteristics (age,gender), neurologic examination, MRI findings and definitive diagnosis.

Results

Different etiological reasons were found in the evaluation of 9 patients who applied with the TM clinic. 4 patients are male and 5 patients are female. The lesion was detected in the thoracic region in 7 patients and in the cervical cord in 2 patients. While all patients with thoracic lesion had sensory defect giving level and deep sensory loss was detected in 2 patients with cervical lesion. 3 patients had paraparesia and 1 patient had monoparesia. The lesion was visualized by MRI in all patients. For the differential diagnosis, further investigations were made and a definitive diagnosis was made. Differential diagnoses of TM and cases features are shown in the table.

PATIENT	AGE	GENDER	SYMPTOM	MR LESION	DIAGNOSIS
1	56	F	T4 sensory defect that giving level-paraparesia	T6-7 lesion	TM
2	55	F	T4 sensory defect that giving level -paraparesia	Lesion extending from the cranio-cervical junction to the upper thoracic cavity.	NMO
3	36	F	T4 sensory defect that giving level -paraparesia	T1-4 multipl MS lesions	MS
4	86	F	Deep sensory loss	C3-6 dorsal cord lesion	SCD-B12
5	37	M	T5 sensory defect that giving level	T5 lesion	TM-COPPER
6	55	M	Parestesia	C2-5 dorsal cord lesion	TSCI
7	64	F	Left T8-9 sensory defect that giving level-left leg monoparesia	Left T8-9 subdural lesion	MENINJIOMA
8	73	M	T4 sensory defect that giving level	T2-4 solid lesion	TUMOR
9	36	M	T4 sensory defect that giving level	T3 lesion	METASTASIS

Table: Differential diagnoses of TM and cases features

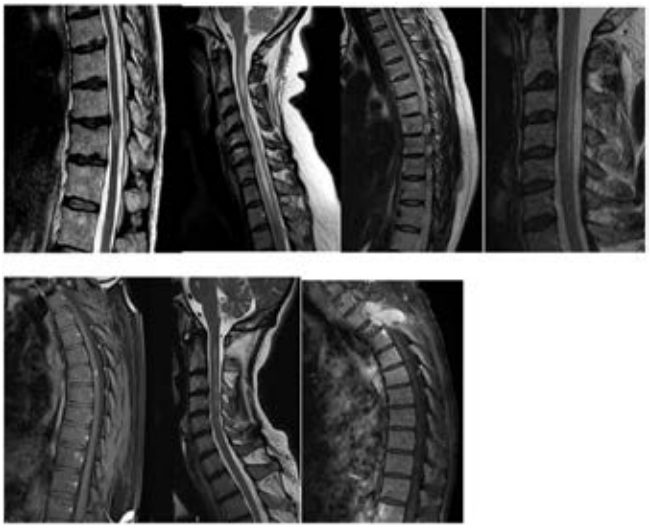

Table 2: MR lesions of patients 1:TM 2: NMO 3:MS 4:SCD 5:TM 6:TSCI 8:TUMOR

Discussion

TM typically has an acute to subacute onset with neurologic deficits reaching within a few weeks. TM may present as one of several syndromes of the spinal cord. Acute complete TM (ACTM) manifests as paresis/plegia, sensory dysfunction (characterized by numbness, paresthesias, or other manifestations in conjunction with a sensory level) and autonomic impairment below the level of the lesion. Acute partial TM (APTM) results in asymmetric manifestations or deficits specific to particular anatomic tracts; manifestations include the hemi-cord (Brown-Sequard), central cord, or posterior column syndrome, as well as selective tract impairment. The list of differential diagnoses of TM is long; hence, a meticulous history and detailed physical examination are indispensible. A systematic and careful history may help exclude other mimics of TM. Acute transverse myelitis is a subgroup of various conditions characterized by focal inflammation of the spinal cord and resultant neural injury. The etiologies of myelopathies are varied and can be subdivided into compressive and noncompressive causes. While compressive myelopathies stem from trauma and intra or extraspinal tumors, the etiologies of non-compressive myelopathies can be classified as delayed radiation effects, ischemic, paraneoplastic, systemic ADs and infectious or parainfectious. Cerebrospinal fluid (CSF) analysis is essential in the evaluation of TM. CSF cell count, differential, protein, glucose, oligoclonal bands (OCBs) and IgG index should be checked on all patients with TM. MRI of the complete spinal axis is mandatory in any patient with myelopathic features to exclude structural lesions [3]. In this report; the lesion was detected in the thoracic region in 7 patients and in the cervical cord in 2 patients. While all patients with thoracic lesion had sensory defect giving level and deep sensory loss was detected in 2 patients with cervical lesion presented with acute complete TM symptoms. All patients were diagnosed with MRI. CSF examination was performed in some patients for differential diagnosis and blood tests (B12 level, thyroid function tests, syphilis, HIV serologies and inflammatory markers) that may cause myelitis were requested.

NMO usually causes attacks of severe AON (sometimes bilateral) and brainstem lesions resulting in intractable nausea, vomiting or hiccups (5,6). Although the manifestations of NMO may be similar to MS, attacks are typically more devastating. Serum aquaporin-4 specific autoantibodies (NMO-immunoglobulin Ig G) should be checked on all patients with TM because of its high specificity for NMO or NMO spectrum disorders (NMOSD) (7). NMO-IgG sero-positivity is rarely found in patients with APTM but its presence would have profound implications on treatment. Inflammatory markers should be checked if SAID is suspected (8). NMO is diagnosed on the basis of the revised Wingerchuk criteria requiring the presence of optic neuritis and TM as well as 2 of 3 of the following: NMO antibodies, LETM, and/or brain MRI lesions inconsistent with MS. In making the diagnosis of TM, it is essential to remember that many noninflammatory etiologies may mimic the appearance of TM. Recognizing these entities is important, as the treatment and management strategies would be vastly different (9). Patient with number 2 presented with loss of strength in the legs. A lesion compatible with NMO was detected in thoracic MRI. Cranial MRI, NMO antibodies and CSF examinations performed afterwards confirmed the diagnosis of NMO.

MS is a disabling progressive neurologic disorder affecting approximately 400,000 people in the United States (10). TM in MS most commonly presents with sensory phenomena. Spine MRI typically reveals an asymmetrically placed lesion (usually occurring in the posterolateral or lateral portion of the spinal cord) less than 2 segments in length with a predilection for the cervicothoracic cord. White matter lesions predict higher risk of conversion to MS (with rates of up to 88% reported). If the lesions meet at least 3 of the Barkhof criteria this risk is increased substantially (11). The third patient presented with loss of strength in the legs and sensory defect that gives level. In the thoracic MRI, the lesions were found to be compatible with MS. Afterwards, a definitive diagnosis of MS was made with cranial MRI and CSF examination.

Serum vitamin B12 level, thyroid function tests, syphilis and HIV serologies always should be obtained to evaluate for potentially treatable causes of myelopathy. Vitamin E, serum copper and ceruloplasmin levels are checked in those at risk of deficiency. Subacute combined degeneration (SCD) of the spinal cord is the most common neurologic manifestation of vitamin B12 (cobalamin) deficiency and is usually secondary to autoimmune gastritis, but may also be seen in malnutrition syndromes such as chronic alcoholism, strict vegetarianism, gastrectomy and also in nitrous oxide abuse. Although traumatic spinal cord injury is routinely encountered in the medical examiner's office, medical causes of spinal cord abnormalities such as SCD should be considered in the appropriate clinical setting (12). Patient with number 4 was admitted with the complaint of imbalance due to profound sensory loss. A lesion compatible with SCD was observed in thoracic MRI. Therefore, the reasons that could cause SCD were investigated and B12 deficiency was detected in this patient.

Copper deficiency myelopathy represents an often underdiagnosed, acquired neurological syndrome, clinically characterized by posterior column dysfunction. The main causes of copper

deficiency are bariatric surgery, increased consumption of zinc, and malabsorption. However, even after a careful history taking and extensive laboratory researches, the etiology of copper deficiency remains undetermined in a significant percentage of cases. Patients affected by copper deficiency myelopathy usually present with sensory ataxia due to dorsal column dysfunction and sometimes with mild leg spasticity. In such patients, spinal cord MRI may show hyperintense lesions in T2-weighted sequences involving the posterior columns of cervical and thoracic cord (13). Patient with number 5 presented with profound sensory loss and leveling sensory defect. A myelitis compatible lesion was detected in the thoracic MRI. Since type 4 OCB was positive in the CSF examination, metabolic causes were investigated and copper deficiency was detected.

Traumatic spinal cord injury (TSCI) is associated with higher morbidity, affecting young individuals causing severe and permanent disabilities that incur huge healthcare burden on the patients. TSCI is a clinicoradiological diagnosis of post-traumatic neurological weakness with bony deformity based on radiography and computed tomography and confirmed by MRI (14). Spinal cord injury without radiographic abnormality (SCIWORA) is a recognized form of SCI, infrequently reported in adults and characterized by the absence of any radiographically evident fracture or dislocation (15). It is defined as spinal cord lesions that are observed by MRI but do not show lesions in plain radiography. The reported incidence of SCIWORA among adult SCI cases ranges from 10% to 12%,7 and falls from height, motor vehicle accidents, and sports-related injuries are the major injury mechanisms. The epidemiology and pathophysiology of SCIWORA differ among adults and children, with most adult patients presented with radiographic abnormalities showing degenerative changes (16,17). The patient with number 6 applied with the complaint of numbness in the whole body prominent in the arms. Posterior cord lesion was observed in Cervical MRI. Since the patient's complaints started after falling from a height, a diagnosis of SCIWORA was made.

Spinal meningiomas are found in all age groups, predominantly in women aged over 50 years. Their incidence is approximately 3 per 100.000. The clinical symptoms of this condition may range from mild to significant neurological deficit, varying widely depending on the location, position in relation to the spinal cord, size and histological type of the tumor. MRI is the diagnostic tool of choice because it shows the location, size, the axial position of the tumor, and the presence of concomitant conditions such as spinal malformations, edema or syringomyelia. The goal of the surgery is total resection which is achievable in 82%-98% of cases. Advances in radiosurgery have led to its increased use as primary or adjunct therapy (18,19).Seventh Patient applied with monoparesia. A lesion compatible with meningioma was detected in Thoracic MRI. The patient was treated surgically.

Intramedullary tumors represent only 10% of the spectrum of spinal tumors as these lesions develop in 60% of the cases on the epidural aspect and are in 30% of the cases intradural and extramedullary. They are mostly glial tumors. Ependymomas and lowgrade astrocytomas are the leading histological types. Their management is not an easy task and depends essentially on the surgical resection which can be very challenging. Adjuvant therapies have a very controversial role. They represent 30% of intramedullary tumors. They mostly affect children and usually are of low grade. Cervical and thoracic localizations are the most frequent. Astrocytomas are usually not well limited; they are mostly infiltrative. They present a tumoral cyst in 48% of the cases. Hemorrhage is less frequent than ependymomas. Clinical presentations join those of ependymoma; it is usually progressive with motor and sensitivity troubles. They present on the MRI as a noncentral infiltrative tumor enlarging the spinal cord. They are hypointense on T1 and hyperintense on T2. Enhancement is heterogeneous and has no correlation to the grade of the tumor contrarily to cerebral astrocytomas. Leptomeningeal enhancement can orient for highgrade lesions (20,21). Patients 8 and 9 presented with sensory defect that gives level. Solid lesion was detected in thoracic MRI in both of them. Both patients were diagnosed with biopsy. One had primary spinal cord tumor and the other had metastasis.

Conclusion

Differential diagnoses of acute/subacute intrinsic spinal cord lesions includes demyelinating, inflammatory or infectious diseases, tumors and metabolic deficits. Despite the improvement of ancillary diagnostic techniques, including neuroimaging and serological testing, the correct diagnosis of acute and subacute myelopathy is challenging. In general, MRI findings of acute myelitis may be multifocal and/or longitudinally extensive, as in neuromyelitis optica, where hyperintensity in T2 weighted images characteristically spans over three or more contiguous vertebral segments. On the other hand, intramedullary neoplasms typically show gadolinium enhancement, mass effect and cord expansion. SCD of the spinal cord is the most common neurologic manifestation of vitamin B12 and copper deficiencies. SCD can be completely cured with early treatment. TSCI and SCIWORA can effect the spinal cord by mimicking SCD.

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PS-02 / 1889

TÜMEFAKTİF MULTİPL SKLEROZ OLGULARINDA TEDAVİ YAKLAŞIMI : OLGU SERİSİ

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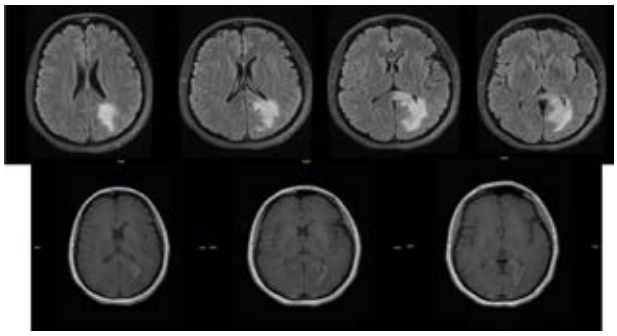
AMAÇ: Tümefaktif MS, tanısal ve terapötik bir zorluk oluşturan nadir bir multipl skleroz varyantıdır. Tümefaktif MS'de kitle etkili büyük beyin inflamatuar demiyelinizan lezyonlar ve perilezyonel ödem yani tümefaktif demiyelinizan lezyonlar bulunur. (TDL)

(1,2) Akıllardaki soru, tümefaktif MS'in rastgele daha büyük lezyonlarla ortaya çıkan MS mi veya farklı patolojisi, klinik prezentasyonu ve öyküsü olan kendi başına bir hastalık olup olmadığı sorusudur.(1) Bu çalışmada kliniğimizde Tümefaktif MS tanısı alan 3 olguyu sunmak istedik.

YÖNTEM: İlk ziyaretler, hastaneye yatışlar, klinik takip notları dahil olmak üzere multipl sklerozlu bu üç hastanın tablosunu geriye dönük olarak inceledik ve demografik olarak, başvuru belirti ve semptomları, görüntüleme yöntemleri, beyin omurilik sıvısı analiz sonuçları hakkında veriler topladık.

OLGU 1

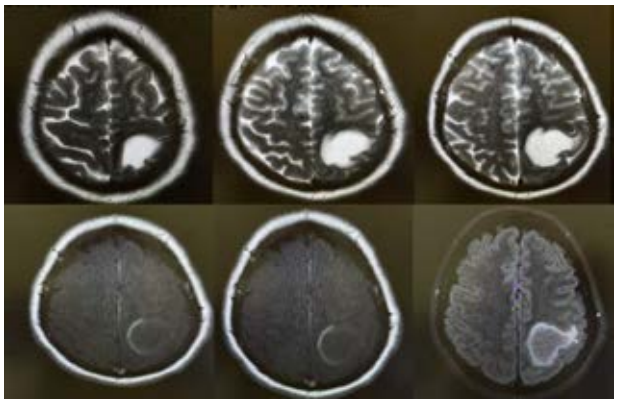
Bilinen kronik hastalığı olmayan 38 yaşında kadın hasta, COVID-19 enfeksiyonundan 2 hafta sonra başlayan her iki bacakta uyuşukluk ve sağ bacakta güç kaybı şikayetleri ile başvurdu. Nörolojik muayenesinde sağ alt ekstremite 4+/5 motor kuvvette, Derin tendon refleksleri alt ekstremitelerde hiperaktif, sağda aşil klonusu +, T4 seviyesi altında hipoestezik, bilateral babinski pozitif olduğu görüldü. Spinal MRG'de Th1-2-3 segmentlerinde 3 adet demyelinizan plak görüldü.. Kranial MRG'de, Sol parietooksipitalde yaklaşık 3,5x1x1,5 cm (periferal kontrastlanan, çevresinde ödematöz değişiklikler bulunan lezyon izlendi. BOS OKB ve anti-AQP4: Negatif olarak sonuçlandı.7 gün pulse steroid tedavisi başlanan hasta, tedaviden fayda gördü. Hastada Tümefaktif MS düşünüldü. Takibinde glatiramer asetat başlandı.



OLGU 2

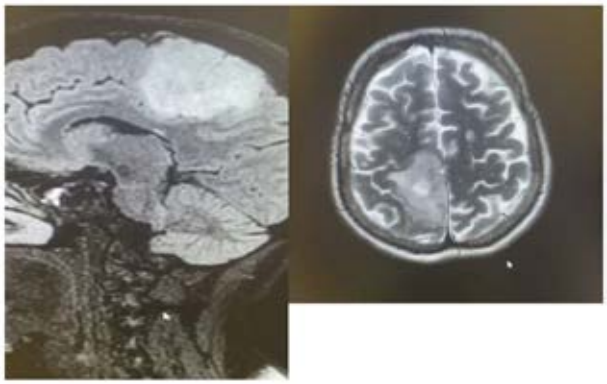
37 yaş kadın hasta, 2-3 haftadır devam eden sağ kol ve bacakta hissizlik uyuşma şikayetleri ile başvurdu. Nörolojik muayenede, sağ hemihipoestezi ve hiperaktif derin tendon refleksleri görüldü. Daha önce optik nörit atağı nedeniyle göz hastalıkları yatışı olan hastaya tarafımızca lp yapılmış olup BOS'ta OKB ve NMO negatif bulunmuştu.

Hastanın Kranial MR'ında Sol parietalde yaklaşık 39x35mm buyutlu, düzgün sınırlı, kısmen ince periferal kontrastlanan intraaksiyal lezyon, lezyon çevresinde minimal ödem örüldü. Lezyonun ince duvarı diffüzyon kısıtlılığı gösteriyordu. Servikal MR'da belirgin patoloji izlenmedi. Tümefaktif MS düşünülen hastada pulse steroid tedavisi sonrası hastanın şikayetlerinde gerileme olduğu izlendi.



OLGU 3

Bilinen MS tanısı olan 39 yaşında gebe hasta yeni başlangıçlı jeneralize tonik-klonik ile nöbet ile başvurdu. Nörolojik muayenede sol üst ve alt ekstremiteleri 4 / 5 motor kuvvette ve sol alt ekstremite hipoestezik olduğu görüldü. Kontrastsız kranial mr'da Sağda parietal lobda yaklaşık 55x50 mm boyutunda ödematöz yüksek sinyalli kortikal- subkortikal lezyon alanı görüldü. Servikal MR'da servikal spinal kord boyunca ağırlıklı olarak spinal kordun sağ periferal yarımı tutan yaygın dağınık yerleşimli demiyelinizan plak alanları olduğu görüldü. EEG sol temporal bölgede hafif paroksismal düzensizliğin varlığıyla uyumluydu. Hastaya öncelikle 5 gün pulse tedavisi başlandı. Ardından belirgin düzelme görülmemesi nedeniyle 5 gün gün aşırı plazmaferez tedavisi uygulandı. Video EEG monitorizasyon yapılarak hastanın antiepileptik ilaçları düzenlendi. Tümefaktif MS tanısı ile hastanın takibine devam ediliyor.



TARTIŞMA VE SONUÇ

Çalışmada üç olgununda 3. Dekatta kadın olduğunu yaşlarının birbirine çok yakın olduğunu gördük. Literatürde Kadın/Erkek oranının 3:1 olduğunu, Tümefaktif demiyelinizan lezyonların her yaşta görülebileceğini fakat 20-30 yaş arasının daha sık etkilendiği görülmektedir.(2) Sunulan 2 hastanın daha önce bilinen MS tanısı yoktu. İlk iki olgu piramidal semptomlar ile diğer olgu ise nöbet ile prezente oldu. Yine hastaların yarısından fazlasında, piramidal sistem en sık etkilenen fonksiyonel sistem olduğunu biliyoruz.(1) Her üç olguda Kranial MRG'de periferik kontrastlanmanın ve diffüzyon

kısıtlamasının eşlik ettiği, çevresel ödemin olduğu büyük hemisferik lezyonlar gördük. İki hastada kliniğimizde BOS incelemesi yaptık. Hastalarda BOS'ta OCB, anti-AQP4 pozitifliği saptamadık. TDL, multipl skleroz(MS), nöromiyelitis optika spektrum bozukluğu (NMOSD, akut dissemine ensefalomiyelit (ADEM) veya diğer immünolojik hastalıklarda da ortaya çıkabildiğinden TDL tanımlandığında, ve otoantikorların tahlilini içeren non-invaziv yöntemler (örn. aquaporin-4 otoantikorları) önerilir.(4) Akut atak tedavisi olarak üç hastaya da pulse steroid bir hastaya ek olarak plazmaferez tedavisi uyguladık. Literatürde TDL'lerin, steroid tedavisine zayıf yanıtı olduğu söylenmekle birlikte biz hastalarımızdan ikisinin pulse steroid tedaviye iyi yanıt verdiğini gözlemledik.(1) Bu çalışmada nadir görülen bu MS varyantı ile ilgili deneyimlerimizi paylaşmak istedik. Hastaların takiplerine MS polikliniğinde devam ediyoruz.

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TREATMENT MANAGEMENT IN TUMEFACTIVE MULTIPLE SCLEROSIS : CASE SERIES

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OBJECTIVES: Tumefactive MS is a rare variant of multiple sclerosis that poses a diagnostic and a therapeutic challenge. In tumefactive MS, are found of large brain inflammatory demyelinating lesions with mass effect and perilesional edema, that is, tumefactive demyelinating lesions are found. (TDLs). (1,2) The question remains whether tumefactive MS is just MS randomly presenting with larger lesions, or rather a disease on its own with distinct pathology, clinical presentation and natural history. (1) The aim of this study, is to present three cases diagnosed as Tumefactive MS in our clinic.

METHODS: We retrospectively reviewed the chart of the theese three patients with multiple sclerosis including initial visits, hospitalizations, clinic follow up notes and collected data on demographic, presenting signs and symptoms, imaging modalities, cerebrospinal fluid analysis results.

CASE 1: Patient 1 was a previously healthy 38-year-old woman who presented with numbness in both legs and right leg weakness, which started 2 weeks after the COVID-19 infection. Neurological examination revealed 4+/5 motor strength in the right lower extremity, hyperactive deep tendon reflexes in the lower extremities, + Achilles clonus on the right, hypoaesthesia below the T4 level, bilateral Babinski. Spinal MRI showed 3 demyelinating plaques in Th1-2-3 segments. On cranial MRI, a lesion of approximately 3.5x1x1.5 cm (peripherally enhanced, with edematous changes around it) was observed in the left parietooccipital. Oligoclonal band in CSF: Negative, anti-AQP4: Negative. Pulse steroid therapy was started for 7 days. The patient benefited from the treatment. Tumefactive MS was considered in the patient. In the follow-up, glatiramer acetate was started.

CASE 2: Patient 2 was 37-year-old female is admitted with complaints of numbness in the right arm and leg that has persisted for 2-3 weeks. Neurological examination revealed right hemihypoesthesia and hyperactive deep tendon reflexes. She had previously been hospitalized in the ophthalmology clinic due to optic neuritis. In the patient who underwent lumbar puncture by us, OCB and anti-AQP4 in CSF: negative was found. On Cranial MRI, approximately 39x35mm in size, well-circumscribed, partially thin peripherally enhanced intraaxial lesion in left parietal region There is minimal edema around the lesion and The thin wall of the lesion showed diffusion restriction. No obvious pathology was observed in cervical MRI. Tumoractive MS was considered. After pulse steroid treatment, the complaints of the patient was improved.

CASE 3: Patient 3 was a 39 year old pregnant woman with MS presenting with new onset generalized tonic clonic seizures. Neurological examination revealed that the left upper and lower extremities were 4 / 5 motor strength and the left lower extremity was hypoesthetic. On non-contrast Cranial MRI, an edematous high-signal cortico- subcortical lesion area of approximately 55x50 mm is observed in the parietal lobe on the right. In cervical MRI, diffusely located demyelinating plaque areas are observed along the cervical spinal cord, predominantly involving the right peripheral half of the spinal cord. EEG was consistent with the presence of mild paroxysmal irregularity in the left temporal region. Firstly, pulse therapy was started for 5 days. Since there was no significant improvement, plasmapheresis treatment was applied every other day for 5 days. Video EEG monitoring was performed and the patient's antiepileptic drugs were arranged. The patient continues to be followed up with the diagnosis of tumefactive MS.

CONCLUSION: In this study, we saw that three of the cases were female in the 3rd decade and their ages were very close to each other. In the literature, that the female/male ratio is 3:1 and that tumefactive demyelinating lesions can be seen at any age, but it is more common between the ages of 20-30. (2) 2 of the 3 patients had no known previous diagnosis of MS. The first two cases presented with pyramidal symptoms and the other case presented with seizures. We know that in more than half of the patients, the pyramidal system is the most frequently affected functional system.(1) In cranial MR of all three cases, we observed large hemispheric lesions with peripheral edema accompanied by peripheral enhancement and diffusion restriction. We performed CSF examination in two of the three patients in our clinic. We did not detect OCB, anti-AQP4 positivity in CSF of the patients. Once TDL has been identified, non-invasive methods (eg, aquaporin-4 autoantibodies) are recommended, including assay of autoantibodies. Because TDL can also occur in multiple sclerosis (MS), neuromyelitis optica spectrum disorder (NMOSD), acute disseminated encephalomyelitis (ADEM) or other immunological diseases.(4) As acute attack treatment, we applied pulse steroid in three patients and plasmapheresis in one patient. Although it is said in the literature that TDLs respond poorly to steroid therapy, we observed that two of our patients responded well to pulse steroid therapy.(1) In this study, we

would like to share our experience with this rare variant of MS.

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PS-03 / 8950

NUMMULAR HEADACHE ASSOCIATED WITH MULTIPLE SCLEROSIS

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ABSTRACT

Nummular headache (NH) or coin shaped cephalgia is a rare headache disorder. Mostly a baseline mild-to-moderate pressure like pain is present in a circumscribed round or elliptical area. Patients usually suffer from superimposed exacerbations lasting seconds to hours besides this baseline pain. The pathogenesis of NH is still unclear. The topography of NH suggests that the pain has a probable peripheral source affecting the sensory branches of pericranial nerves. On the other hand, a central mechanism of pain has also been proposed for NH. We report a case of multiple sclerosis (MS) patient with nummular headache. Although in some cases NH can be seen as a comorbid condition of migraine and tension type headache and it can be secondary to brain trauma and intracranial mass lesions.MS and NH association has not been reported before. To our knowledge, this is the first case of NH associated with MS.

INTRODUCTION

Nummular headache (NH) or coin shaped cephalgia is a rare headache disorder. In one study in a general neurology outpatient clinic, nummular headache counted for 1.25% of all headache patients (1). Female predominance has been reported (2).Mostly a baseline mild to moderate pressure-like pain is present in a circumscribed area of 1-6 cm diameter in the parietal region, albeit it may also be located in other cranial regions. In one series there is slight occipital predominance (2). This pain never shifts sides, changes its shape or size. Vast majority of the cases are unifocal but bifocal and very rare multifocal cases are also described and it was suggested that central mechanims of pain may facilitate the multifocality(3). Although it is a chronic dull, pressure-like or burning pain, it is commonly associated with superimposed exacerbations of sharp, stabbing or throbbing pain and periods of spontaneous remissions lasting weeks to months. Exacerbations may be spontaneous or evoked by an external stimulus such as combing or touching (4). Paresthesia, hypoesthesia, hyperesthesia, dysesthesia, tenderness, allodynia and trophic changes may accompany the pain in that focal area (5). In migraine and tension-type headache a more diffuse scalp sensitivity may accompany the headache. In chronic tension type headache (CTTH) increased pericranial tenderness may be present and clinically distinguishes CTTH from

NH (6).There are some cases with no baseline pain where the pain only comes in attacks and it may be difficult to distinguish it from primary stabbing headache (5).It was first described by Pareja et. al in 2002 (7) and was included in International Classification of Headache Disorders, 3rd edition beta version under other primary headache disorders (8).

The mechanisms are not clear, peripheral and also central pathways are accused in the pathophysiology.It is thought to be a complex regional syndrome arising from epicranial structures (9-11). Secondary forms of NH might coexist with classical primary NH (4,5,12). A small number of cases have been reported to be successfully treated with various therapies, yet there is no effective standard treatment (13-17).

Although in some cases NH can be seen as a comorbid condition of migraine, tension type headache and can be secondary to brain trauma, intracranial mass lesions and arachnoid cysts, so far there is no reported case of NH secondary to MS (4,5,7,18-23). The relationship of MS and specific headache syndromes is poorly known. Case reports illustrated that isolated MS lesions in the midbrain may cause severe headaches often resembling migraine (22). Treatment with disease-modifying agents may sometimes trigger headache in MS patients (23). To our knowledge, this is the first report of an MS patient with NH.

CASE REPORT

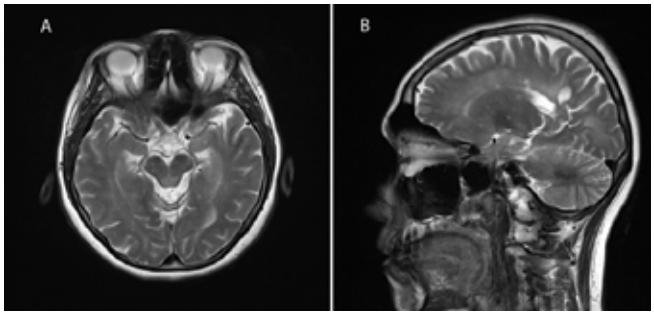
A 32 year old woman presented with left hemiparesis and hemihypoesthesia. She also defined severe throbbing headache attacks which was associated with photophobia, phonophobia, nausea and vomiting but not aura. The headache was predominantly in the left frontotemporal region. Her MRI revealed demyelinating lesions as show in Figure 1. Cerebrospinal fluid showed increased levels of IgG and presence of oligoclonal bands. She was treated with high doses of intravenous methylprednisolone and obtained moderate improvement of the neurological symptoms and signs. One month later, she presented with optic neuritis in the left eye and glatiramer acetate treatment was started after another pulse treatment with iv methylprednisolone. Over the period of 3 months all her symptoms and signs improved considerably and after 2 years she only had hypoesthesia in the left arm. She still has occasional migraine attacks without aura, which is responsive to treatment with triptans.

After four years, she began to suffer from a new focal headache for the past 5 months. The pain was localized in the left posterior parietal region in a rounded zone, 4 cm in diameter. It was mild, continuous, with periods of exacerbations lasting hours; nausea, vomiting, photophobia, phonophobia, increment with physical efforts were distinctly absent. She suffered from migraine without aura since the age of 22. This pain was clearly segregated from migraine with its clinical characteristics and response to treatment. During the periods of frequent episodes of migraine she was successfully treated with triptans, although this focal pain persisted unchanged. There was no response to NSAIDs, triptans, amytriptyline, valproic acid but topiramate. She was successfully treated with topiramate 100 mg/day. She quitted topiramate spontaneously after 6 months without recurrence of pain until now .

In her last examination, there were no signs or symptoms of sensory dysfunction in the symptomatic area and touch-evoked pain or trigger points were not found. Routine blood work-up was normal. Brain MRI showed numerous periventricular and callosal white matter hyperintensities in T2 weighted images which were consistent with demyelination (Figure 1).

FIGURES

Figure 1.(A) Demyelinating lesions at periaquaductal regions in midbrain, T2 weighted axial section, (B) Periventricular lesions T2 weighted sagittal section, Brain MRI



DISCUSSION

The present case fulfilled the criteria for the diagnosis of NH. Since the first description of NH, more than 200 cases have been reported worldwide. The majority of them were primary forms. However probably NH prevalance is underestimated since not all NH patients admit to a neurological out-patient clinic and there are cases misdiagnosed as psychiatric disorders. In a previous study, no association was found between NH and depression and anxiety substantiating that NH is not a psychogenic disorder (24). Coexistent conditions such as primary headaches (migraine and tension-type headache), brain trauma, intracranial mass lesions, arachnoid cysts have been already described (1,4,5,14-16,24). Menstrual related NH is also described accentuating the role of estrogen affecting both peripheral and central nociception (25). A high prevalence of autoimmune diseases such as Sjogren syndrome, Sicca syndrome, romatoid arthritis, antiphospholipid antibody syndrome in NH patients are reported earlier(26). Probably NH spectrum is larger as seen by increasing number of symptomatic cases.

Our case was diagnosed as MS and complained of migraine without aura. MS lesions in the midbrain and disease-modifying agents may cause migraineous headaches. However for the past 5 months, she suffered from a different headache in a circumscribed area of 4 cm in diameter in the left posterior parietal region. This pain was different from migraine and only responded to topira-

mate. Several therapeutic interventions for NH are discussed in the literature, such as gabapentin, indomethacin, topiramate, tricyclic antidepressants, carbamazepine,beta blocker,local injections for nerve block, botulinum toxin type A injections, transcutaneous electrical nerve stimulation and surgical intervention, but still there is no standard treatment for NH (13-17,27-31).

The neurological examination is normal in NH patients. However bitrigeminal hyperalgesia is reported in one case where there was no underlying disease (27). The exact etiology of NH is not clear. Possible mechanisms proposed are a neuropathy of a terminal branch of a cutaneous nerve and a focal nociceptive-type pain originating from epicranial tissues (3). In a previous study, increased pain sensitivity, decreased pressure pain threshold is found in that localized area and underlined a peripheral origin (32). In autoimmune disorders such as Sjogren syndrome and antiphospholipid antibody syndrome, autoimmune-associated distal sensory axonopathy is suggested as the underlying mechanism of NH (27). Parietal region is commonly involved in NH which is innervated by ophtalmic branch of trigeminal nerve, thus a neuropathy of the ophthalmic branch of trigeminal nerve can be the cause in most cases. A central mechanism of pain has also been proposed for NH (11). Guillem et al pointed out a concept of circumscribed pain arising from intracranial pain-sensitive structures such as meningeal arteries and dural sinus in NH secondary to arachnoid cysts (21). In our patient probably NH was triggered by involvement of the trigeminal nucleus or the trigeminothalamic tract by demyelination. Although in some cases NH can be seen secondary to brain trauma, intracranial mass lesions, arachnoid cysts and autoimmune disorders (26,33), to our knowledge, NH secondary to MS has not been previously reported.

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LESIONS MIMICKING TRANSVERSE MYELITIS

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Abstract

Transverse myelitis (TM) is a rare, acquired focal inflammatory disorder often presenting with rapid onset weakness, sensory deficits and bowel/bladder dysfunction. Generally occurring independently as a complication of infection; however, it may also exist as part of a continuum of other neuro-inflammatory disorders. The list of differential diagnoses of TM is long; hence, a meticulous history and detailed physical examination are indispensible. Despite the improvement of ancillary diagnostic techniques, including neuroimaging and serological testing, the correct diagnosis of acute and subacute myelopathy is challenging. A systematic, careful history and may help exclude other mimics of TM. In this article, we aimed to evaluate different etiological diagnosis in patients who presented with TM clinic.

Introduction

Transverse myelitis (TM) includes a pathobiologically heterogeneous syndrome characterized by acute or subacute spinal cord dysfunction resulting in paresis, a sensory level and autonomic (bladder, bowel, and sexual) impairment below the level of the lesion. Etiologies for TM can be broadly classified as parainfectious, paraneoplastic, drug/toxin-induced, systemic autoimmune disorders (SAIDs) and acquired demyelinating diseases like multiple sclerosis (MS) or neuromyelitis optica (NMO) (1). Patients with isolated TM present a diagnostic dilemma, as it is common in both MS and NMO, but may also be the initial manifestation of SAIDs. Also, there are noninflammatory etiologies (eg, vascular, metabolic) that may mimic the clinical and radiologic appearance of TM. The cause of TM remains unknown despite an extensive workup in about 15% to 30% of patients and is therefore referred to as "idiopathic" according to set criteria (2,3).

The annual incidence of TM ranges from 1.34 to 4.60 cases per million, but increases to 24.6 cases per million if acquired demyelinating diseases like MS are included. TM can occur at any age, although a bimodal peak in incidence occurs in the second and fourth decades of life (4). Half of patients have an antecedent

infection. The list of differential diagnoses of TM is long; hence, a meticulous history and detailed physical examination are indispensible. A systematic and careful history may help exclude other mimics of TM. Magnetic resonance imaging (MRI) of the complete spinal axis is mandatory in any patient with myelopathic features to exclude structural lesions. Treatment is directed towards the cause.

Methods

We retrospectively studied 9 consecutive patients with clinical evidence of TM. All patients underwent spinal MRI, 4 underwent cerebrospinal fluid analysis. Solid lesions were diagnosed with biopsy. Various other studies were performed to assess for connective tissue disease and causes of TM other than demyelinating disease. The inclusion criteria were as follows: patients with sensory defect giving level or deep sensory loss. Exlusion criteria included patients aged<18 years, pregnant women, who cannot have MRI. The lesion was visualized by MRI in all patients. The variables of interest were patient demographic characteristics (age,gender), neurologic examination, MRI findings and definitive diagnosis.

Results

Different etiological reasons were found in the evaluation of 9 patients who applied with the TM clinic. 4 patients are male and 5 patients are female. The lesion was detected in the thoracic region in 7 patients and in the cervical cord in 2 patients. While all patients with thoracic lesion had sensory defect giving level and deep sensory loss was detected in 2 patients with cervical lesion. 3 patients had paraparesia and 1 patient had monoparesia. The lesion was visualized by MRI in all patients. For the differential diagnosis, further investigations were made and a definitive diagnosis was made. Differential diagnoses of TM and cases features are shown in the table.

PATIENT	AGE	GEN-DER	SYMPTOM	MR LESION	DIAGNOSIS
1	56	F	T4 sensory defect that giving level- para-paresia	T6-7 lesion	TM
2	55	F	T4 sensory defect that giving level -para-paresia	Lesion extending from the cranio-cervical junction to the upper thoracic cavity.	NMO
3	36	F	T4 sensory defect that giving level -para-paresia	T1-4 multipl MS lesions	MS
4	86	F	Deep sensory loss	C3-6 dorsal cord lesion	SCD-B12
5	37	M	T5 sensory defect that giving level	T5 lesion	TM-COPPER
6	55	M	Parestesia	C2-5 dorsal cord lesion	TSCI
7	64	F	Left T8-9 sensory defect that giving level-left leg mono-paresia	Left T8-9 subdu-ral lesion	MENINJIO-MA
8	73	M	T4 sensory defect that giving level	T2-4 solid lesion	TUMOR
9	36	M	T4 sensory defect that giving level	T3 lesion	METASTASIS

Table: Differential diagnoses of TM and cases features

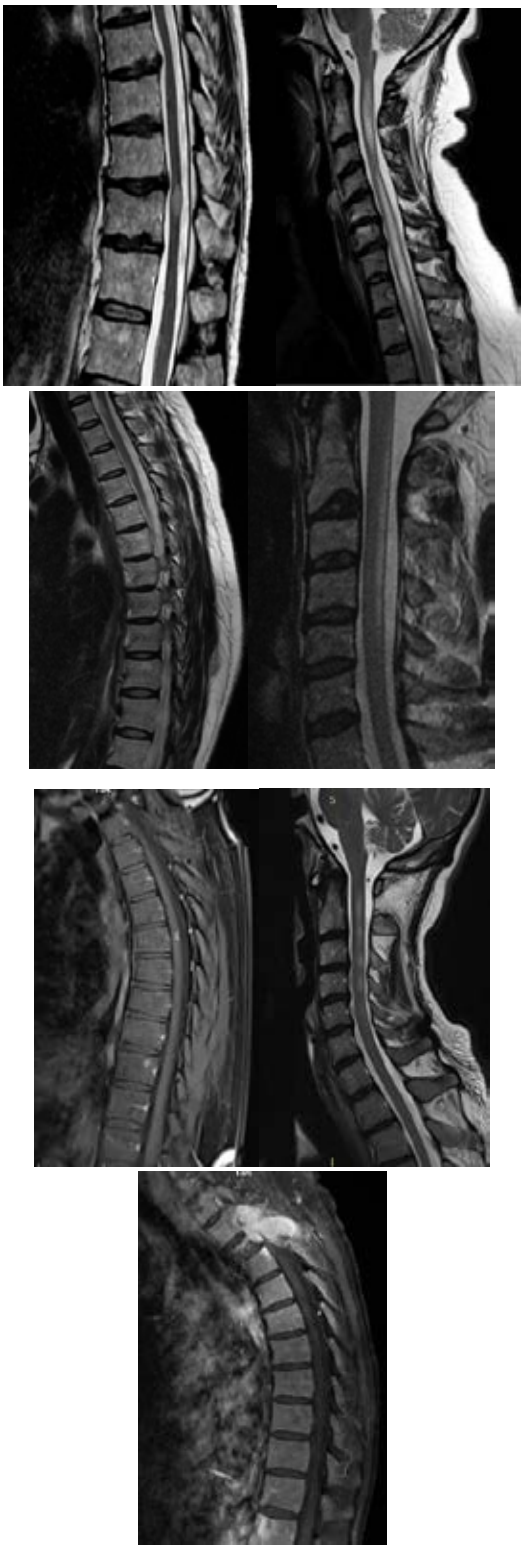

Table 2: MR lesions of patients 1:TM 2: NMO 3:MS 4:SCD 5:TM 6:TSCI 8:TUMOR

Discussion

TM typically has an acute to subacute onset with neurologic deficits reaching within a few weeks. TM may present as one of several syndromes of the spinal cord. Acute complete TM (ACTM) manifests as paresis/plegia, sensory dysfunction

(characterized by numbness, paresthesias, or other manifestations in conjunction with a sensory level) and autonomic impairment below the level of the lesion. Acute partial TM (APTM) results in asymmetric manifestations or deficits specific to particular anatomic tracts; manifestations include the hemi-cord (Brown-Sequard), central cord, or posterior column syndrome, as well as selective tract impairment. The list of differential diagnoses of TM is long; hence, a meticulous history and detailed physical examination are indispensible. A systematic and careful history may help exclude other mimics of TM. Acute transverse myelitis is a subgroup of various conditions characterized by focal inflammation of the spinal cord and resultant neural injury. The etiologies of myelopathies are varied and can be subdivided into compressive and noncompressive causes. While compressive myelopathies stem from trauma and intra or extraspinal tumors, the etiologies of non-compressive myelopathies can be classified as delayed radiation effects, ischemic, paraneoplastic, systemic ADs and infectious or parainfectious. Cerebrospinal fluid (CSF) analysis is essential in the evaluation of TM. CSF cell count, differential, protein, glucose, oligoclonal bands (OCBs) and IgG index should be checked on all patients with TM. MRI of the complete spinal axis is mandatory in any patient with myelopathic features to exclude structural lesions [3]. In this report; the lesion was detected in the thoracic region in 7 patients and in the cervical cord in 2 patients. While all patients with thoracic lesion had sensory defect giving level and deep sensory loss was detected in 2 patients with cervical lesion presented with acute complete TM symptoms. All patients were diagnosed with MRI. CSF examination was performed in some patients for differential diagnosis and blood tests (B12 level, thyroid function tests, syphilis, HIV serologies and inflammatory markers) that may cause myelitis were requested.

NMO usually causes attacks of severe AON (sometimes bilateral) and brainstem lesions resulting in intractable nausea, vomiting or hiccups (5,6). Although the manifestations of NMO may be similar to MS, attacks are typically more devastating. Serum aquaporin-4 specific autoantibodies (NMO-immunoglobulin Ig G) should be checked on all patients with TM because of its high specificity for NMO or NMO spectrum disorders (NMOSD) (7). NMO-IgG sero-positivity is rarely found in patients with APTM but its presence would have profound implications on treatment. Inflammatory markers should be checked if SAID is suspected (8). NMO is diagnosed on the basis of the revised Wingerchuk criteria requiring the presence of optic neuritis and TM as well as 2 of 3 of the following: NMO antibodies, LETM, and/or brain MRI lesions inconsistent with MS. In making the diagnosis of TM, it is essential to remember that many noninflammatory etiologies may mimic the appearance of TM. Recognizing these entities is important, as the treatment and management strategies would be vastly different (9). Patient with number 2 presented with loss of strength in the legs. A lesion compatible with NMO was detected in thoracic MRI. Cranial MRI, NMO antibodies and CSF examinations performed afterwards confirmed the diagnosis of NMO.

MS is a disabling progressive neurologic disorder affecting approximately 400,000 people in the United States (10). TM in MS most commonly presents with sensory phenomena. Spine MRI typically reveals an asymmetrically placed lesion (usually occurring in the posterolateral or lateral portion of the spinal cord) less than 2 segments in length with a predilection for the cervicothoracic cord. White matter lesions predict higher risk of conversion to MS (with rates of up to 88% reported). If the lesions meet at least 3 of the Barkhof criteria this risk is increased substantially (11). The third patient presented with loss of strength in the legs and sensory defect that gives level. In the thoracic MRI, the lesions were found to be compatible with MS. Afterwards, a definitive diagnosis of MS was made with cranial MRI and CSF examination.

Serum vitamin B12 level, thyroid function tests, syphilis and HIV serologies always should be obtained to evaluate for potentially treatable causes of myelopathy. Vitamin E, serum copper and ceruloplasmin levels are checked in those at risk of deficiency. Subacute combined degeneration (SCD) of the spinal cord is the most common neurologic manifestation of vitamin B12 (cobalamin) deficiency and is usually secondary to autoimmune gastritis, but may also be seen in malnutrition syndromes such as chronic alcoholism, strict vegetarianism, gastrectomy and also in nitrous oxide abuse. Although traumatic spinal cord injury is routinely encountered in the medical examiner's office, medical causes of spinal cord abnormalities such as SCD should be considered in the appropriate clinical setting (12). Patient with number 4 was admitted with the complaint of imbalance due to profound sensory loss. A lesion compatible with SCD was observed in thoracic MRI. Therefore, the reasons that could cause SCD were investigated and B12 deficiency was detected in this patient.

Copper deficiency myelopathy represents an often underdiagnosed, acquired neurological syndrome, clinically characterized by posterior column dysfunction. The main causes of copper deficiency are bariatric surgery, increased consumption of zinc, and malabsorption. However, even after a careful history taking and extensive laboratory researches, the etiology of copper deficiency remains undetermined in a significant percentage of cases. Patients affected by copper deficiency myelopathy usually present with sensory ataxia due to dorsal column dysfunction and sometimes with mild leg spasticity. In such patients, spinal cord MRI may show hyperintense lesions in T2-weighted sequences involving the posterior columns of cervical and thoracic cord (13). Patient with number 5 presented with profound sensory loss and leveling sensory defect. A myelitis compatible lesion was detected in the thoracic MRI. Since type 4 OCB was positive in the CSF examination, metabolic causes were investigated and copper deficiency was detected.

Traumatic spinal cord injury (TSCI) is associated with higher morbidity, affecting young individuals causing severe and permanent disabilities that incur huge healthcare burden on the patients. TSCI is a clinicoradiological diagnosis of post-traumatic neurological weakness with bony deformity based on radiography and computed tomography and confirmed by MRI (14). Spinal cord injury without radiographic abnormality (SCIWORA) is a recognized form of SCI, infrequently reported

in adults and characterized by the absence of any radiographically evident fracture or dislocation (15). It is defined as spinal cord lesions that are observed by MRI but do not show lesions in plain radiography. The reported incidence of SCIWORA among adult SCI cases ranges from 10% to 12%,7 and falls from height, motor vehicle accidents, and sports-related injuries are the major injury mechanisms. The epidemiology and pathophysiology of SCIWORA differ among adults and children, with most adult patients presented with radiographic abnormalities showing degenerative changes (16,17). The patient with number 6 applied with the complaint of numbness in the whole body prominent in the arms. Posterior cord lesion was observed in Cervical MRI. Since the patient's complaints started after falling from a height, a diagnosis of SCIWORA was made.

Spinal meningiomas are found in all age groups, predominantly in women aged over 50 years. Their incidence is approximately 3 per 100.000. The clinical symptoms of this condition may range from mild to significant neurological deficit, varying widely depending on the location, position in relation to the spinal cord, size and histological type of the tumor. MRI is the diagnostic tool of choice because it shows the location, size, the axial position of the tumor, and the presence of concomitant conditions such as spinal malformations, edema or syringomyelia. The goal of the surgery is total resection which is achievable in 82%-98% of cases. Advances in radiosurgery have led to its increased use as primary or adjunct therapy (18,19).Seventh Patient applied with monoparesia. A lesion compatible with meningioma was detected in Thoracic MRI. The patient was treated surgically.

Intramedullary tumors represent only 10% of the spectrum of spinal tumors as these lesions develop in 60% of the cases on the epidural aspect and are in 30% of the cases intradural and extramedullary. They are mostly glial tumors. Ependymomas and lowgrade astrocytomas are the leading histological types. Their management is not an easy task and depends essentially on the surgical resection which can be very challenging. Adjuvant therapies have a very controversial role. They represent 30% of intramedullary tumors. They mostly affect children and usually are of low grade. Cervical and thoracic localizations are the most frequent. Astrocytomas are usually not well limited; they are mostly infiltrative. They present a tumoral cyst in 48% of the cases. Hemorrhage is less frequent than ependymomas. Clinical presentations join those of ependymoma; it is usually progressive with motor and sensitivity troubles. They present on the MRI as a noncentral infiltrative tumor enlarging the spinal cord. They are hypointense on T1 and hyperintense on T2. Enhancement is heterogeneous and has no correlation to the grade of the tumor contrarily to cerebral astrocytomas. Leptomeningeal enhancement can orient for highgrade lesions (20,21). Patients 8 and 9 presented with sensory defect that gives level. Solid lesion was detected in thoracic MRI in both of them. Both patients were diagnosed with biopsy. One had primary spinal cord tumor and the other had metastasis.

Conclusion

Differential diagnoses of acute/subacute intrinsic spinal cord lesions includes demyelinating, inflammatory or infectious diseases, tumors and metabolic deficits. Despite the improvement of ancillary diagnostic techniques, including neuroimaging and serological testing, the correct diagnosis of acute and subacute myelopathy is challenging. In general, MRI findings of acute myelitis may be multifocal and/or longitudinally extensive, as in neuromyelitis optica, where hyperintensity in T2 weighted images characteristically spans over three or more contiguous vertebral segments. On the other hand, intramedullary neoplasms typically show gadolinium enhancement, mass effect and cord expansion. SCD of the spinal cord is the most common neurologic manifestation of vitamin B12 and copper deficiencies. SCD can be completely cured with early treatment. TSCI and SCIWORA can effect the spinal cord by mimicking SCD.

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