Kumandaş & Canpolat

Pediatrik Nöroloji

Algoritmalar ve İlaç Rehberi

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Pediatrik Nöroloji: Algoritmalar ve İlaç Rehberi

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SUNUŞ

Çocuk nöroloji hastasına tanı koyma ve istenecek tetkikler konusunda çoğu zaman zorlanmaktayız. Çünkü, farklı hastalıklar çocuklarda aynı semptomlarla seyredebilmektedir. Hastanın acil tedavisinin yapılabilmesi için tanının konulması ve tedavilerinin ona göre planlanması gerekmektedir.

Hastaların tanısında fizik muayane, nörolojik bulgular yanında biyokimyasal ve genetik tetkikler, bazen de görüntüleme bulguları bize yardımcı olmaktadır. Bu bağlamda istenecek tetkiklerin yanında, tetkiklerin istenme sırasının da bir algoritma dahilinde yapılması gerekmektedir.

Pediatrik nöroloji hastalarının tanı ve tedavisinin erken başlanması konusunda bizlere bir yol haritası sunan "Pediatrik Nöroloji Algoritmalar ve İlaç Rehberi" adlı bu kitabın çocuk nöroloji doktorlarına, çocuk hekimlerine ve acil tıp hekimlerine faydalı bir eser olacağını umuyoruz.

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Saygıdeğer meslektaşlarım;

Türkiye'de çocuk nörolojisi bilim dalları ilk kez 1963 yılında Hacettepe Tıp Fakültesinde kurulmuş olup daha sonra 1966 yılında İstanbul Üniversitesi İstanbul Tıp Fakültesi ve 1972 yılında Ege Üniversitesi Tıp Fakültesinde kurulmuştur. Bu üniversitelerden yandal ihtisası alan değerli çocuk nörolojisi hocalarımız Türkiye'nin çeşitli tıp fakültelerinde ve Sağlık Bakanlığının eğitim kurumlarında çocuk nörolojisi bilim dallarını kurmuş ve yeni çocuk nörologları yetiştirmişlerdir.

Bugün itibarıyla çocuk nörologlarının sayısı yaklaşık 400 kişiye ulaşmış olup büyük bir çocuk nörolojisi ailesi olarak Türkiye'ye hizmet vermekteyiz.

Bu bağlamda ben de 1993-1996 yılında İstanbul Üniversitesi İstanbul Tıp Fakültesinde yandal ihtisasımı tamamlayıp 1997 yılında Erciyes Üniversitesi Tıp Fakültesinde Çocuk Nörolojisi Bilim Dalını kurdum ve onlarca çocuk nöroloğu yetiştirdim. Bilim dalı olarak 24 yıl süreyle yüz binlerce çocuk nörolojisi hastasına hizmet verdik. On bir yıl Türkiye'nin değişik tıp fakülteleri ve devlet hastanelerinde pratisyen doktor ve Çocuk Sağlığı Hastalıkları uzmanı olarak çalıştım ve son 30 yılımı akademik kadroda hizmet verdiğim Erciyes Üniversitesi Tıp Fakültesinden 2 yıl önce emekli oldum. Hâlen serbest hekim olarak çocuk nörolojisi hastalarına hizmet vermekteyim.

Bizlerin yetişmesinde emeği geçen ve ikinci baharını yaşayan saygı değer hocalarımıza "Bana bir harf öğretenin kölesi olurum mantığı ile" saygı, minnet ve şükranlarımı sunuyor, ebediyete intikal edenlere Allah'tan rahmet diliyorum.

Bu eserin hazırlanmasında emeklerini esirgemeyen değerli bilim insanları arkadaşlarıma, yayınevi çalışanlarına ve emeği geçen herkese teşekkürlerimi bir borç bilirim.

Bu eserin çocuk doktorları, acil hekimleri, yoğun bakım doktorları, erişkin nörologları ve çocuk nörologlarına faydalı olacağını ümit ediyorum.

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"Kumandaş & Canpolat Pediatrik Nöroloji: Algoritmalar ve İlaç Rehberi" kitabının 1. baskısında yer alarak katkı sağlayan değerli hekimlerimize, bilim insanlarımıza, Akademisyen yayın evinin çok değerli yönetici ve çalışanlarına teşekkürlerimi sunuyorum. Bu kitabın Çocuk Sağlığı ve Hastalıkları Anabilim Dalı araştırma görevlilerinin, Çocuk Sağlığı ve Hastalıkları uzmanlarının, Çocuk Nörolojisi Bilim Dalı yan dal araştırma görevlilerinin ve Çocuk Nörolojisi uzmanlarının hem eğitim süreçlerine katkı sağlayacağını hem de günlük hasta pratiğinde bir başucu kitabı olacağını umut ediyorum. Bu eser, bizleri yetiştiren Türk Milletine ve Ailelerimize karşı olan vefa borcu ve şükranlarımızı bir nebze de olsa sunma imkânı vermiştir. Türkiye Cumhuriyeti'nin kuruluşunun 100. yılı olan 2023'e emin ve kararlı adımlarla ilerlerken, kitabımızın 1. baskısının "Beni Türk Hekimlerine Emanet Ediniz" diyen Cumhuriyetin kurucu lideri Gazi Mustafa Kemal ATATÜRK'ün bizlere emanetleri olan ülkemiz çocuklarını"Her bir Türk evladının, birer Türk Bayrağı olduğu" ülküsü, hekimlik mesleğinin evrensel ilkeleri ve modern tıbbın bilimsel gereklilikleri doğrultusunda tedavi edecek kıymetli hekimlerimize hayırlı olmasını temenni ediyor, Cumhuriyetimizin 99. yılını kutluyor ve bu eseri Türkiye Cumhuriyeti'nin 100. yılına ithaf ediyorum.

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12. Ajitasyonun Yönetimi

Ajitasyon kafa içi basıncını artırabilir ve solunumu kontrol etmeyi zorlaştırabilir. Sedasyon kararı, hastanın durumundaki değişikliğinin takibinde seri nörolojik muayeneyi zorlaştırabilir. Bu durumlarda EEG takibi değerli olabilir.

13. Kronik Bozuklukların Tedavisi

Daha önce bahsedilen yönetim ilkeleri, komadaki hastaların akut bakımı ile ilgilidir, ancak ilkelerin çoğu, bilincini tam olarak geri kazanamayan ve uzun vadeli fonksiyonel engelleri olan hastalar için de geçerlidir. Bundan sonraki basamak nörorehabilitasyonun genel ilkeleri ve daha özel olarak kaza sonucu ve kazara olmayan travmatik beyin hasarından ve global hipoksik-iskemik beyin hasarından sonraki yönetimi içermelidir.

Uygun klinik deneylerden geçmiş ve daha sonra bilinç bozukluğu olan çocuklarda kullanım için onaylanmış hiçbir farmakolojik ajan yoktur. Yetişkinlerde, amantadin ve zolpidemin farkındalığı artırdığı gösterilmiştir. bilinç bozukluğu olan yedi çocuğu içeren küçük bir vaka serisi rapor edilmiş ve güvenliğini göstermiş ancak etkinlik göstermemiştir. Nöromodülasyon, artan ilgi gören başka bir yaklaşımdır.

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BT ANJİOGRAFİ (BTA) VE MRA

BTA ve MRA noninvaziv teknikler olup endikasyonları DSA endikasyonları ile benzerdir. BTA'nın DSA'ya üstünlüğü hızlı ve kolay olmasıdır. BTA, tanısal BT'den hemen sonra elde olunabilir. Acil durumlarda MRA'ye kıyasla daha pratiktir. Özellikle hematom boşaltılması için acil kraniotomi yapılması gereken hastaların görüntülenmesinde hızlı olması nedeniyle tercih edilir. Bununla birlikte BTA için yeterli zaman bulunmaması durumunda acil cerrahi geciktirilmemelidir. BTA, MRA'den daha hızlı olup büyük çocuklarda sedasyon gerektirmez. MRA ise genellikle sedasyon ihtiyacı duyulan küçük çocuklarda ionizan radyasyon içermemesi nedeniyle tercih edilmektedir.[3]

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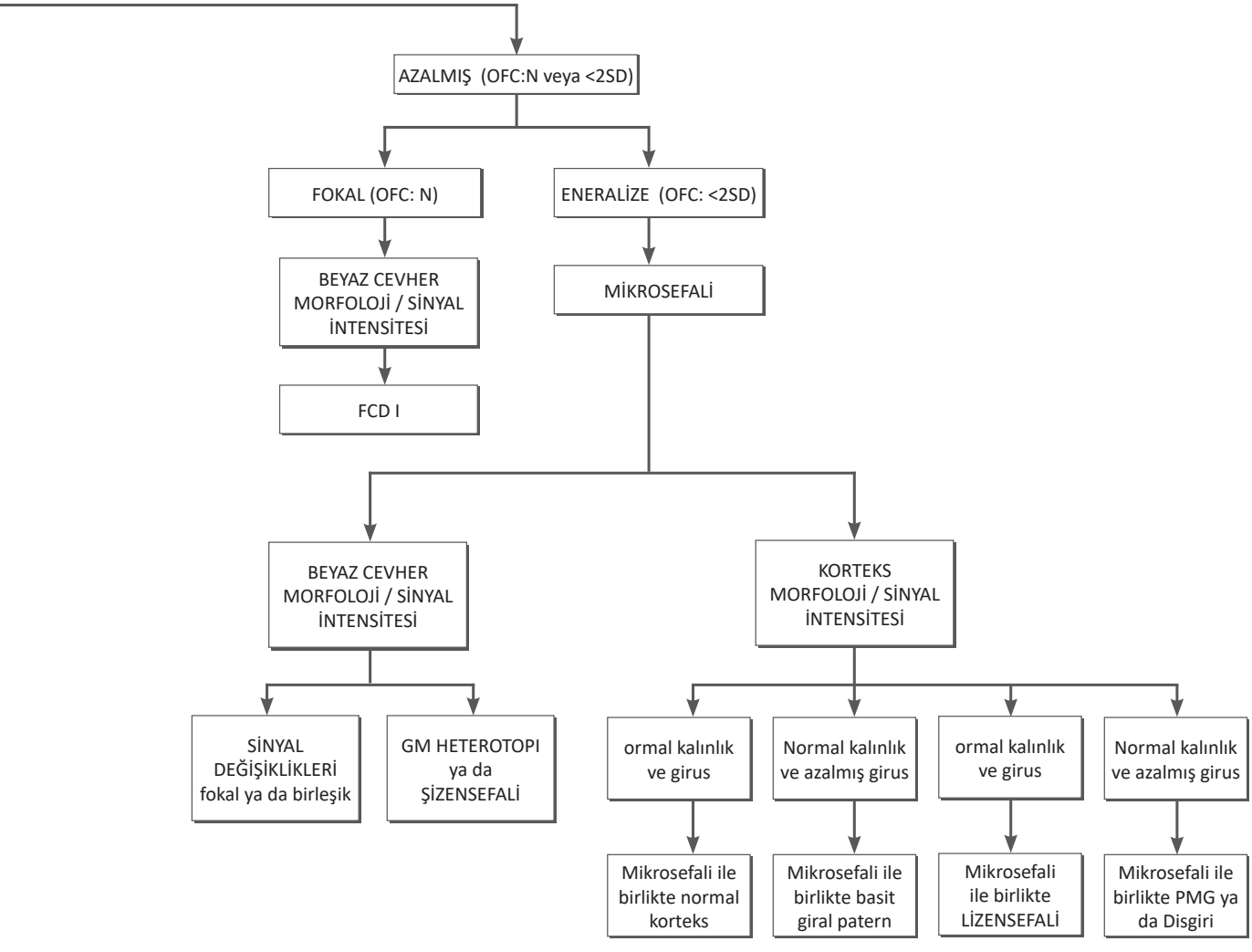

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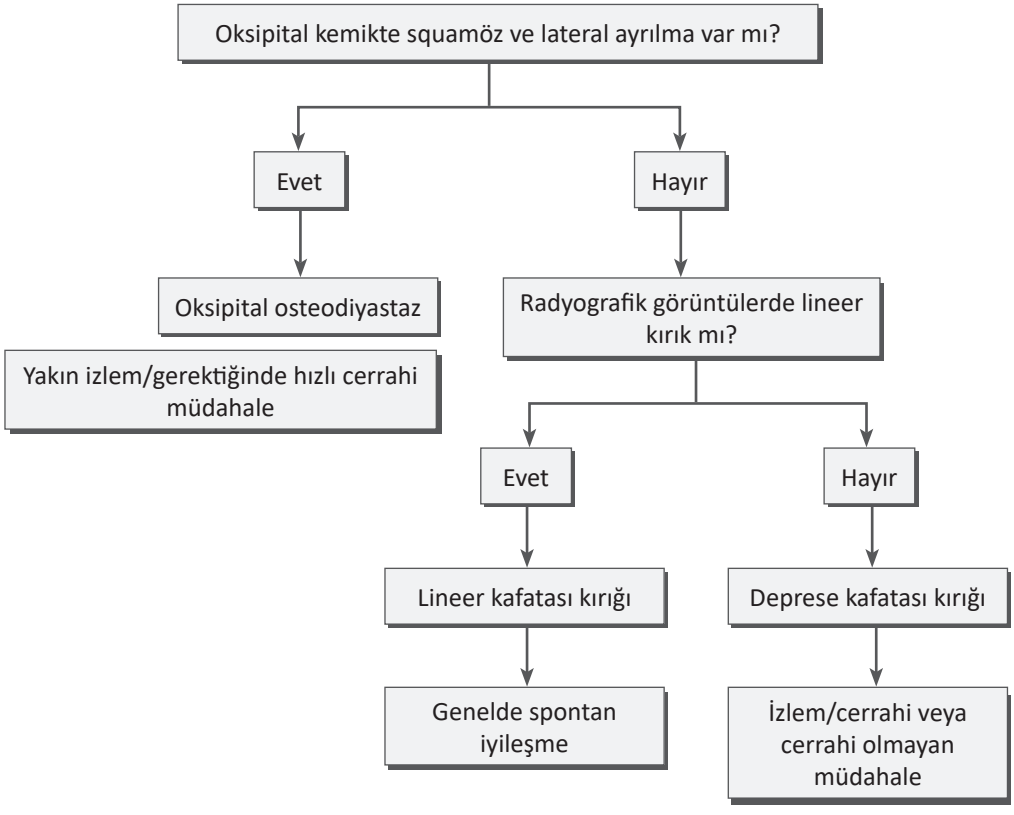
KAFATASI KIRIKLARI



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Tablo1: Araknoid Kistin Spesifik ve Non-spesifik Semptomları

Spesifik nörolojik semptom	Non spesifik semptom
Fokal nöbet	Baş ağrısı
Görme bozukluğu	Bulantı/kusma
Nistagmus	Baş dönmesi
İşitme Kaybı	Ataksi
Konuşma Bozukluğu	Jeneralize nöbet
Servikal Miyopati	Kognitif bozukluk
Fasial paralizi	

Tablo 2: Tipik ve Atipik Kistin MRG Özellikleri

MR bulguları	Tipik Kist Özellikleri	Atipik Kist Özellikleri
Sayı	Tek	Çok
Sınıflandırma	Ekstra aksiyal	İntra aksiyal
Yer	Orta fossa, retoserebellar, konveksite	Serebolopontin bileşke, subaraknoid sisterna
Boyut	<2,5cm	>2,5cm BOS dansitesi ile uyumsuz
Dansite	BOS ile izointens	Kalın, düzensiz sınırlı
Duvar Kalınlığı	İnce, düzgün sınırlı	Olabilir
Kanama	Hayır	Olabilir
Bası	Hayır	

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KRANİOSİNOSTOZ RADYOLOJİK TANI ALGORİTMASI[2]

(KS: Kraniosinostoz , BT: Bilgisayarlı tomografi, US: Ultrasonografi, MRG: Manyetik rezonans görüntüleme, MRG* : black bone MRG)

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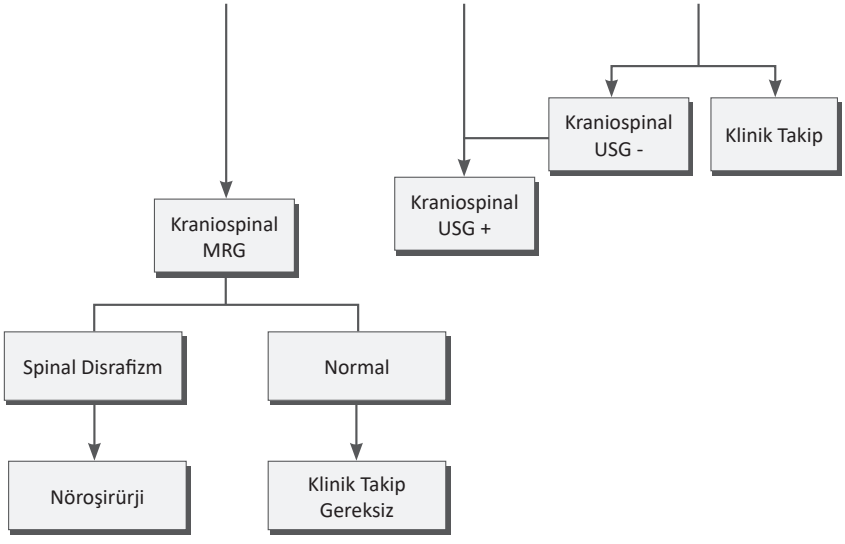

VACTERL: vertebral defektler, anal atrezi, kardiyak defekt, trakeoözefagial fistül, renal anomaliler, ekstremite anomalileri
OEIS: omfalosel, ekstropi, imperforat anüs, spinal disrafizm
USG: ulstrasonografi
MRG: manyetik rezonans görüntüleme

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Hipoksik-iskemik ensefalopati tanı kriterleri *

1. **Bebek NE tanımına uyuyor mu?**
   35+ doğum haftası, anormal bilinç değişikliği ya da solunumu başlatmada ve sürdürmede zorluk, nöbet, anormal tonus/refleks varlığı

- **Aşağıdaki bulgular/akut olaylar eşlik ediyor mu?**
  - Apgar skoru<5 (5. ve 10. dakika)
  - Umbilikal kord kan gazında pH<7, veya baz açığı ≥ 12
  - Nörogörüntülemede akut beyin hasarı (Beyin manyetik rezonans görüntüleme (MRG) veya manyetik rezonans spektroskopide (MRS) derin gri cevher veya kanlanma sınırlarında zedenlenme bulguları)
  - HİE ile ilişkili çoklu organ yetmezliği varlığı (Renal, pulmoner, kardiyak, hematolojik, metabolik)
- **Akut peripartum-intrapartum olay eşlik ediyor mu?**
  - Doğum sırasında hipoksi veya iskemiye neden olabilecek patolojiler (uterus rüptürü, maternal hipoksemi, maternal kardiyovasküler kollaps, ablasyo plasenta, vasa previa ya da feto-maternal kanama olması, kord prolapsusu, maternal hipotansiyon, amniyon sıvı embolisi)
  - Fetal kalp hızı paterninin kötüleşmesi: tekrarlayan geç veya değişken deselerasyonlar veya bradikardi veya ≥20 dakika sinüzoidal patern olması
  - Ensefalopatiye yol açabilecek diğer etkenlerin yokluğu (neonatal sepsis, anormal fetal büyüme, maternal enfeksiyonlar, kronik plasental lezyonlar)

Terapötik hipotermi tedavi kriterleri **

- Klinik olarak orta veya ağır ensefalopati bulgularının olması
- Gebelik yaşının ≥36 hafta olması ve postnatal yaşın ≤6 saat olması
- Kord kan gazında ya da doğumdan sonraki ilk 60 dakika içerisinde bakılan pH ≤7 ya da BE ≤-16 mmol/L olması
- 10. dakika Apgar skoru ≤5 olması veya devam eden resusitasyon ihtiyacı
- aEEG'de anormal zemin aktivitesi veya nöbet görülmesi

Akisu M, Kumral A, Canpolat FE. Türk Neonatoloji Derneği yenidoğan ensefalopati rehberi. Türk Pediatri Arşivi. 2018;53(Supp:1):32-44.

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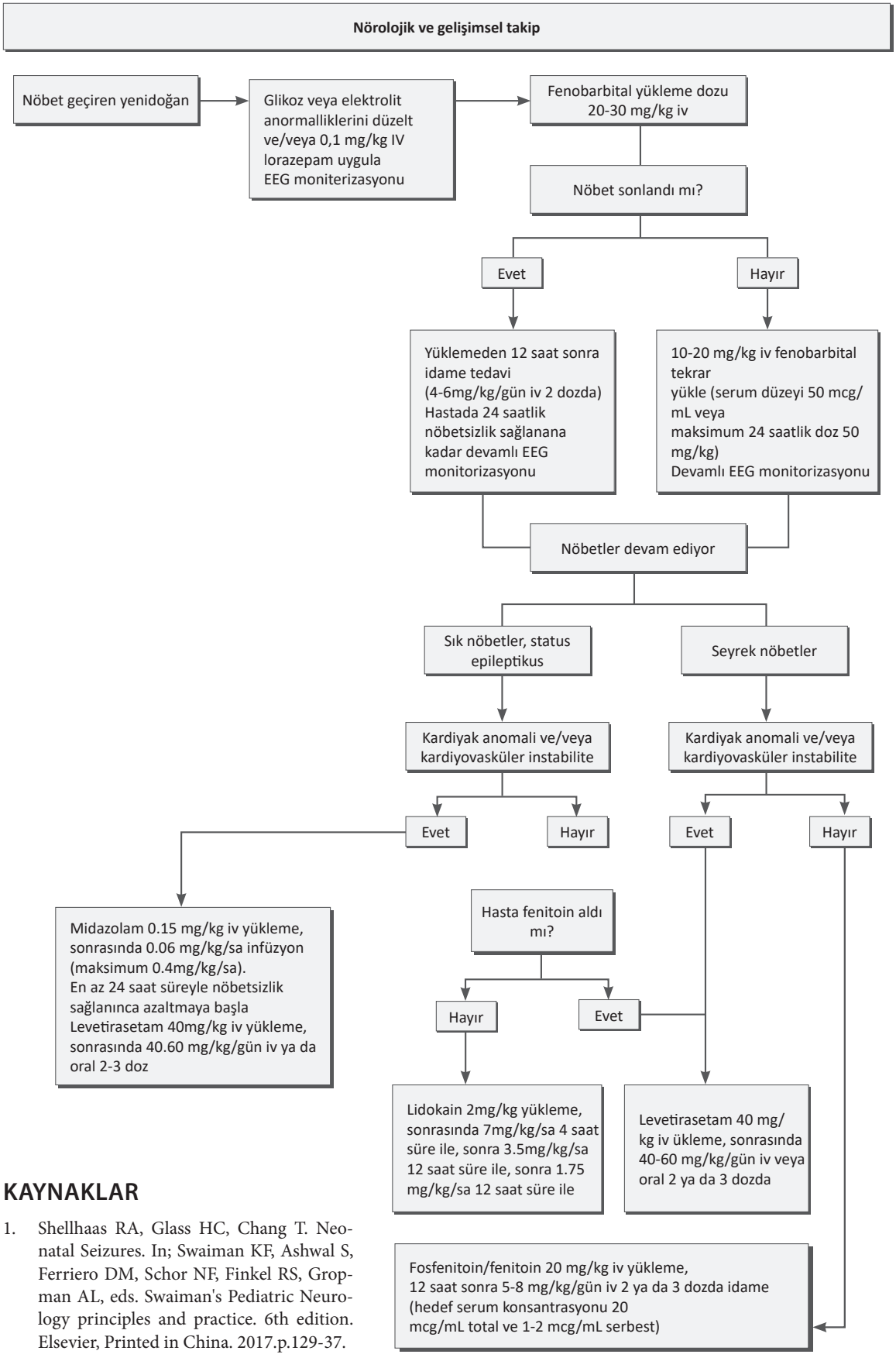

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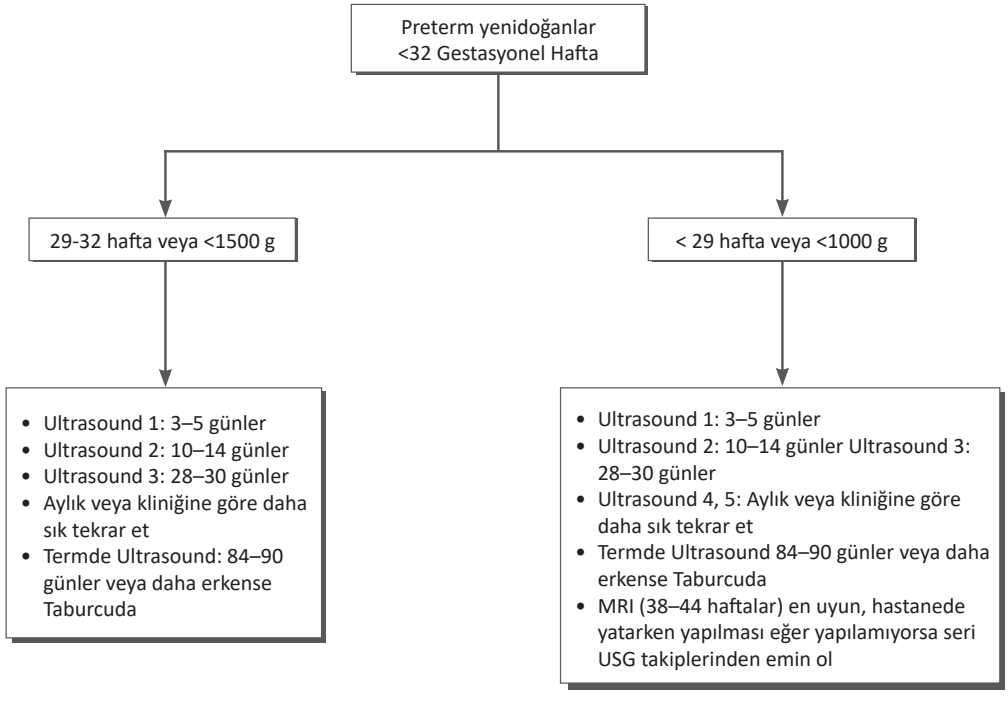
ASEMPTOMATİK PRETERM YENİDOĞANLARA YAKLAŞIM ALGORİTMASI

BÖLÜM 19

Tamer GÜNEŞ [1]



Şekil 1. Preterm bebekler için beyin görüntüleme algoritması

Bu algoritma asemptomatik preterm yenidoğanlar için geçerlidir. Ancak, klinik endikasyon varsa, MRI gebelik yaşına bakılmaksızın herhangi bir preterm yenidoğan için düşünülür. Örnek olarak:

- Fizik muayenede anormal bulgular: Fokal nörolojik anomaliler veya hipotoni.
- Nöbet/anormal davranışların varlığı veya seri ultrasound 'ta ekolüsensler.
- Prenatal beyin anomalisi varlığı
- Hipoksik iskemik ensefalopati.
- Galen anomalisi veya metabolik bozuklukların şüphesi.
- Açıklanamayan apneler, bradikardiler veya desatürasyonlar
- Posterior fossa lezyonları veya sereberlar kanama.
- Sepsis
- Nekrotizan enterokolit.

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TRAVMATİK PERİFERİK SİNİR YARALANMALARINDA ALGORİTMA

Periferik Sinir Yaralanmalarının Değerlendirilmesi

- Ağrı
- Eklem hareket açıklığı
- Ödem
- Kas gücü
- Duyu
- Otonomik fonksiyon

Periferik Sinir Yaralanmalarının Değerlendirilmesinde Kullanılan Tanısal Testler

- EMG, Ultrason, Magnetik rezonans görüntüleme
- Periferik sinir yaralanmalarının değerlendirilmesinde kullanılan sınıflandırmalar.
- Periferik sinir yaralanmalarının değerlendirilmesinde Seddon sınıflandırması ve Sunderland sınıflandırması kullanılmaktadır.

Tablo 1: Periferik sinir yaralanmalarının değerlendirilmesinde kullanılan sınıflandırmalar[2]

Seddon	Süreç	Sunderland
1-Nöropraksi	• Periferik sinir üzerine yapılan baskı nedeniyle sinir iletimi veya impulsların geçişinin kısa süreli kesilmesiyle oluşan sinir hasarıdır. • Dejenerasyonun olmadığı bir lezyondur. Schwann hücresi ve akson sağlam olduğundan akson devamlılığı korunur. • Duyu kaybı kısmen görülebilir. Baskı ortadan kalktığı zaman tam iyileşme görülür.	1. Derece
2-Aksonotmezis	• Akson ayrılmış -endonöryum sağlam • Rejenerasyon için uygun, iyileşme tam	2. Derece
Aksonotmezis	• Akson ,endonöryum etkilenmesi var, • Perinöryum ve fasiküller korunmuş • İyileşme tam olmaz	3. Derece
Aksonotmezis	• Akson ,endonöryum tüp, perineurium ve fasikül sürekliliği kaybolmuş, • Epinöryum korunmuş • Geniş skar alanı, restorasyon sürekliliği için cerrahi gerekli	4. Derece
3-Nörotmezis	• Sinirin tam olarak zedelenmesine neden olan travmalar sonucunda oluşur. • Akson, Schwann hücresi ve dış yapılar tamamen hasarlanmıştır. • Tam kesi olmuştur • Yenilenme çok zayıftır. • Cerrahi girişim olmadan spontan iyileşme gözlenemez.	5. Derece

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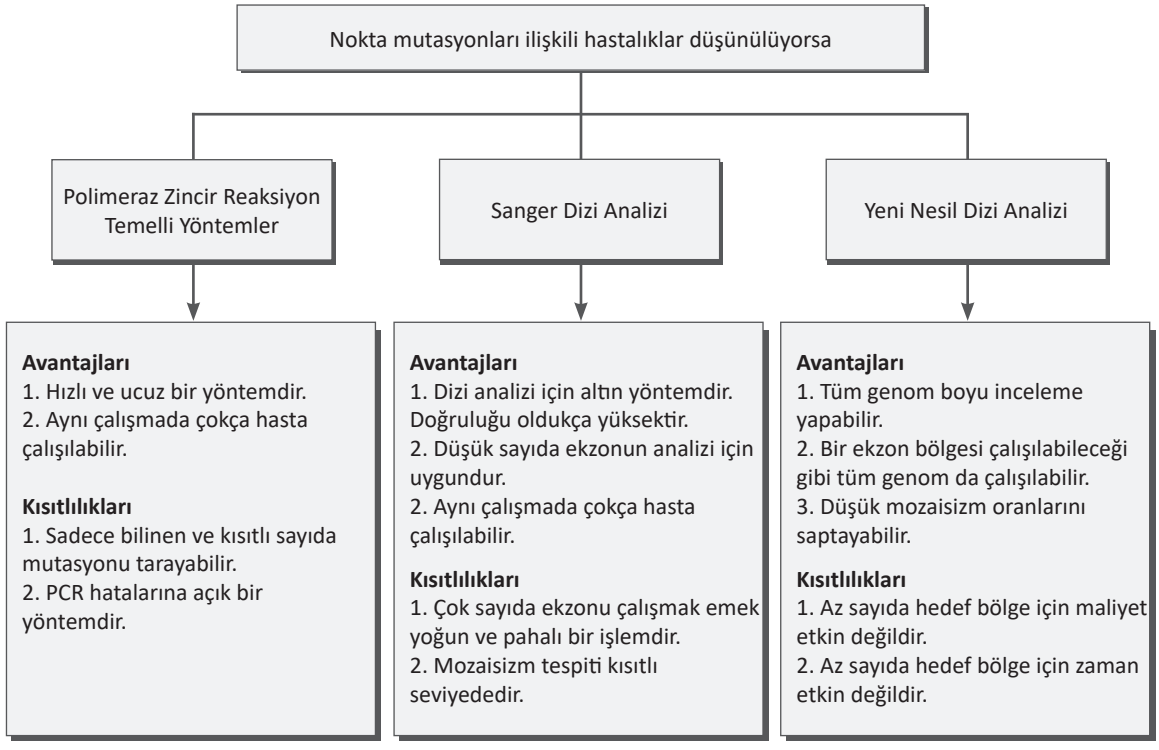
KÜÇÜK ÖLÇEKLİ MUTASYONLARIN TESPİTİNDE KULLANILABİLECEK YÖNTEMLERİN AVANTAJ VE KISITLILIKLARI[4,13]



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TAKİP ALGORİTMASI

- **Santral Sinir Sistemi:** Subependimal dev hücreli astrositoma (SEGA)'nın erken teşhisi için 25 yaşına kadar 1-3 yılda bir yapılmalı, bu yaşdan sonra riskin azalması nedeni ile sürveyans magnetik rezonans görüntülerine (MRG) ancak asemptomatik bir SEGA varsa ihtiyaç duyulur. Semptomatik bir SEGA veya artan ventriküler genişlemeye sahip bir SEGA kanıtı mevcutsa, daha sık nörolojik ve beyin cerrahisi izlemi gereklidir. Elektroensefalogram (EEG) gereksinim oldukça, tüberoz skleroz kompleksi ile ilişkili nöropsikiyatrik bozukluklar (TAND) ise yıllık değerlendirilmelidir. [6-8]
- **Dermatolojik-oftalmolojik-diş:** Yıllık dermatoloji ve göz değerlendirilmesi, iki yılda bir diş değerlendirmesi yapılmalıdır. [6-8]
- **Kardiyo vasküler sistem:** Kardiyak tarama ihtiyacı, intrakardiyak rabdomiyom varlığına bağlıdır. Prenatal veya infantil dönemde rabdomyom saptandı ise ekokardiyogram (EKO) her 1-3 yılda bir lezyon regrese olana kadar yapılmalıdır. Çoğu intrakardiyak rabdomyomun doğal seyri aylar veya yıllar boyunca spontan gerilemedir. 1-15 elektrokardiyogram (EKG) her 3-5 yılda bir değerlendirilmelidir.[6-8]
- **Renal sistem:** Abdominal MRG 1-3 yılda bir, anjiyomiyolipomun (AML) gözetimi ve tespiti için tercih edilen modalitedir, çünkü bunlar "yağdan fakir" olabilir ve renal ultrasonda gözden kaçabilir. Yıllık tansiyon ve glomerüler filtrasyon hızı (GFR) ölçümü yapılmalıdır. [6-8]
- **Solunum sistemi:** Solunum fonksiyon testi yıllık, 5-10 yılda bir yüksek çözünürlüklü bilgisayarlı tomografi (HRCT), lenfanjioleiomyomatozis (LAM) saptanırsa 2-3 yılda bir HRCT yapılmalıdır. [6-8]
- **Oftalmolojik:** Önceden tanımlanmış oftalmolojik lezyonları veya görme semptomları olan hastalarda yıllık oftalmolojik değerlendirme yapılmalıdır. Yeni görsel belirtiler gelişirse yeniden değerlendirme gereklidir. Vigabatrin ile tedavi edilenler de dahil olmak üzere daha sık değerlendirmenin faydası sınırlıdır. Epilepsi nedeni ile vigabatrin kullanımı dışında lezyonu bulunmayan olgularda yeni klinik endişeler ortaya çıkmadıkça rutin oftalmolojik kontrol önerilmemektedir.[6-8]
- **Dental:** Deforme edici diş veya semptomatik oral fibrom kemik çene lezyonları var ise tedavi edilmelidir. [6-8]

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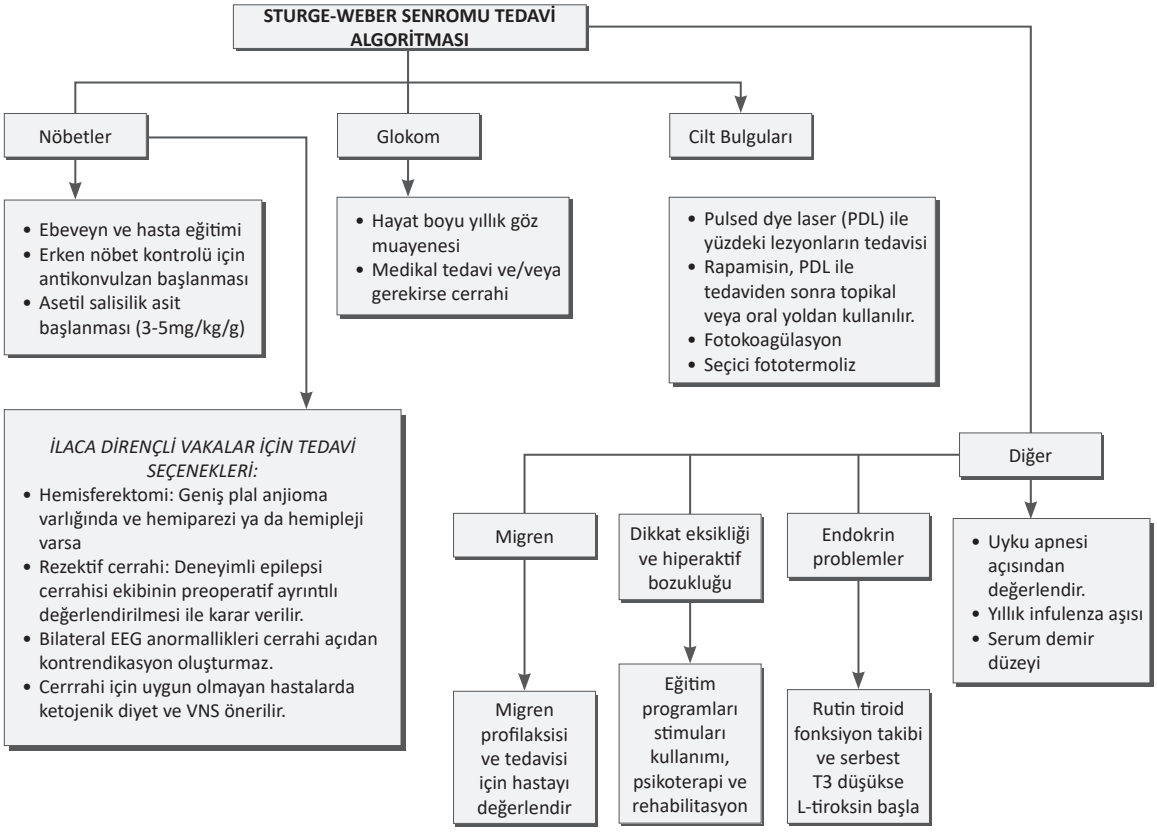

Şekil 2. Sturge-Weber Sendromu tedavi algoritması[1-5]

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Tablo 3: Von Hippel–Lindau (VHL)'de Nefrolojik Bulguların Yönetimi

1	Hemanjiyoblastomlu tüm hastalarda aile öyküsü sorgulanmalıdır.[17]
2	Hemanjiyoblastomlu tüm hastalarda Von Hippel – Lindau (VHL) için genetik test yapılmalıdır.[17]
3	0-15 yaş arası yıllık fizik muayene yapılmalıdır.[18]
4	Bir yaş ve üstü etkilenen bireylerde yıllık kan basıncı değerlendirmesi yapılmalıdır.[2]
5	Tip 2 VHL tanılı hastaların 2 yaşından itibaren abdominal ultrason görüntülemesi yanında kan basıncı, üriner katekolamin metabolitlerinin ölçümü yılda bir yapılmalıdır. Anormallik tespit edilirse abdominal MRI ve iyot-131 metaiodobenzil-guanidin (MIBG) taramaları yapılmalıdır.[19]
6	4-5 yaşından sonra, her yıl feokromositoma için laboratuar taraması yapılmalıdır.[2,17]
7	12 yaşından itibaren renal hücreli karsinom ve pankreas tümörlerini tarama amacı ile yılda bir kez batın manyetik rezonans görüntüleme (MRI) yapılmalıdır.[17].
8	16 yaşından itibaren, visseral lezyonları belirlemek için yılda bir kez abdominal ultrason yapılmalı ve her 2 yılda bir karın ve tüm nöral aksın manyetik rezonans görüntülemesi (MRI) yapılmalıdır.[2]
9	VHL hastalarındaki renal hücreli karsinom (RCC)'un optimal tedavisi, renal ven invazyonu ve uzak metastazlar meydana gelmeden önce cerrahidir. Metastatik lezyonlar varsa kemoterapi ve radyoterapiye zayıf yanıt verir.[15]
10	Çoklu tirozin kinaz inhibitörleri gibi pVHL-HIF-VEGF yolunu inhibe eden ilaçlar sunitinib, sorafenib, pazopanib ve monoklonal anti-VEGF antikoru bevacizumab'ın renal hücreli karsinom tedavisinde kanıtlanmış bir rolü mevcuttur.[15]

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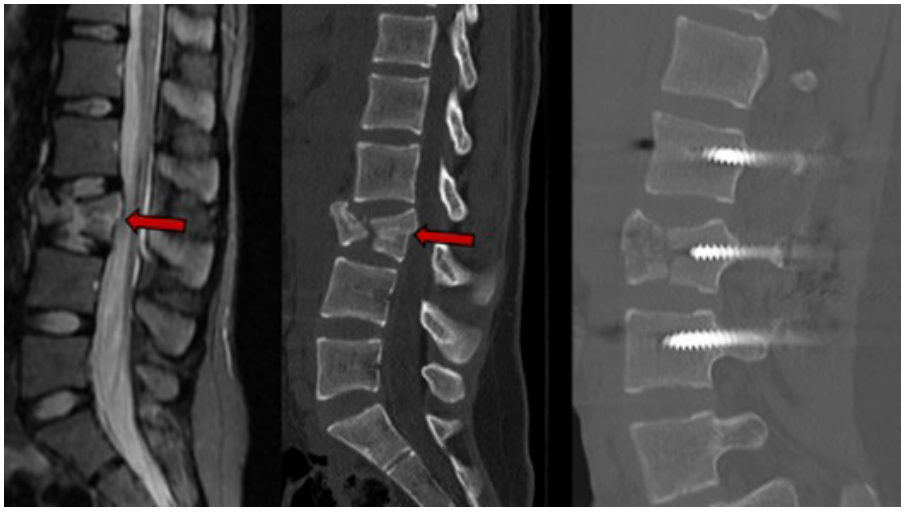
tajdır. SCIWORA tedavisinin ilk yaklaşım omurganın immobilizasyonunun sağlanmasıdır. Yüksek doz metilprednizolon tedavisinin erken dönem sekonder hasarı önlemede etkili olduğu; morbidite ve mortaliteyi azalttığı bildirilmektedir.[12] Dikkat edilmesi gereken diğer hususlar ise eşlik eden kranial travma ve spinal kord hasarında görülebilen nörojenik, hemorajik ve miks şok riskidir. Bu hastalarda vital bulguların stabilizasyonu sonrası yoğun bakım takibi önerilmektedir. [1]



Şekil 1. Adölesan yaş grubu cerrahi olarak tedavi edilen unstabil L3 patlama kırığı

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	Herniyasyon bulgusu yoksa sedasyona geç • Kısa etkili opiyatlar (propofol) bolus başlanıp infüzyona geçilebilir • Narkotik analjezik (fentanil) eklenebilir • Hastanın klinik durumu çok ciddiyse non depolarize ajanlar ile nöromuskuler blokaj (cisatracurium,rocuronium,vecuronium) düşünülmelidir.Bolus ile başlanıp infüzyona geçilir. 'Train of four periferal sinir stimulatörü" ile saat başı atım kontrolü yapılır, stabillendikten sonra 4 saatte bir takip edilebilir. Hedef uyarı ile 2 atım sağlanmasıdır. • 0-1 atım arası dozu %10 azalt • 3-4 atım arası dozu %10 arttır
	Hipotermi uygula: • İntravasküler veya yüzeyel soğutmayı hedef vücut ısısı 32-34°C olacak şekilde düzenle • Hedef IKB ulaşıldıktan sonra saatte 0.2°C yi geçmeyecek şekilde ısıtmaya başla • Vücut kor ısısını özefageal veya mesane probu ile takip et • Titremenin önüne geç Sürekli kardiyak monitorizasyon ve günlük EKG takibi yap Günlük Hemogram, PT/INR ve aPTT takibi yap 6 saatte bir plazma Mg ve P düzey takibi yap *immünsupresyon, enfeksiyon, trombositopeni, koagulopati, bradikardi, hipokalemi, hipomagnezemi,hipofosfatemi riski vardır
	• Pentobarbital infüzyonu başla. 5-20mg/kg 30-60 dk da yükleme sonrası 1-4mg/kg/st infüzyon • Sürekli kardiyak monitorizasyon, kan basıncı ve EEG takibi yapılmalıdır. *ciddi hipotansiyon, immunsupresyon/enfeksiyon riski mevcuttur.

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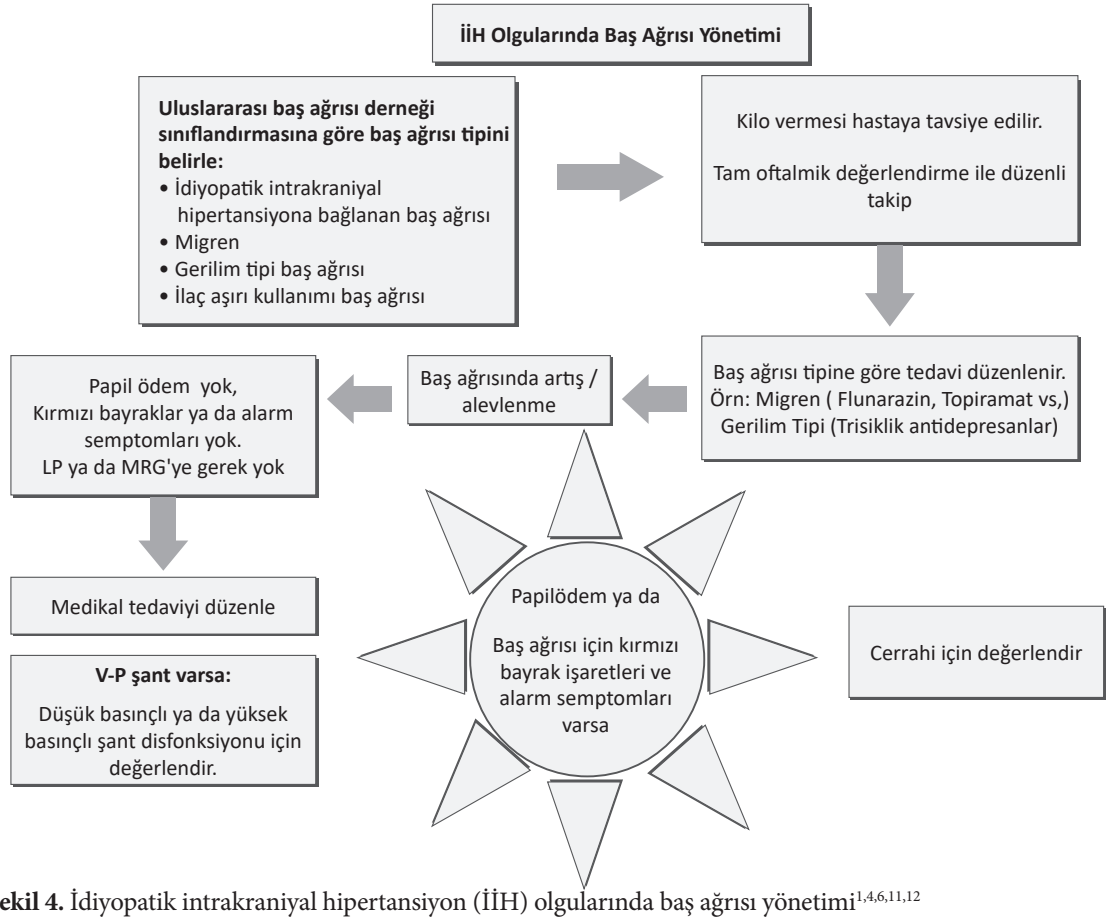

Şekil 4. İdiyopatik intrakraniyal hipertansiyon (İİH) olgularında baş ağrısı yönetimi[1,4,6,11,12]

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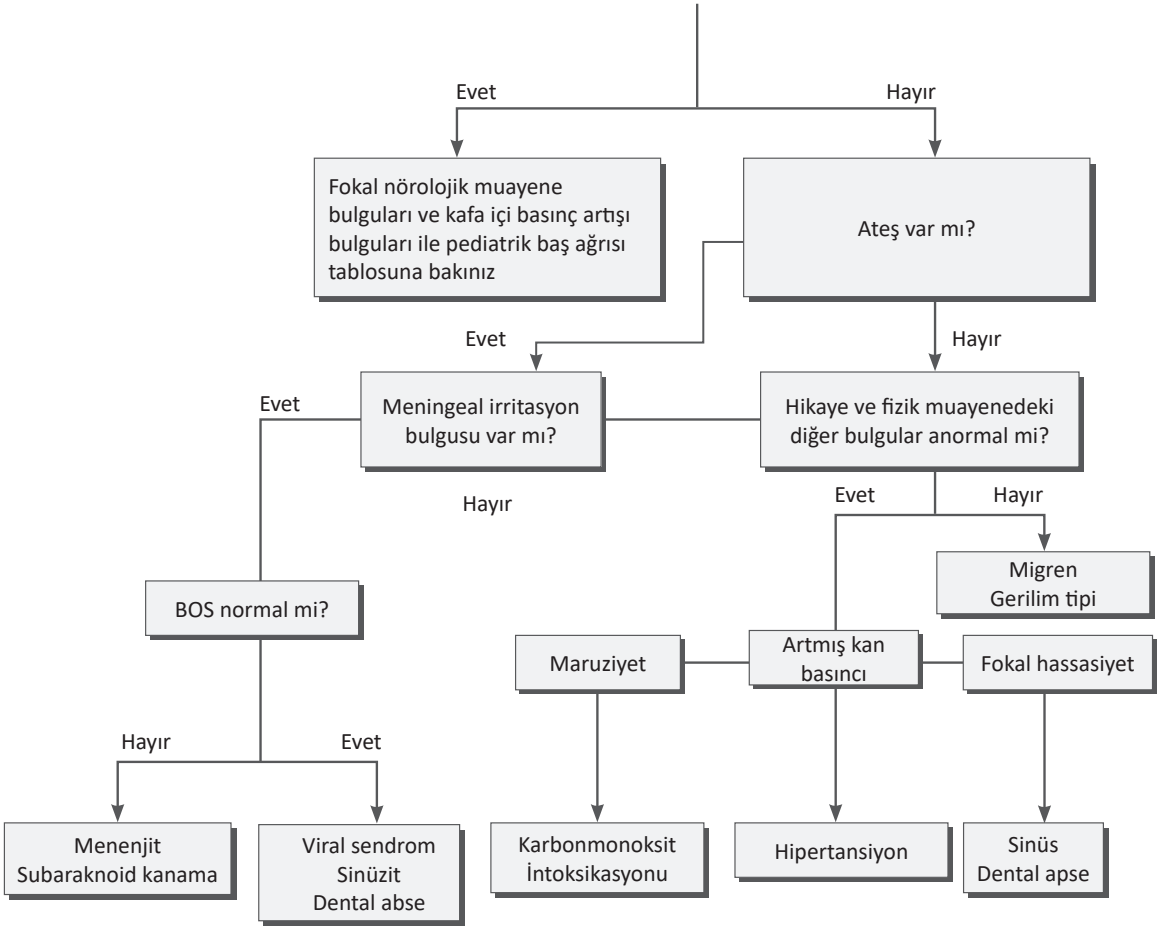

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Tablo 1: ICHD-3'e göre GTBA tanı kriterleri[2]

Epziodik ve kronik GTBA tanısı **için A ve B kriterlerine ilave olarak C-E kriterleri de sağlanmalı**dır.
Seyrek epizodik GTBA tanı kriterleri
A. Ayda bir günden seyrek (yılda 12 günden az) ortaya çıkan ve B-D kriterlerine uyan en az 10 atak
B. Otuz dakikadan 7 güne kadar süren baş ağrısı
Sık Epizodik GTBA tanı kriterleri
A. En az üç aydan beri, ayda bir gün veya daha sık, ancak 15 günden az ortaya çıkan (yılda 12 gün veya daha fazla ve yılda 180 günden az) ve B-D kriterlerini karşılayan en az 10 atak
B. Otuz dakikadan 7 güne kadar süren baş ağrısı
Kronik GTBA tanı kriterleri
A. En az üç aydan beri, ayda ortalama 15 gün veya daha sık ortaya çıkan (yılda 180 gün veya daha fazla) ve B-D kriterlerine uyan baş ağrısı
B. Baş ağrısının saatler sürmesi veya sürekli olması
C. Baş ağrısının aşağıdaki özelliklerden en az ikisine sahip olması:
1. Bilateral yerleşim
2. Sıkıştırıcı/basıcı (zonklayıcı olmayan nitelik)
3. Hafif veya orta şiddet
4. Yürüme ya da merdiven çıkma gibi günlük fiziksel aktivitelerle şiddetlenmemesi
D. Aşağıdakilerden her ikisinin bulunması:
1. Bulantı ya da kusmanın olmaması (iştahsızlık olabilir)
2. Fotofobi ya da fonofobiden sadece birinin olması
E. Baş ağrısının başka bir bozukluğa bağlanamaması
Olası GTBA tanı kriterleri
A. Seyrek epizodik, sık epizodik ve kronik gerilim tipi baş ağrısı A-D tanı kriterlerinin biri dışında tümüne uyan ataklar
B. Atakların aurasız migren tanı kriterlerine uymaması
C. Baş ağrısının başka bir bozukluğa bağlanamaması

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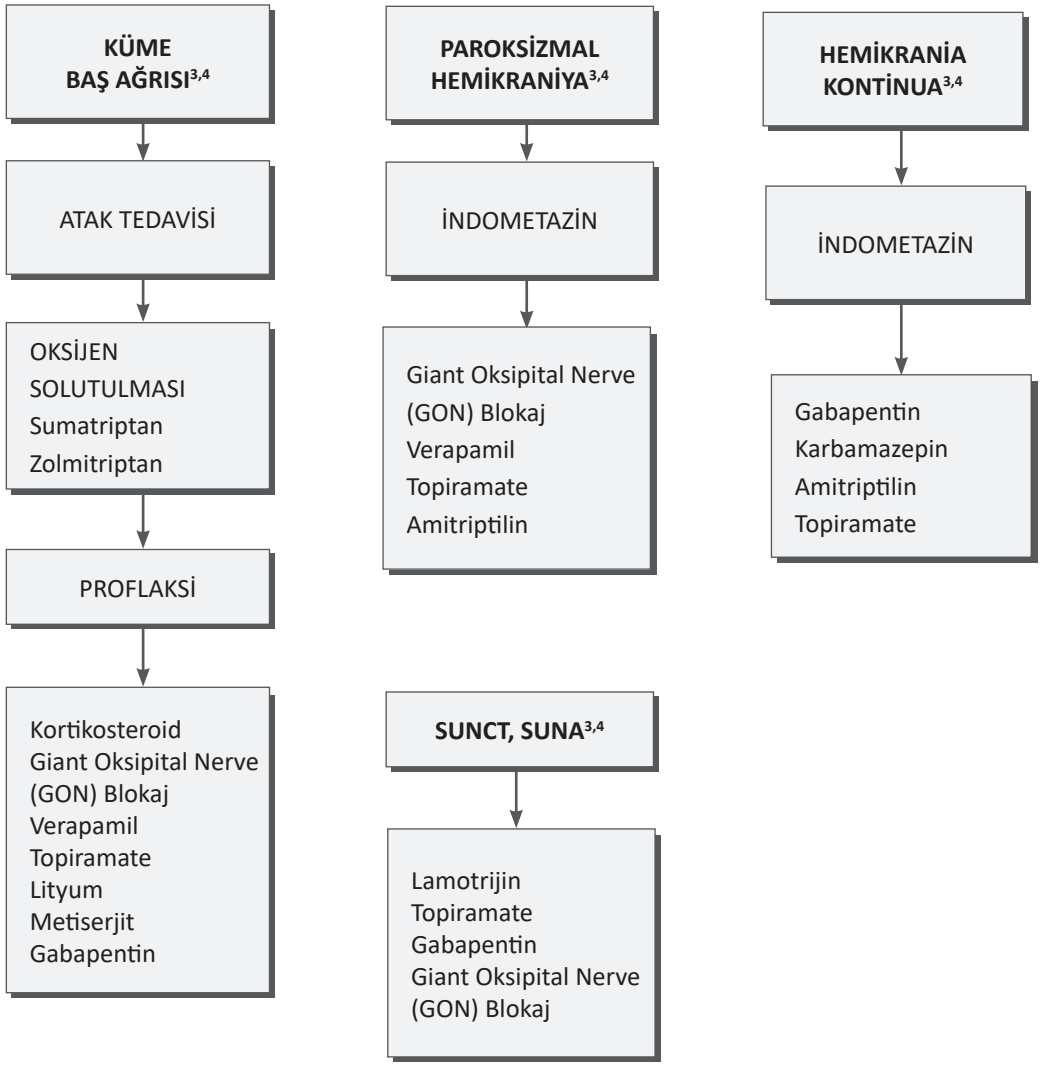

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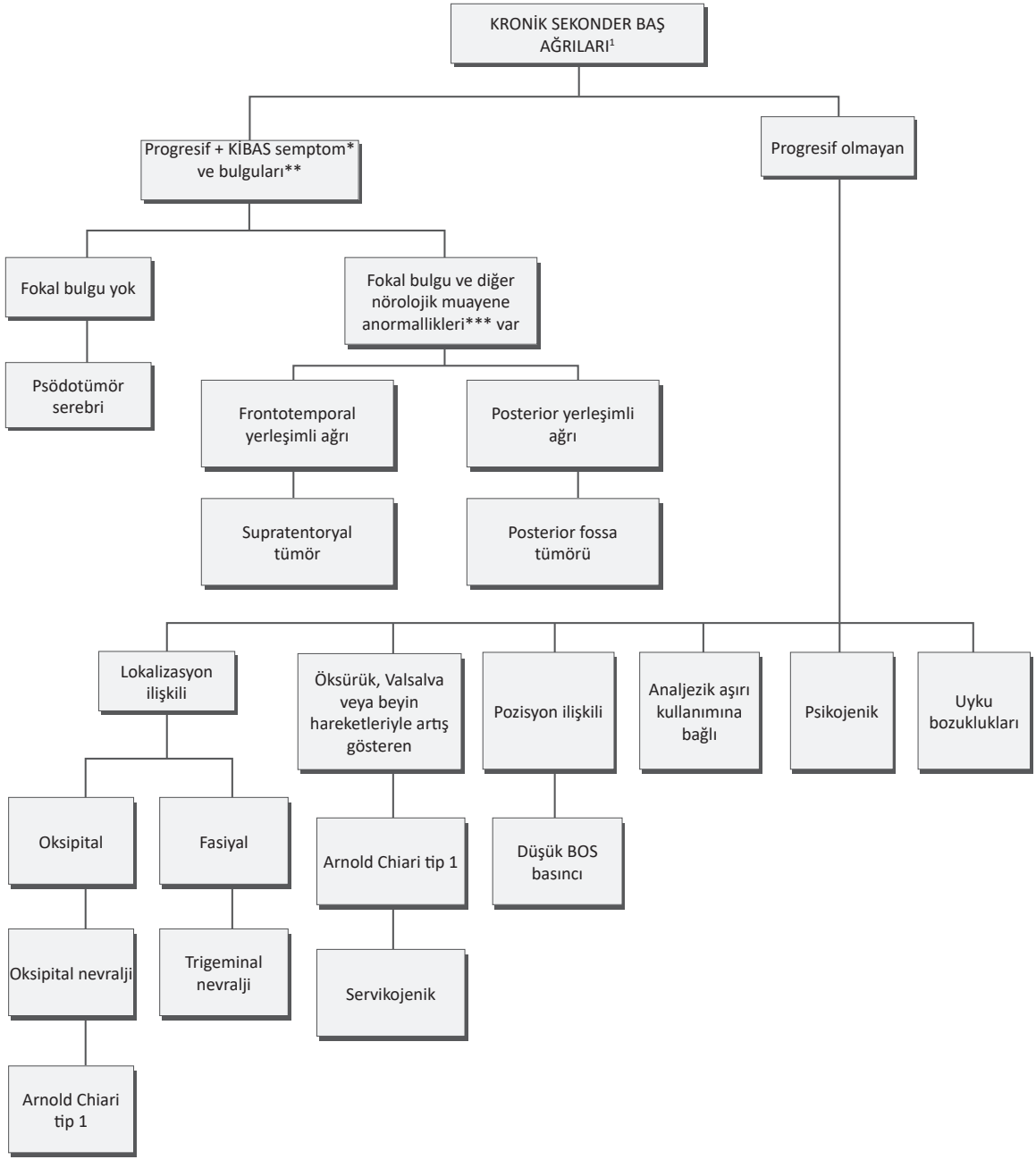

* KİBAS semptomları; bulantı, kusma, görsel semptomlar, gece ve sabaha karşı uykudan uyandıran baş ağrısı
** KİBAS bulguları; papil stazı, anormal göz hareketleri, görme alanı veya görme keskinliğinde değişiklikler, ense sertliği
*** Fokal bulgu ve diğer nörolojik muayene anormallikleri; fokal güçsüzlük, anormal göz hareketleri, anormal refleksler, kişilik değişikliği, ataksi.

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DİĞER	İLAÇLAR	NSAİİ (indometazin), steroid ve IVIG kullanım öyküsü			Tedavilerin modifiye edilmesi
	FIRSATÇI ENFEKSİYONLAR	Otoimmün hastalıkların tedavisinde verilen immünsüpresanlara sekonder bakteriyel, viral, fungal ya da paraziter enfeksiyonlar	Meningoensefalit ya da abselere bağlı kitle ve bası etkileri gözlenebilir	BOS kültürleri	Enfeksiyona spesifik antimikrobiyal tedaviler

MSS: Merkezi Sinir Sistemi, MR: Manyetik Rezonans, KB: Kan Basıncı, HT: Hipertansiyon, PAN: Poliarteritis Nodoza, ANCA: Antinötrofilik Sitoplazmik Antikor, IVIG: İntravenöz İmmünglobulin, mPAN: Mikroskobik Poliarteritis Nodoza, HLA: İnsan Lökosit Antijeni,
AFAS: Anti Fosfolipid Antikor Sendromu, ANA: Antinükleer Antikor, aPTT: Aktive Parsiyel Tromboplastin Zamanı, ACE: Anjiotensin Dönüştürücü Enzim,
JIA: Juvenil İdiopatik Artrit, RA: Romatoid Artrit, NSAİİ: Non-steroidal Antiinflamatuvar ilaç, IL: İnterlökin, MS: Multiple Skleroz,
PRES: Posterior Reversible Ensefalopati Sendromu, KİBAS: Kafa İçi Basınç Artış Sendromu, PLEX: Plazmaferez, BOS: Beyin Omurilik Sıvısı

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Nöbetle ilişkili baş ağrılarının migrenle ayırıcı tanı algotritması: Migren ve epilepsi, spesifik klinik özelliklere ve altta yatan patofizyolojik mekanizmalara sahip farklı nörolojik hastalıklardır. Ancak, tek bir hastada her iki durumun sıklıkla birlikte görülmesi ve olası klinik benzerlikler olması nedeniyle ortak ve ayırt edici özellikler vurgulanmıştır.

*Hemiplejik migrende de olabilir.

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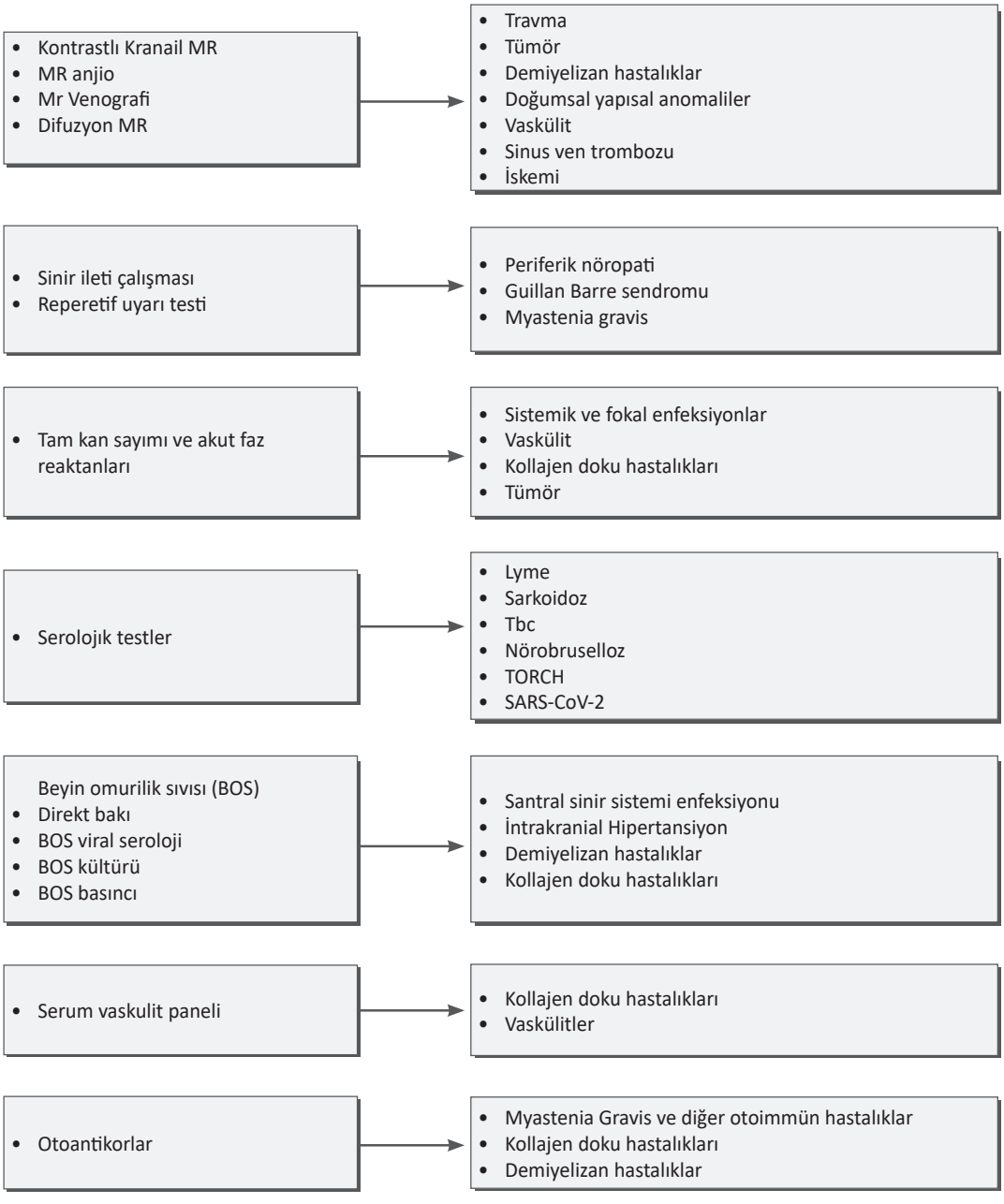
HASTANIN AYRINTILI ÖYKÜSÜNÜ AL



Kranial sinir hastalığı ile başvuran hastanın değerlendirilmesi [1,2,3]

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Pediatrik hareket bozuklukları anlayışımız monogenik nörogenetik bozuklukların keşfindeki ilerleme ve buna bağlı olarak yapılandırılmış tanı ve tedavi yaklaşımlarının geliştirilmesiyle uyarılarak hızla gelişmektedir. Anormal hareketlere neden olan nörometabolik bozukluklarda spesifik metabolitlerin araştırılması klinik tablonun yanı sıra tanı için önemli ipuçları verebilir. Öncelikli olarak tedavi edilebilir durumların erken teşhisi için mantıklı bir dizi araştırma izlenmelidir.

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TEDAVİ

Amonyak yüksekliğinin tedavisi acildir. Tedavide amonyak atılımı artırılmalı ve yeni amonyak oluşumu önlenmelidir.[1] Akut atak tedavisinde protein alımı kesilerek, damar yolundan yüksek dekstrozlu mayi verilmelidir. Kan şekerlerinin yüksek seyretmesi durumunda endojen protein katabolizması önlemek için mayinin dekstroz konsantrasyonunu azaltmak yerine aynı damar yolundan insülin infüzyonu başlanılmalıdır. Protein alımı 24-48 saatten daha uzun süreyle kesilmemelidir. Sodyum benzoat ve sodyum fenilasetat ile amonyak düzeyleri düşürülmelidir. L-arjinin kullanımı amonyak üretimini azaltmaktadır ancak arjinaz eksikliğinde kullanılmamaktadır. NAGS eksikliğinin tedavisinde karglumik asit verilmektedir. Sitrin eksikliğinin tedavisinde diğer üre döngüsü bozukluklarından farklı olarak yüksek protein ve düşük karbonhidrat içeren diyet uygulanmaktadır. Plazma amonyak düzeyinin 400 µmol/L'den yüksek olması durumunda veya tedaviye dirençli hiperamonyemi durumunda hemodiyaliz/hemodiyafiltrasyon yapılmalıdır.[2]

Üre döngüsü bozukluklarında ömür boyu protein kısıtlaması yapılmaktadır.[10] Akut atak gelişmesini önlemek için stres, enfeksiyon ve uzun süreli açlık gibi durumlardan kaçınılmalıdır. Büyüme geriliği, nöromotor gerilik, öğrenme güçlüğü, spastisite, konuşma geriliği, dikkat eksikliği/hiperaktivite bozukluğu, hepatomegali ve epilepsi gibi uzun dönemde görülebilen komplikasyonlar bakımından hastalar izlenmelidir.[11]

PROGNOZ

Hastalığın mortalite ve morbidite oranları yüksektir.[12] Yenidoğan başlangıçlı formları geç başlangıçlı formlarına göre daha ağır seyretmektedir. Hastalığın prognozu amonyak yüksekliğinin düzeyi ve amonyağa maruziyet süresi ile ilişkilidir. Sık atak geçirmek prognozu olumsuz etkilemektedir.

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Van der Knap da ----N-asetil aspartat / kreatinin oranı (NAA/cr) azalmıştır

Vanishing whıte matter da --- Beyaz cevherde tüm metabolitler genelde azalmıştır.

Glutarük asidürü tip 2 ---N asetil aspartat düzeyinde azalma , kolın ve inositolde artma

Metabolik taramalar : İdrar organik asit analizi; Canavanda------ N –asetil aspartik asitte artış

Glutarik asidüride----- glutarik asit ve 3OH glutarük asit artış,

Plazma aminoasit analizi ; Glutarük asidürü ---lizin artışı

BOS incelemesında¸ Glutarük asidürü tip 2 ---lizin ve pipekolat mıktarın artması

EEG bulgusu; Krabbe de epileptiform aktivite ve jeneralize yavaşlama ve zemın ritminde disorganizasyon gözlenmektedir.

Spesifik enzim çalışmaları ayırıcı tanıda yardımcı olur.

Aşagıda tabloda en sık görülen makrosefalinin eşlik ettiği nörometabolık hastalıkların ayırıcı özellıkleı ile belirtilmiştir

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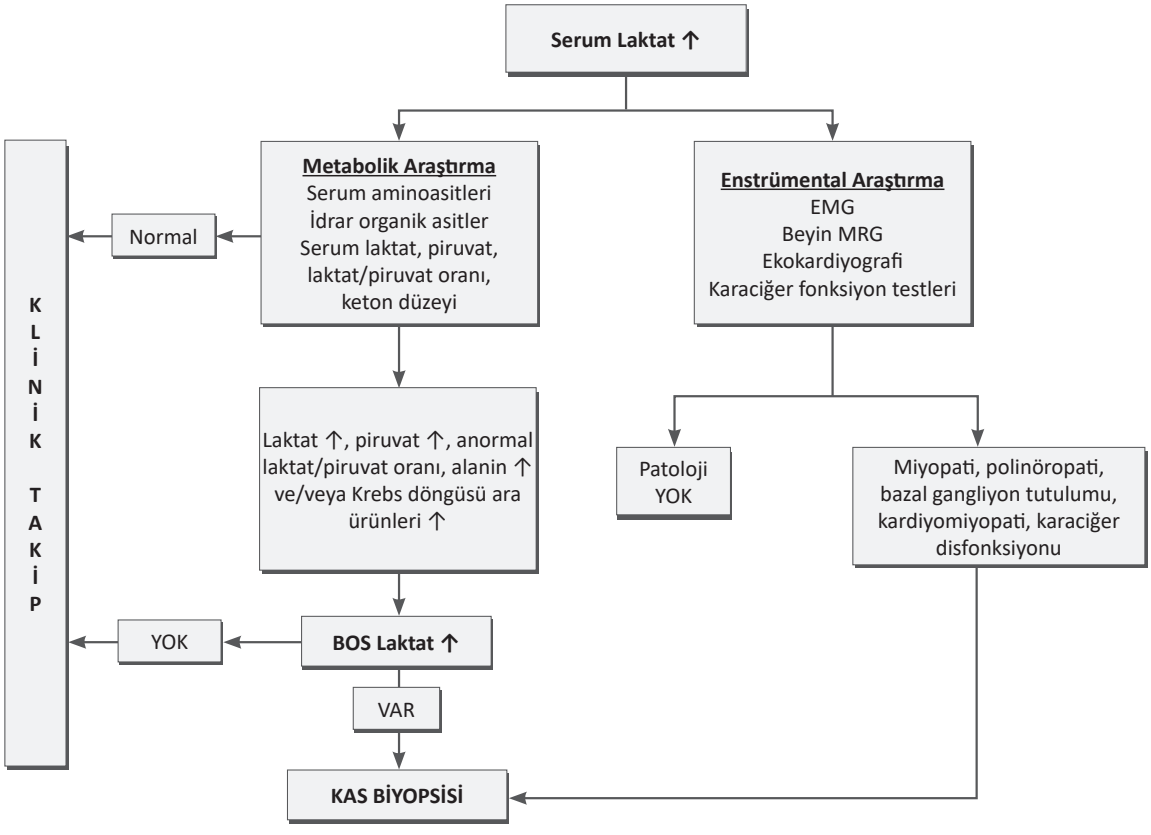
MİTOKONDRİYAL HASTALIK ŞÜPHESİNDE KAS BİYOPSİSİNE YÖNLENDİRME ALGORİTMASI



EMG; elektromiyografi, MRG; manyetik rezonans görüntüleme, BOS; beyin omurilik sıvısı. (4)

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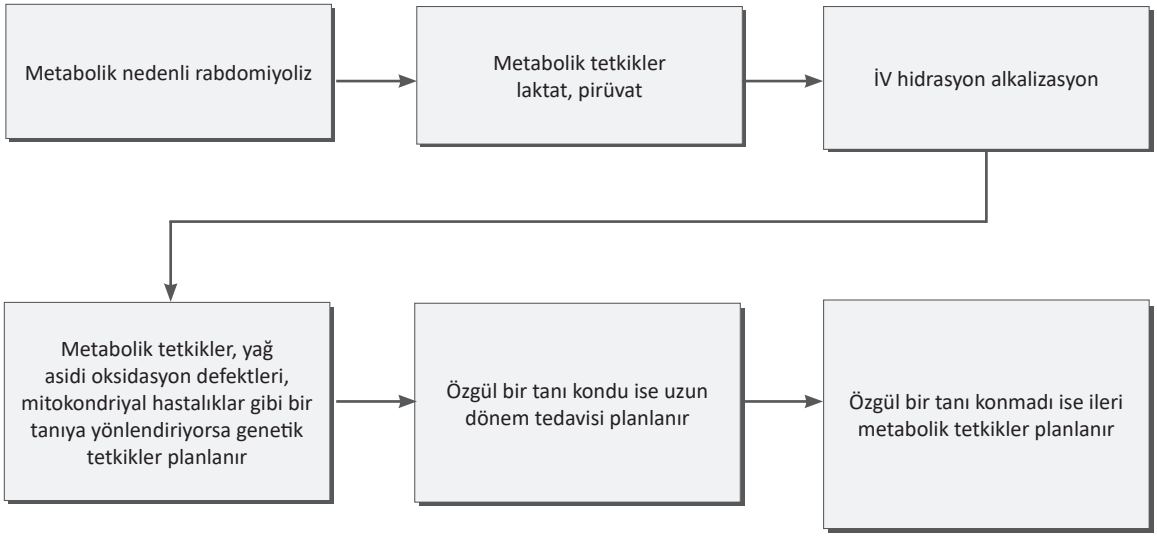
METABOLİK NEDENLİ RABDOMİYOLİZ [9,10]



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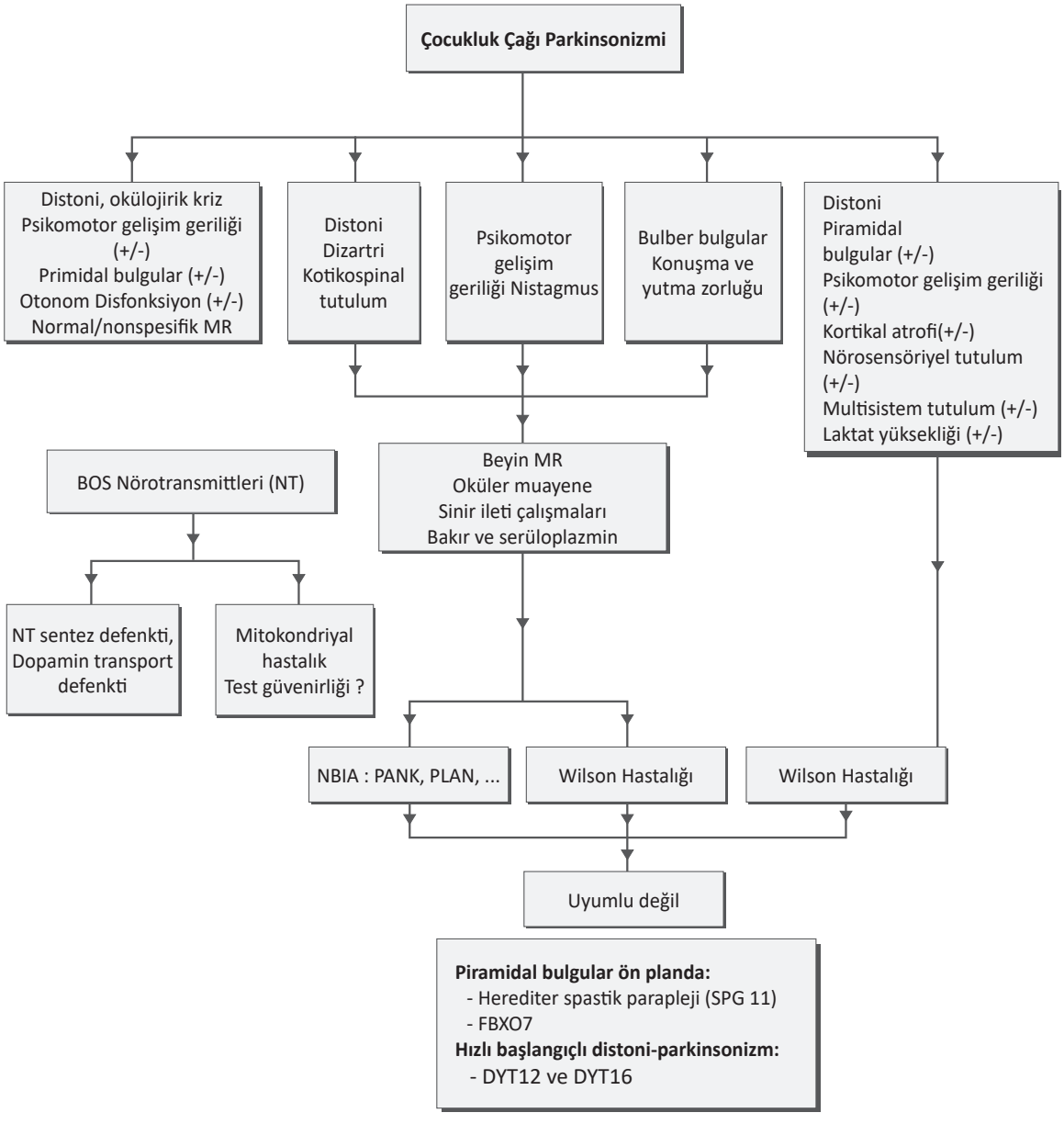
HİPOKİNETİK HAREKET BOZUKLUKLARINA TANISAL YAKLAŞIM[3]



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Tablo 10: Önceden Sağlıklı Çocukta Yaygın Görülebilen Akut Hareket Bozukluğu Sunumları

Hareket Bozukluğu	Etyolojiler	Açıklamalar
Kore	Poststreptokokal (Svdenham kore), diğer otoimmün ensefalit	Anti-N-mentil-D-aspartat (NMDA) reseptöründen ve diğer otoimmün ensefalitte sıklıkla karışık hareket bozukluğu (distoni, stereotipler). Tedavi: otoimmün ensefalit için immünomodülatör tedavi
	İlaca bağlı	Antikolinerjik, dopaminerjik ilaçlar (akut kore); dopamin reseptör blokerleri (tarif: genellikle akatizi ile mixt sendrom, distoni ile yoksunluk-ortaya çıkan diskinezi: ataksi ile hiperkinetik hareket bozukluğu)
Distoni	Akut distonik reaksiyon	Antipsikotikler ve antiemetikler (örneğin metoklopramid dahil olmak üzere dopamin reseptör, bloke edici ilaçların neden olduğu. Akut atak tedavisi: antikolinerjik ilaçlar
Miyoklonus	Opsoklonus Miyoklonus Ataksi Sendromu	Ataksi, uyku bozukluğu, sinirlilik eşlik edebilir. Genellikle nöroblastom ile ilişki. Tedavi: immünsupresyon semptomların remisyonunu indüklemek, uzun süreli motor ve zihinsel engelliliği sınırlamak amacıyla
Ataksi	Postinfeksiyöz Serebellar Ataksi	Bulasıcı hastalıklar veya aşıları takip edebilir. Başlangıç genellikle çok akuttur, %90'ı 2 ila 3 aylık bir süre içinde tamamen iyileşir.
	İlaca Alımı	Örneğin, antiepileptikler, benzodiapinler, antihistamimikler; tipik olarak zihinsel durum değişiklikleri eşlik eder.
Parkinsonizm	İlaca bağlı	Dopamin reseptör bloke edici ilaçların neden olduğu
	Otoimmün Ensefalit	Nadir; bazal gangliyonların inflamatuar lezyonlar ile ilişkili
Tremor, distoni yürüme bozukluğu	Psikolojenik, hareket bozukluğu	Klinik ipuçları: sabit duruşlu ani distoni başlangıcı, epizodik titreme. Tedavi, fizik tedavi ve komorbid duygudurum semptomlarının ilaç veya psikoterapi ile tedavisini içerebilir

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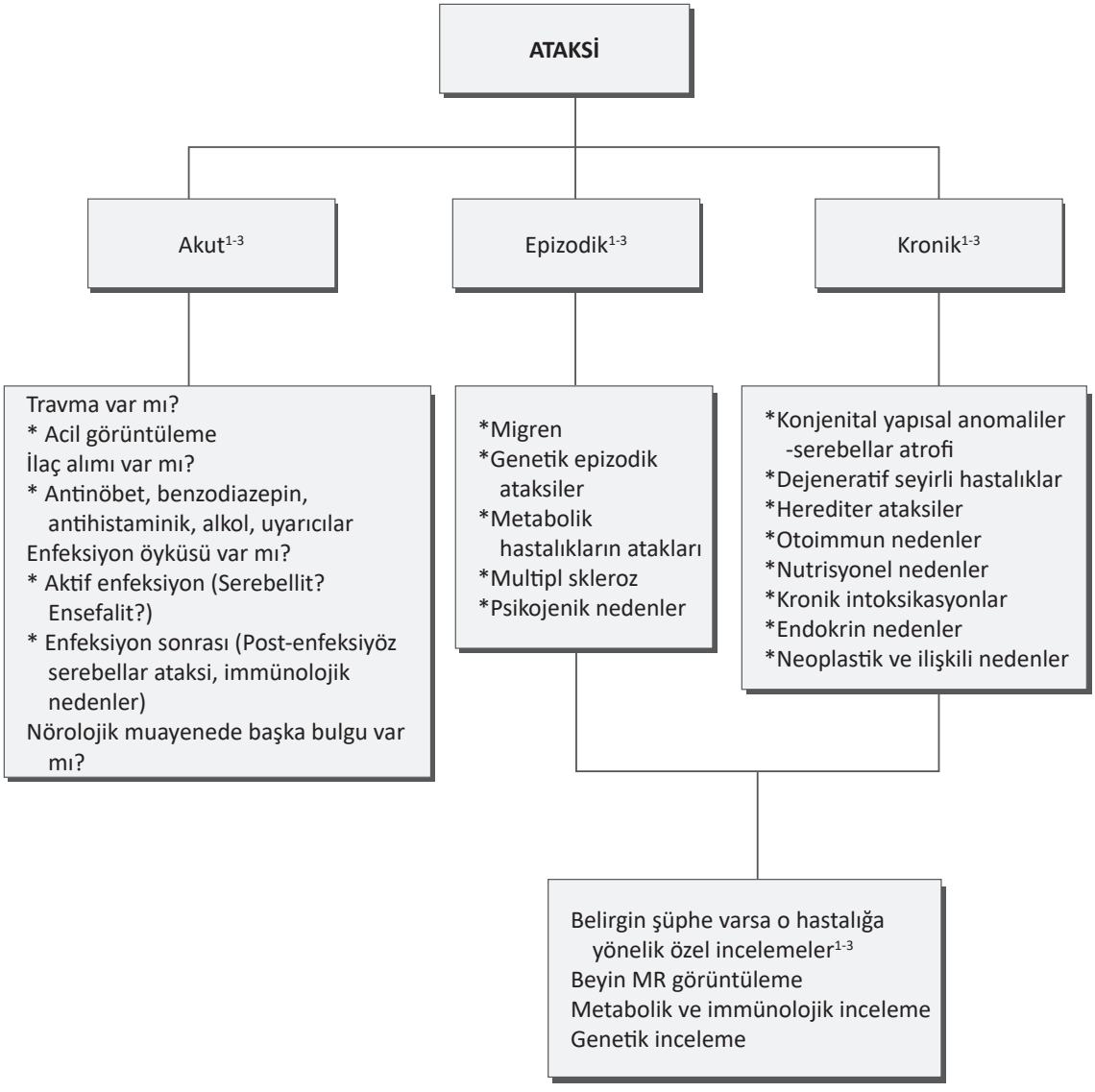
ATAKSİ TANI VE TEDAVİ ALGORİTMASI

BÖLÜM 69

İlknur CANKURT[1] Esra SERDAROĞLU [2]



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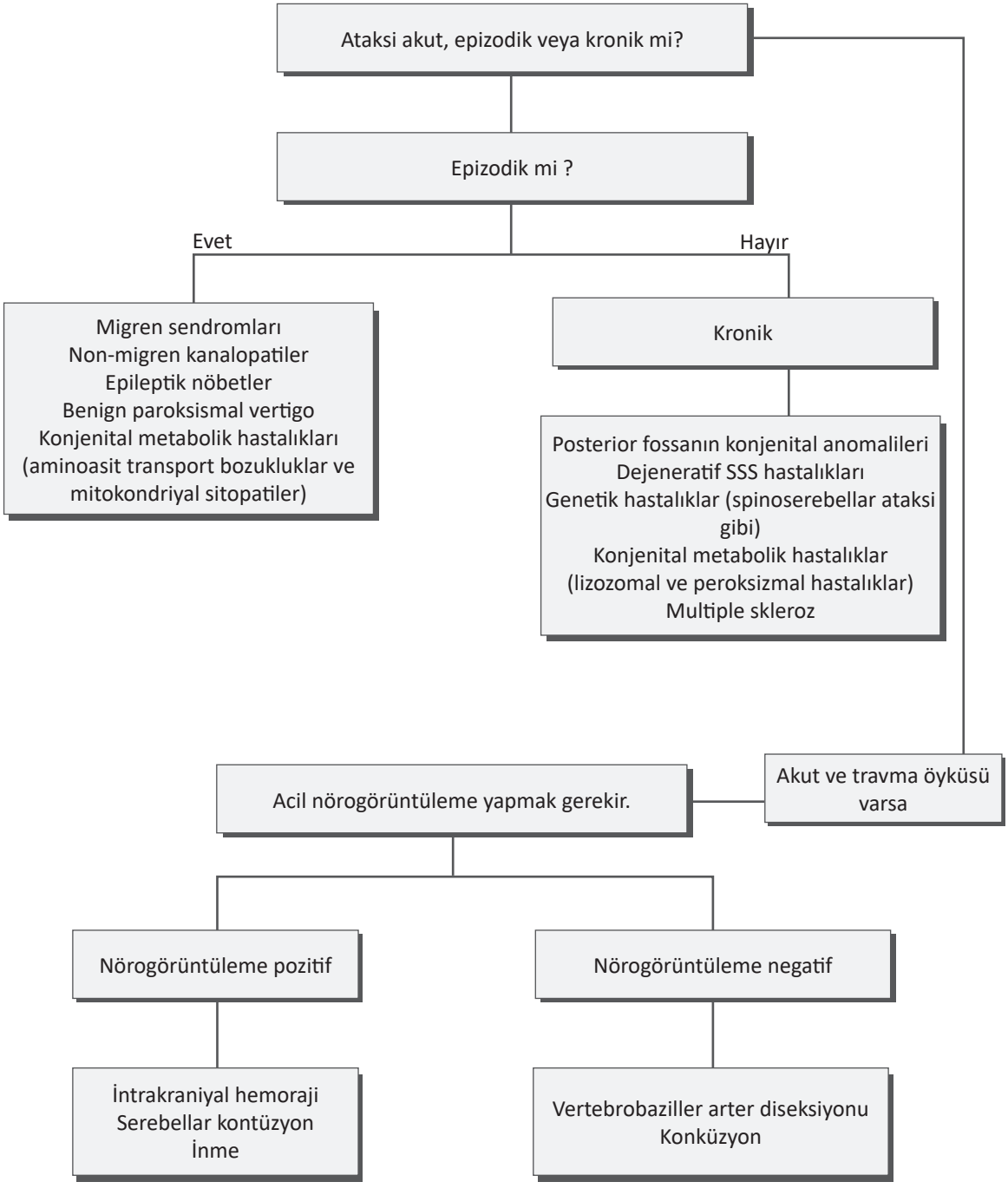
[2] Doç. Dr., Gazi Üniversitesi Tıp Fakültesi, Çocuk Nörolojisi Kliniği esra.serdaroglu@gmail.com



Şekil 2. Epizodik ve kronik ataksi[1,2]

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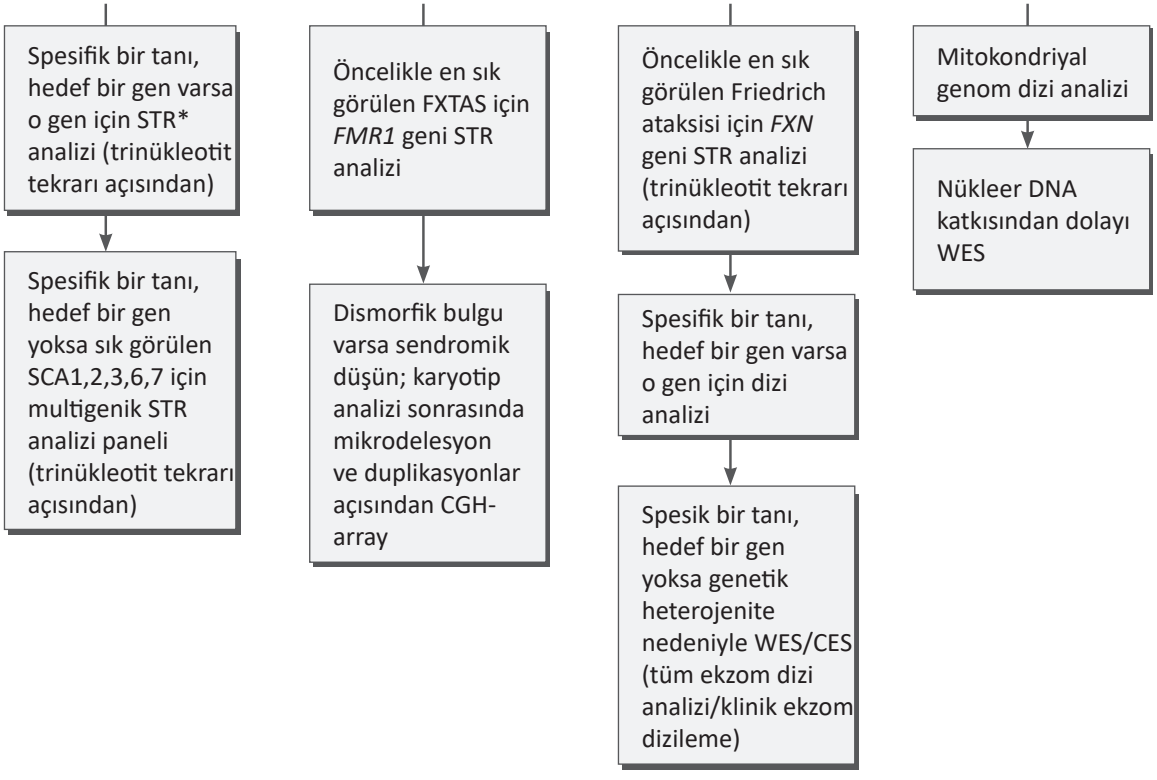

*: STR: tekrar dizisi analizi(short tandem repeat)
GAD: glutamik asid dekarboksilaz, FXTAS: Frajile X-tremor-ataksi sendromu

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NMOSD veya Devic hastalığı; spinal kordda ve/veya optik sinirde otoinflamatuar primer demiyelinizan durumla karakterize klinik bir spektrumdur. ON, TM ve area postrema bulguları (AP) gibi İKS'larda ve ADEM gibi ADS ile gelen hastalara AQP4-Ak pozitifliği veya negatifliğine göre, kriterlerin sağlanması halinde NMOSD tanısı konulabilmektedir.[8] MOG Ak pozitif olup NMOSD tanı kriterlerini sağlayan küçük bir hasta grubu ise MOG (+) NMOSD olarak isimlendirilebilmekte beraber bu grup MOGAD spektrumunun alt grubu olarak kabul edilmektedir.[9]

MS de SSS'nin diğer bir multifazik primer demiyelinizan hastalık olup ensefalopatinin olmadığı ilk ADS'de lezyonların zamanda ve mekanda dağılım kriterlerini sağlaması durumunda tanı konulabilir.[4] Prepubertal çocuklarda MS'in ilk klinik bulguları ADEM ile çok benzerlik gösterebilir. Lezyonların şekli ve yerleri ile birlikte oligoklonal (OLB), NMO-Ak, MOG-Ak seroloji sonuçlarına göre MS, diğer primer demiyelinizan durumlardan ayrılabilmektedir. Şekil 1'de SSS'nin primer demiyelinizan hastalıkların ayrıcı tanı algoritması verilmiştir.

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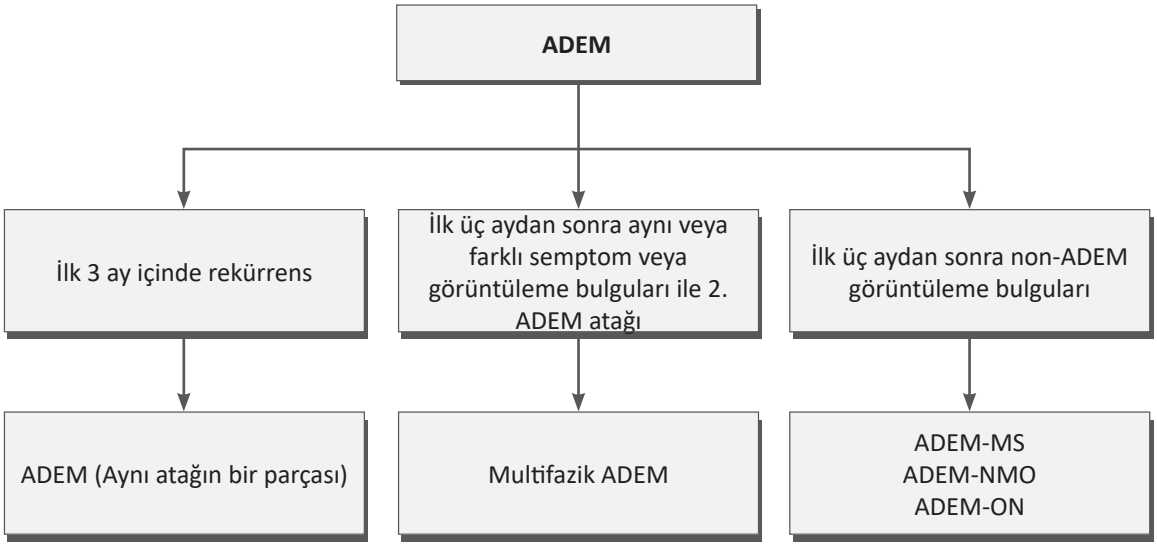
AKUT DİSSEMİNE ENSEFALOMİYELİT SONRASI TANISAL İZLEM



Şekil 4. Akut dissemine ensefalomiyelit sonrası tanısal izlem.[11,12]

ADEM: Akut Dissemine Ensefalomiyelit, ADEM-MS: ADEM-Multiple Skleroz, ADEM-NMOSD:ADEM-Nöromyelitis Optika, ADEM-ON: ADEM-Optik Nörit

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İzole ON olgularında lomber ponksiyon endikasyonu net belirtilmemiştir. Lomber ponksiyon ile SSS enfeksiyonu dışlanmalı ve inflamasyonu belirlemek için de IgG indeks ve oligoklonal bant çalışılmalıdır. Lomber ponksiyonda BOS basınç ölçümü de yapılmalıdır. ADEM, MS veya izole ON basınç artışına neden olabilmektedir. Bu nedenle BOS basınç artışının tespiti, psödotümör serebrideki papil ödem ile bilateral ON'e bağlı papil ödemin ayırıcı tanısında yeterli değildir.[2]

Bugüne kadar, pediatrik hastalarda prospektif kanıta dayalı bir ON tedavi çalışması yoktur. Bu nedenle yetişkin hastalardan elde edilen veriler kullanılmaktadır. Bu veriler ışığında 3-5 gün iv metilprednizolon (30mg/kg/g maksimum1g/gün) sonrasında da 1mg/kg/g dozunda oral steroid idamesi ve 1-1.5 aylık sürede kademeli kesilmesi çocukluk çağı ON'lerde genellikle tercih edilen tedavi seçeneğidir.[7] NMO çocuk hastalarda iv metilprednizolon 7 güne kadar da uzatılmaktadır.[8] Bununla birlikte uzun dönem oral steroid gerekliliği konusu tam bilinmemektedir. Pulse steroid tedavisinin prognoz üzerine belirgin etkisi gösterilmemiş olsa da iyileşme süresini kısaltıyor olması ve buna bağlı çocuk hastanın yaşam kalitesi üzerine olumlu etkisinin de olabildiği dikkate alınmalıdır. Dirençli olgularda iv kortikosteroid tekrar uygulaması önerilmektedir. İVİG de dirençli olgularda tedavi seçeneği iken birinci basamakta tercih edilmesi konusunda literatür desteği yoktur.[7] Özellikle izole ON ve çocukluk vakalarda çalışmalar yeterli olmamakla beraber steroid sonrası dirençli ve ağır görsel bozukluklarda plazmaferez düşünülmelidir (Tablo 2).

NMO için ise standart bir kılavuz olmamakla beraber kabul gören rejimde iv kortikosteroid, plazmaferez, İVİG ve immune baskılayıcı ajanlar (azatioprin ve mikofenolat vb) bulunmaktadır.[8] Özellikle NMO olgularında plazmaferez veya plazma değişiminin uygulanma zamanı klinik cevabı etkilemektedir. Tedavinin erken başlanması iyi prognoz olasılığını artırmaktadır. AQP4-IgG pozitif olan olgularda rekürrens ve uzun dönem takiplerde morbidite riski artmaktadır. Dolayısıyla ilk atak esnasında agresif tedavinin tercih edilerek rekürrens olasığının sınırlandırılması da iyi prognoz için önemlidir. AQP4-IgG negatif olgularda MOG antikoru bakılarak MOG-pozitif NMO veya seronegatif NMO-SH tanısının konulması prognoz üzerine etkili olan tedavinin belirlenmesinde yardımcı olacaktır.[5] MOG-IgG pozitif ilişkili hastalıklarda ilk basamak tedavi iv kortikosteroid, İVİG veya plazma değişimini içerir. NMO olgularında uzun dönem immünsupresyon ihtiyacı bulunmaktadır. Rituksimab, azatioprin ve mikofenolat mofetil immünsupresyonda ilk basamak ilaçlar olmakla beraber nüksler üzerine etkisi tam gösterilememiştir (Tablo 2).

KTİON diğer demiyelinizan nedenlerden kaynaklanan ON'lere benzer şekilde aktif fazda iv metilprednizolon ve ardından oral kortikosteroid azaltımı şeklinde tedavi edilir. Eğer bu tedavi etkisiz ise ek İVİG veya plazma değişimi düşünülebilir. ON atakları ve görme keskinliğindeki azalma nedeniyle KTİON'da mikofenolat mofetil, azatioprin, İVİG ve metotreksat gibi farklı immünosupresif ajanlar uzun süreli immünosupresyon için kullanılmaktadır (Tablo 2).[1]

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Alemtuzumabın pedMS'de kullanımına dair sonuçlanmış bir çalışma henüz yayınlanmamış olmasına rağmen birkaç gözlemsel çalışmada pedMS'de etkin olduğu ve hastalar tarafından iyi tolere edilebildiği gösterilmiştir.[18, 19]

Siklofosfamid ile ilgili olarak pedMS'de yapılmış olan çalışmada relaps oranında azalma ve MRG de lezyonlarının progresyonunda azalma gösterilmiş olup[20] yan etkilerinden dolayı pedMS'de son tedavi alternatiflerinden biri olarak değerlendirilmektedir.

Hastalara uygulanacak tedavi rejimleri genellikle klinisyenin tercihine ve uzman görüşüne dayanmaktadır. Birinci ve ikinci basamak tedavilerden yanıt alınamayan hastalarda infüzyon terapilerinin kullanılmasının hastalığın progresyonunu engellediği bilinmekte olup, infüzyon tedavilerine de yanıt alınamayan durumlarda hematopoetik kök hücre nakli önerilmektedir. Hastaların tedavisinde uygulanması önerilen algoritma Şekil 1'de verilmiştir.

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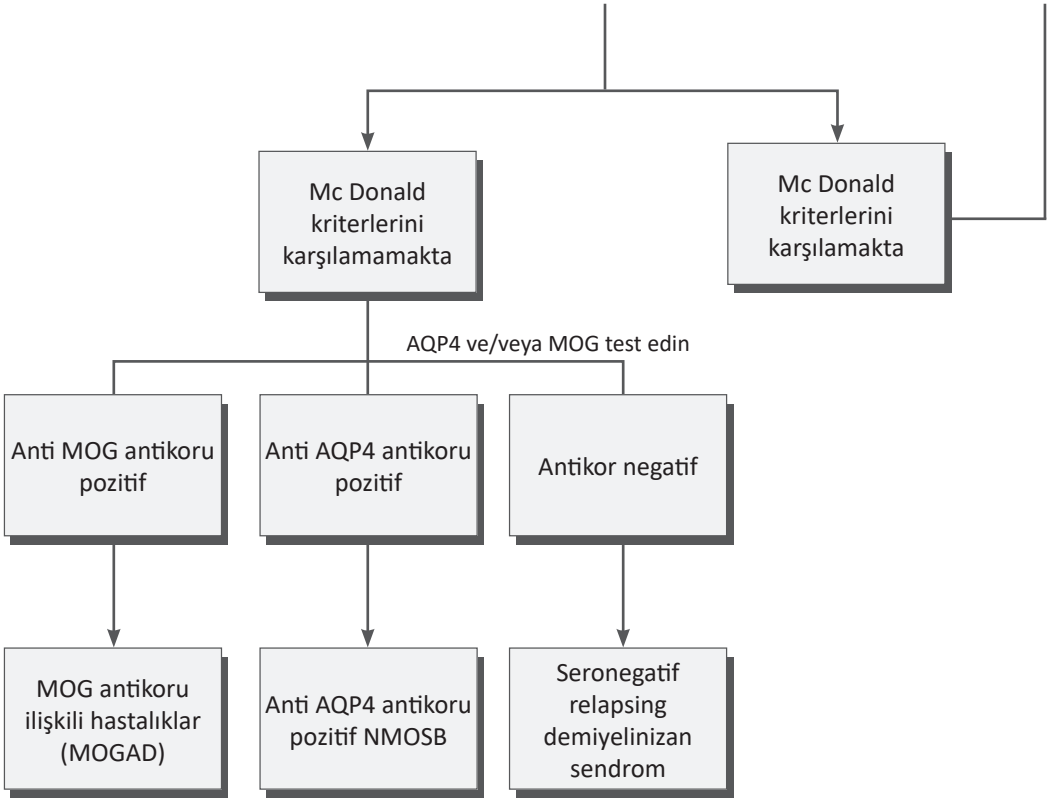

Ensefalopatisi olmayan bir çocukta edinilmiş demiyelinizan sendromun ilk sunumu için tanı algoritması[1]

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Tablo 1: NMOSB tanı kriterleri (2015)[2]

AQP4-IgG antikoru pozitif olan hastalarda NMOSB tanı kriterleri
1. ≥1 karakteristik klinik tutulum 2. Mümkün olan en iyi yöntemle AQP4-IgG antikorlarının pozitif saptanması (hücre temelli yöntemler önerilmektedir) 3. Kliniği açıklayabilecek alternatif tanıların dışlanması
AQP4-IgG antikoru negatif olan ya da bilinmeyen hastalarda NMOSB tanı kriterleri
1. Bir ya da birden fazla atağa bağlı gelişmiş ≥2 karakteristik klinik tutulum ve aşağıdaki gerekliliklerin tümünün karşılanması: a. En az bir karakteristik klinik tutulumun ON, LETM ya da area postrema sendromu olması b. En az iki ya da daha fazla karakteristik klinik tutulumun olması (mekanda yayılım) c. Destekleyici MRG kriterlerinin tamamını karşılanması 2. Mümkün olan en iyi yöntemle AQP4-IgG antikorlarının negatif saptanması ya da test sonucunun bilinmiyor olması 3. Kliniği açıklayabilecek alternatif tanıların dışlanması
Karakteristik klinik tutulumlar
1. Optik nörit 2. Akut miyelit 3. Area postrema sendromu: açıklanamayan aşırı hıçkırık, bulantı ya da kusma atakları 4. Akut beyin sapı sendromu 5. Semptomatik narkolepsi ya da NMOSB için tipik diensefalik MR lezyonlarıyla birlikte akut diensefalik sendrom 6. NMOSB için tipik beyin lezyonları ile birlikte semptomatik serebral sendrom
Destekleyici MRG bulguları
1. Akut optik nörit: a Beyin MRG'nin normal olması ya da nonspesifik beyaz cevher lezyonları içermesi ve b. Optik sinirin uzunluğunun >½'sinde ya da optik kiazmada T2 hiperintensitesi ya da T1 ağırlıklı incelemelerde kontrast tutulumu olması 2. Akut miyelit: Spinal MRG'de üç vertebra segmentini aşan lezyon (LETM) saptanması ya da akut miyelitle uyumlu öyküsü olan hastada 3 vertebra boyunu aşan spinal kord atrofisi izlenmesi 3. Area postrema sendromu: ilişkili dorsal medulla/area postrema lezyonlarının izlenmesi 4. Akut beyin sapı sendromu: ilişkili periependimal beyin sapı lezyonlarının izlenmesi

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- Akut tedavinin başlamasından sonraki bir ay içinde semptomların ilerlemesi veya aynı semptomların tekrarlaması "alevlenme" olarak değerlendirilir. Bu hastalarda yeni akut tedavi uygulamasının düşünülmesi gerekse de, idame tedavisine başlamak veya gerekmez.
- Başlangıç ADEM fenotipi olan hastalarda ise hastalığın ilk üç ayında dalgalı klinik semptomlar ve/veya dalgalı radyolojik anormallikler olabilir. Bu nedenle bu hasta grubunda "alevlenme" dönemi için üç ay dikkat edilmelidir.
- Hastalık relapsı için risk faktörleri: İleri yaş, ısrarlı MOGAD antikor poizitifliği, optik nöropati ile prezentasyon, iki relaps arası sürenin kısalığı.
- İdame tedavi ikinci ataktan sonra başlanır.
- İlk olaydan sonra yeterli iyileşme sağlanamayan hastalarda (esas olarak TM veya ON hastaları); yeni atak sonuçları açısından yıkıcı olabilir. Bu nedenle, ilk olayın akut tedavisinden (EDSS 3.0, modifiye rankin skalası 3) 1 ila 3 ay sonra yetersiz iyileşen hastalarda idame tedavisi hemen başlanabilir.
- MOG antikor pozitifliğinin devam ettiği hatta titresinde artış tespit edildiği durumlarda idame tedavi başlanması gerekmemektedir. Çalışmalarda monofazik olgularda bile 12 aydan fazla pozitif kalabileceği gösterilmiştir.
- İdame tedavisine rağmen relaps geçiren olgularda basamaklı tedavi (eskalasyon tedavisi) uygulanarak daha etkin ilaca geçilir.
- Yüksek hastalık yükü olan, nükseden hastalarda tedavi planında ek olarak MRG ve OCT kullanılabilir. Örneğin yeni MRG lezyonları varlığında veya OCT sonuçlarının kötüleştiği durumlarda idame immünoterapisinin artırılması düşünülebilir.
- İdame tedavisinin sonlandırılması için hastanın son 2 yıl süresinde atak geçirmemiş olması ve vizüel, motor, kognitif ve otonomik alanların stabil olması gerekmektedir.

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Notlar:

* GBS'da kranial MRG'de kranial sinirlerde kontrastlanma görülebilir.

**KBY bağlı nöropatide (üremik nöropati) başka bir bulgu olmadan sadece peroneal sinirde iletim hızında yavaşlama görülebilir. Semptomatik hastalarda alt extremitelerde duyusal semptomlar olabilir ve bunlar flask parapleji veya kuadriplejinin eşlik ettiği sensorimotor polinöropatiye dönüşebilir. Herhangi bir türde nöropati (tamamen aksonal, miks tip veya demiyelinizan) gözlenebilir.

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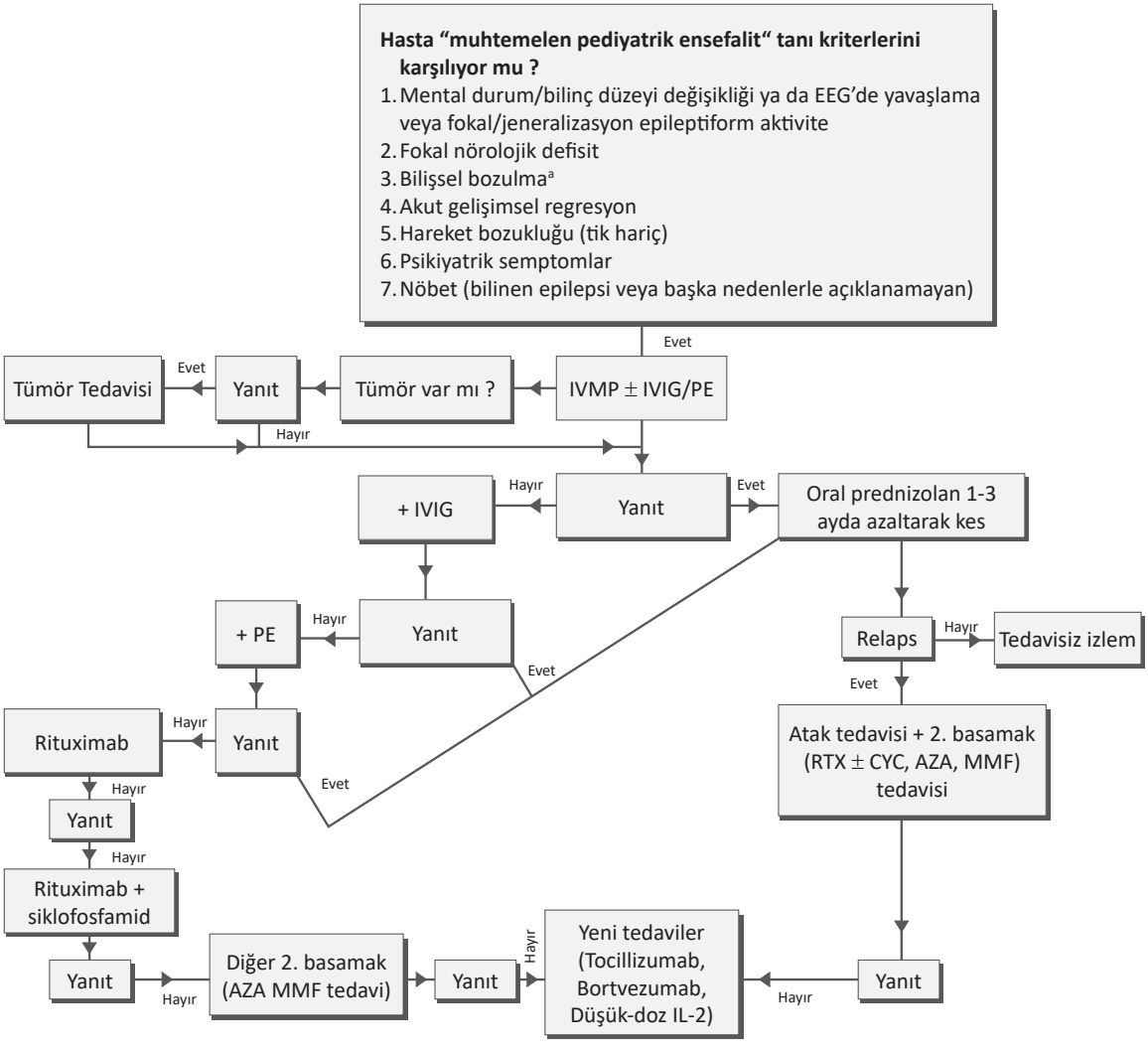

Algoritma 3. Pediyatrik otoimmün ensefalit hastalarında tedavi yaklaşımı

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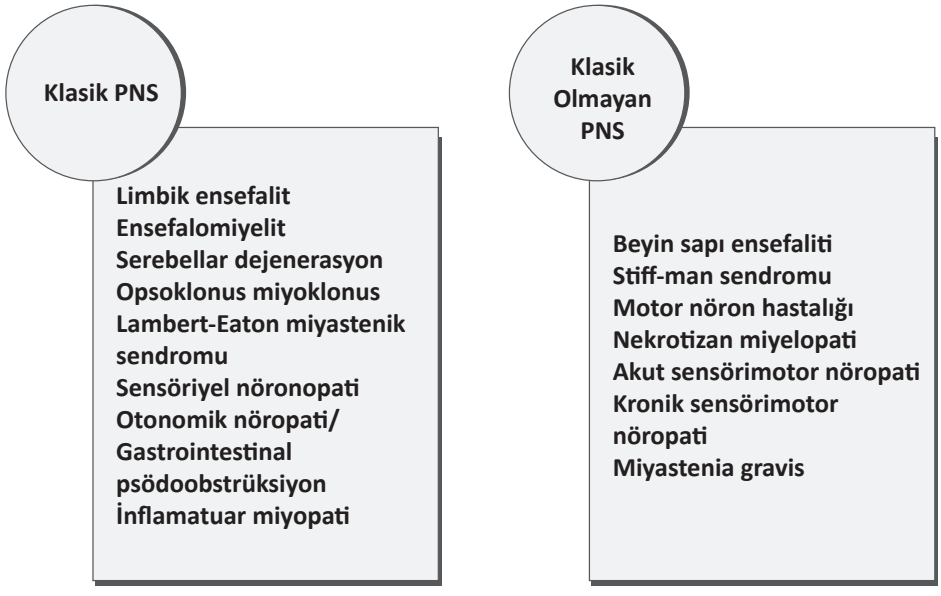

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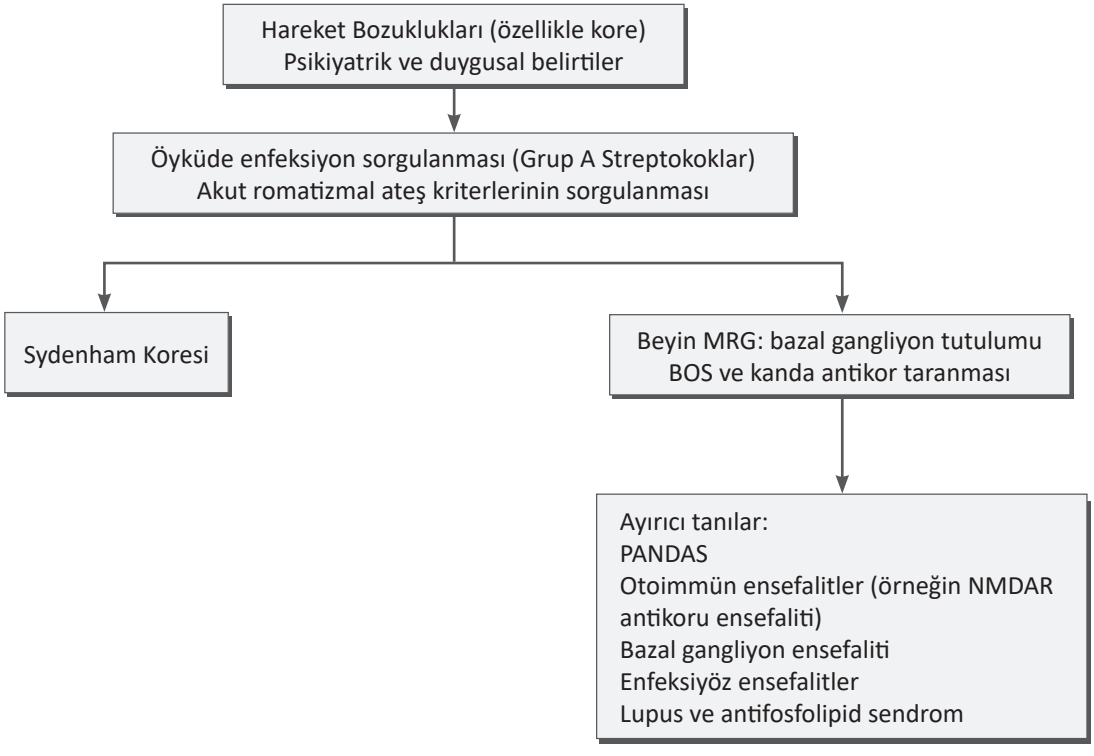
OTOİMMÜN BAZAL GANGLİA HASTALIKLARI TANI VE TEDAVİ ALGORİTMASI

BÖLÜM 91

Emine GÖÇER ÇELİK[1] Esra SERDAROĞLU [2]



PANDAS: Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streproccal infections

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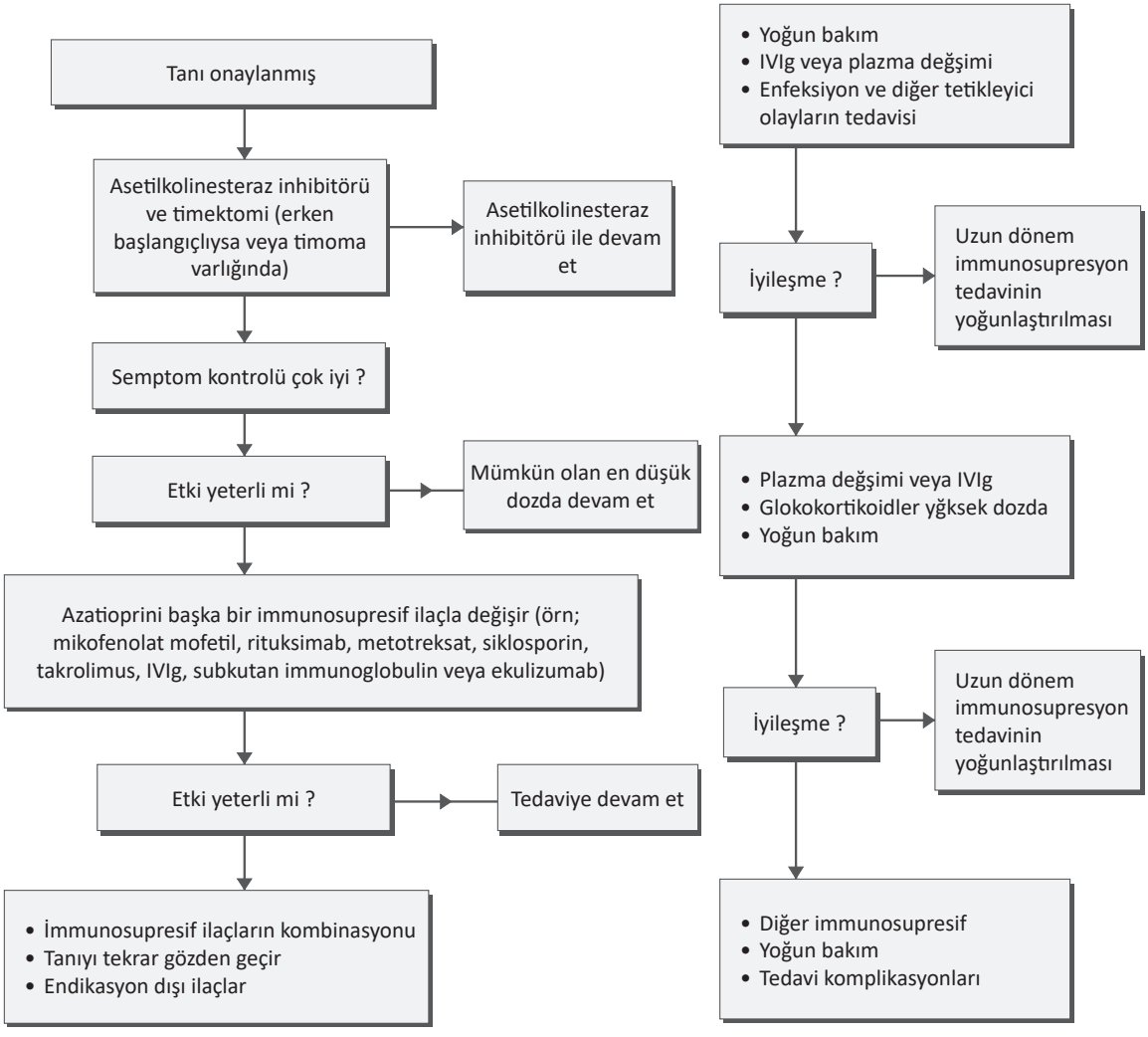

Kronik MG ve akut MG alevlenmeleri için tedavi algoritmaları. a: Kronik myastenia gravis (MG) tedavisi. b: MG'nin akut alevlenmelerinin tedavisi. Semptomatik ilaçlar, immünosupresif ilaçlar, timektomi ve destekleyici tedavinin bireyselleştirilmiş kombinasyonu, MG'li hastalarda iyi bir klinik sonuç hedeflenir. Prednizolon ve azatioprin kombinasyonu önerilir, ancak tek başına prednizolon veya mikofenolat mofetil ile kombine prednizolon alternatif birinci basamak immünosupresif tedavilerdir. IVIg, intravenöz immünoglobulin.[1]

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Fagosit sayısı ve işlevleri için

- Total Nötrofil sayımı (<1500/^{3} nötropeni, <500/mm^3 ağır nötropenidir.)
- NBT(Nitrobluetetrazolium testi), Kemotaksis, Oksidatif patlama (Dihidrorodamin testi)
- Adezyon Molekülleri (CD11, CD18) akım sitometrik yöntemle granülosit kapısında değerlendirilir.

Kompleman Sistemi; CH 50 Total hemolitik kompleman düzeyi ölçülerek değerlendirilir.

(Klasik ve Alternan yol tarama testidir. C1-C9 arası ayrı ayrı düzey bakılabilir. C5-C9 eksikliğinde tekrarlayan Neisseria infeksiyonları görülmektedir.) Ayrıca herediter anjioödem tanısında C1 inhibitör düzeyi ve fonksiyonunun belirlenmesi için tetkik yapılabilir.

Tanıda rutin laboratuvar incelemeleri ve klinik bulgular birlikte değerlendirilerek muhtemel immün yetmezlik kesin tanısı için daha ileri moleküler ve genetik incelemeler yapılabilir.

Hepatosplenomegali, granulomatoz lezyon, otoimmünite bulguları veya ürtikerin eşlik etmediği anjionörotik ödem gibi enfeksiyon dışı nedenlerle hekime getirilen çocuklarda da ayırıcı tanıda immün yetersizlik akılda tutulmalıdır. Primer immün yetersizliklerin erken tanısı morbidite ve mortaliteyi azaltmakta, komplikasyonların gelişimini önlemekte ve sağ kalım süresini uzatmaktadır[3].

Erciyes Üniversitesi'nde yapılan bir çalışmada 2000 bebeğin kordon kanından tam kan sayımı bakılarak lenfopeni olup olmadığı araştırıldı. Lenfosit sayısı <3000/mm$^{3'}$ ün altında olan 42 bebekten, bir ay sonra tekrar tam kan sayımı yapıldı. Sonuçta; 2 bebekte AKİY saptanarak, hematopoetik kök hücre nakli yapıldı ve şimdi hayattalar. Ülkemizde, yenidoğan topuk kanından TREC ile AKİY taraması yapılamadığı için hastanede doğan her bebeğin kordon kanından tam kan sayımı bakılması erken tanı açısından fayda sağlayabilir[4].

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Tedavi Yaklaşımı

1. Korunma (primer ve sekonder önleyici yaklaşımlar)
2. Varsa etyolojik nedene yönelik spesifik tedaviler
3. Destek tedaviler
   a. Fizik tedavi
   b. Çocuk psikiyatri (DEHB, Anksiyete bozukluğu, uyku problemleri, özel eğitime yönlenidirme)
   c. Çocuk psikoloğu
   d. Sosyal hizmet uzmanı
   e. Konuşma Terapisi
   f. Özel eğitim
   g. Bakımevi
   h. Beslenme ve reflü yönünden destek tedaviler(pediatrik gastroenteroloji ve çocuk cerrahisi, PEG, gastrostomi tüpü gereksinimi için)
   i. Ortopedik ve cerrahi müdahaleler
   j. Sosyal destek grupları

Özetle, gelişimsel gecikmeleri olan çocuklar birinci basamak ortamlarında rutin bakım sırasında tanımlanması erken tanı ve tedavi şansı verir. Ebeveynlerden ve/veya öğretmenlerden bilgi geldiğinde, birinci basamak sağlık hizmeti sağlayıcısı bunu ciddiye almalı ve gelişimsel gecikmeyi ve olası nedenlerini dikkatle değerlendirmelidir. Gereken ileri basamak sağlık kuruluşuna yönlendirmeli, mimkün olan en erken zamanda etyolojiye yönelik ve/veya destek tedaviye başlanmalıdır.

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<table>
<tr><td colspan="2">Zihinsel Yetersizliğin Etiyolojisine Yönelik Araştırmaların Yapılması ve Eşlik Edebilecek Sorunların Belirlenmesi</td></tr>
<tr><td>• Nörolojik Değerlendirme
• Genetik Değerlendirme
• İşitme Değerlendirmesi
• Görme Değerlendirmesi
• Endokrinolojik Değerlendirme
• Kardiyolojik Değerlendirme</td><td>Değerlendirmeye İlişkin Tetkiklerin Gerçekleştirilmesi;
Örneğin;
EEG, Görüntüleme Yöntemleri, Kan Tetkikleri, Genetik Tetkikler ya da Şüphelenilen Tıbbi Soruna Yönelik Tetkikler</td></tr>
<tr><td colspan="2">(Şüphelenilen etiyolojik soruna ya da eşlik eden tıbbi soruna göre bu değerlendirmeler ve tetkikler genişletilebilir)</td></tr>
<tr><td colspan="2">Tedavi</td></tr>
<tr><td colspan="2">Eğitim ve destek uygulamaları konusunda ailenin bilgilendirilmesi ve yönlendirilmesi
Etiyolojik nedene ya da eşlik eden sorunlara yönelik tedavinin düzenlenmesi
Davranışsal ya da ruhsal soruna yönelik tedavinin düzenlenmesi</td></tr>
<tr><td colspan="2">Takip</td></tr>
<tr><td colspan="2">Çocuk psikiyatri takibi (Sıklığı klinik duruma göre belirlenir)
Ek tıbbi duruma göre ilgili uzmanlık alanınca takip (Ör; Çocuk nöroloji, çocuk endokrinoloji)</td></tr>
</table>

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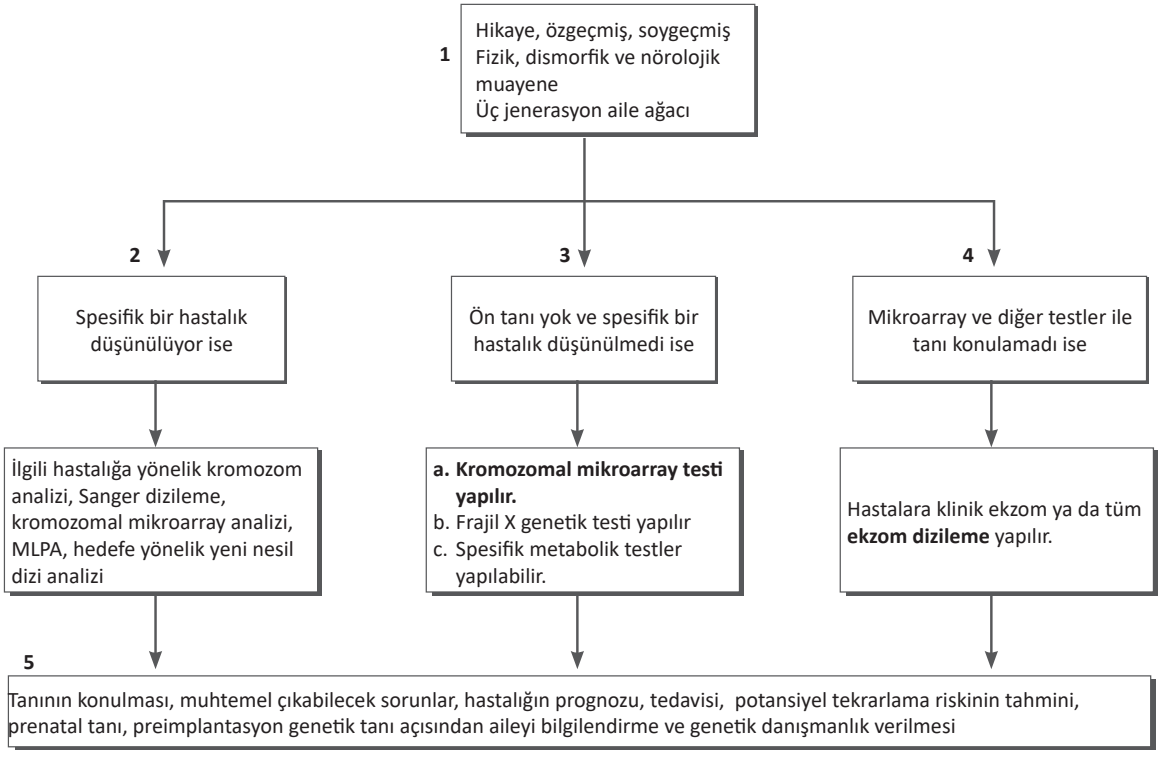

Şekil 1. Entelektüel yetersizliğe genetik yaklaşım algoritması

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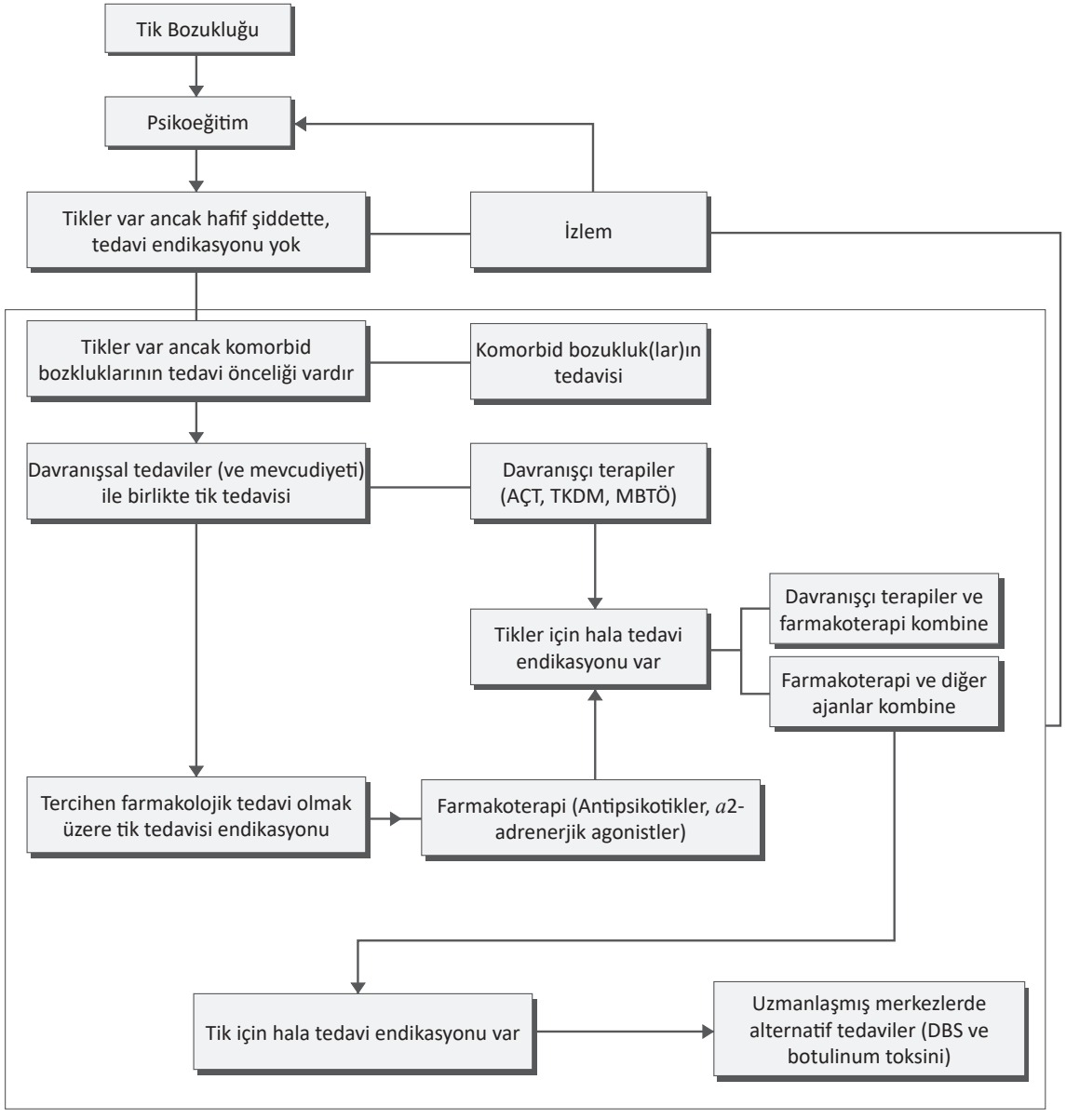

Şekil 2. Tik bozuklukları tedavi algoritması [2,3] Semptomlar rahatsız edici değilse ve/veya işlev bozukluğuna neden olmuyorsa destekleyici tedavi (örneğin psikoeğitim) önerilir. Semptomlar rahatsız edici ise farmakolojik veya farmakolojik olmayan müdahaleler yapılmalıdır. Ancak komorbid durumlar mevcutsa ve tik bozukluğundan daha fazla işlev bozukluğuna neden oluyorsa tedavi önceliği komorbid bozukluklar olmalıdır. Tedavi başarılı olduğunda, izlem esastır. Düz oklar, bir sonraki değerlendirme veya tedavi seviyesini gösterir; kesikli oklar, iki tedavi arasındaki seçenekler olduğunu gösterir. AÇT, alışkanlığı tersine çevirme tedavisi; DBS, derin beyin stimülasyonu; MBTÖ, maruz bırakma ve tepki önleme; TKDM: tikler için kapsamlı davranışsal müdahale.

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1. İlaç tedavileri
   - Stimülanlar ilaçlar
   - Non-Stimülan ilaçlar
2. Aile temelli müdahaleler
3. Okul temelli müdahaleler (davranış yönetimi, akademik müdahale vb.)
4. Sosyal çevre ve akran ilişkilerine dair müdahaleler (sosyal beceri eğitimleri vb.)
5. Çok yönlü tedavi yaklaşımları[1-5]

En sık kullanılan ilaçlar Amerikan Gıda ve İlaç Dairesi (FDA) tarafından 6 yaş ve üzeri için kullanım onayı olan Metilfenidat ve Atomoksetin ve 3 yaş ve üzeri için Dekstroamfetamin'dir.[3-4]

- Tedavi kılavuzları okul öncesi tanı alan DEHB'de ilk seçenek tedavi basamağı olarak psikosoyal yaklaşımları önermektedir.
- Özellikle grup temelli ebeveyn programlarını önerilmekte, davranışçı yaklaşımlardan fayda görmeyen ve belirti şiddeti fazla olan çocuklarda metilfenidat tedavisi başlanabileceğini bildirmektedir.
- Psikofarmakolojik tedavi endikasyonu olduğunda da psikososyal müdahalelerle birlikte kullanılması gerektiği vurgulanmaktadır.[1-5]

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Tablo 2: Deliryum Riski Yüksek Hastalarda Fizyolojik, Farmakolojik, Çevresel Tedbirler

Fizyolojik	Farmakolojik	Çevresel
Vücut fonksiyonunu normalleştir Beslenme ve sıvı alımını destekle Düzenli bağırsak ve mesane eliminasyonunu teşvik et İnvaziv cihazların ve kateterlerin kullanımını en aza indir **Gece/Gündüz Döngüsü Düzenle** Gün boyunca düzenli programlar oluştur Geceleri sağlıklı uyku düzenleri oluştur Gündüz iyi aydınlatma ve geceleri düşük aydınlatma sağla Kesintileri en aza indirmek için bakım zamanını düzenle **Hareketliliği ve aktiviteyi teşvik et**	Hasta ajiteyse antipsikotik eklemeyi düşün BZ, opiat ve antikolinerjik kullanımını en aza indir **Sedasyonu tekrar değerlendir** **Ağrıyı tekrar değerlendir** **Polifamasiyi en aza indir** **Yoksunluğu tekrar değerlendir ve tedavi et** **Uyku için melatonin ekle** **Sedasyon için Deksmedetomidin ekle (BZ ve/veya opiat yerine**)	**Ürkütücü ve şaşırtıcı durumlardan kaçın** **Fiziksel kısıtlamadan kaçın veya azalt** **Hasta güvenliğini sağla ve personel görevlendir** **Oryantasyonu optimize et** Hastaya ismiyle hitap et Sakin ve net konuş Optimal duyusal işleyişi sağla Güven ver ve sık sık yeniden yönlendir Bakım üyelerini tanıt Bakım faaliyetlerini basitçe açıkla Sürekli bir bakım ekibi sağla **Fiziksel ortam yönetimi** Gürültüyü ve dağınıklığı en aza indir Hasta ve aile üyelerine eğitim ver ve süreçte görevlendir Tanıdık aile ortamı yarat Görünür bir saat ve takvim göster **Hasta ve aile üyelerine eğitim ver ve süreçte görevlendir** Aile için özel roller belirle Yazılı bilgilendirici materyaller paylaş

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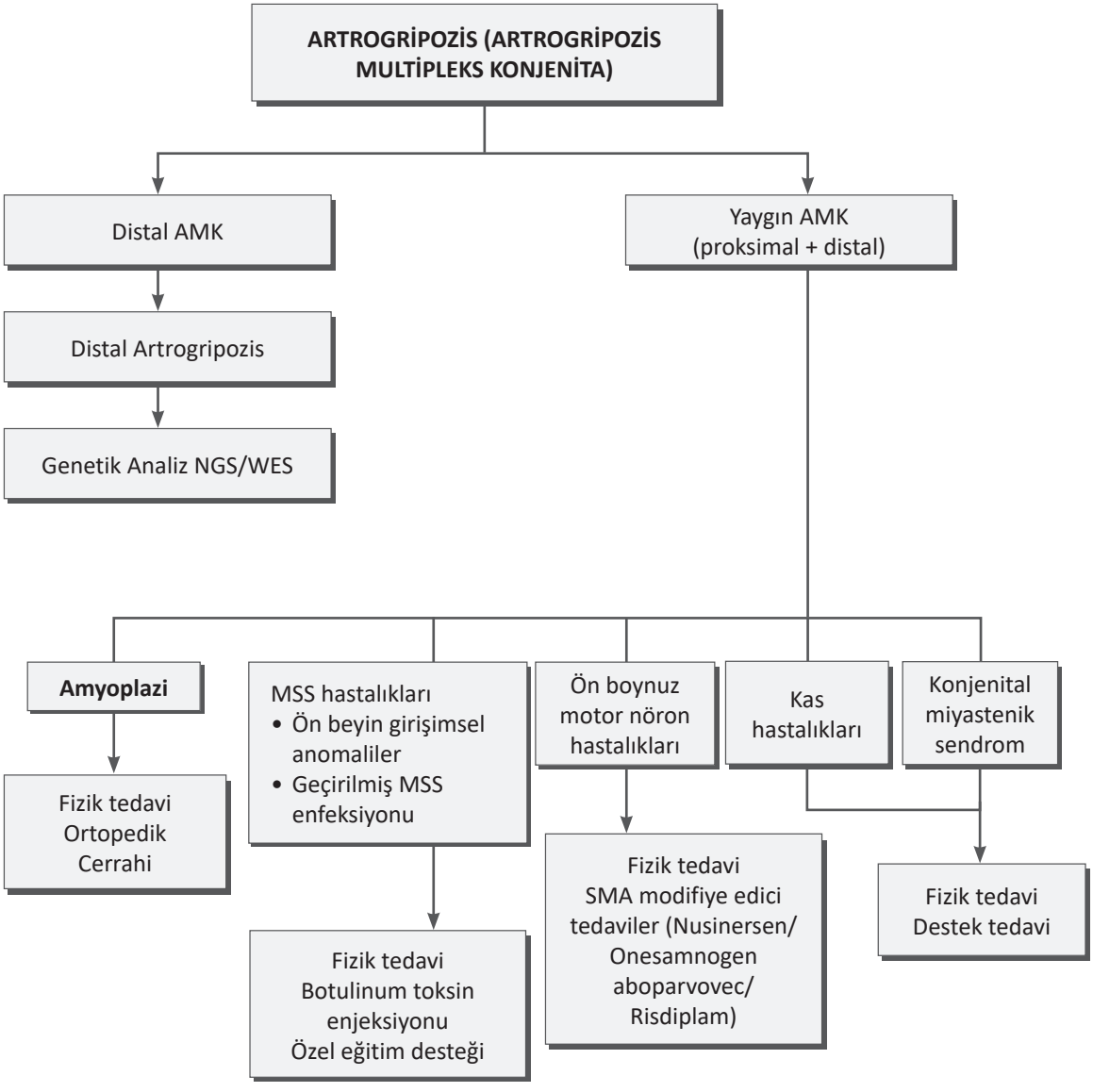

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SMA, TEDAVİ ALGORİTMASI 2

PULMONER TAKİP
- Tanı anında Çocuk Göğüs Hastalıklarınca kontrol ve takip (kan gazı, ön-yan akciğer grafileri)
- İnvaziv olmayan solunum destekleri (BİPAP)-trakeostomi ihtiyaçlarının değerlendirilmesi
- Oksimetre ile takip
- Lüzum halinde polisomnografi
- Evde manuel aspiratör

KAS İSKELET SİSTEMİ VE ORTOPEDİK TAKİP
- Kontraktür oluşumunu önleme
- Ayakta durma sehpası/ortez
- Skolyoz/Kalça çıkığını önleme ve tedavi etme
- Fraktürleri önleme ve tedavi etme (bifosfonatları düşün)
- D vitamini ve yeterli kalsiyum desteği
- Yıllık kemik mineral dansite ölçümü

GASTROİNTESTİNAL SİSTEM VE BESLENME TAKİBİ
- Vücut ağırlığı ve büyüme takibi (3-6 ayda bir)
- Gastroözofageal Reflü, kabızlık, gecikmiş gastrik boşalma için tedavi
- Floroskopik yutma çalışması
- Aspirasyonun önlenmesi için kıvam artırıcılar
- Gastrostomi ihtiyacının değerlendirilmesi

REHABİLİTASYON
- Kontraktür önleyici rehabilitasyon: Düzenli esneme ve germe egzersizleri
- Skolyoz önleyici rehabilitasyon
- Hidroterapi
- Göğüs Fizyoterapisi/Postural drenaj egzersiz ve önerileri

SMA, BAKIM YÖNETİMİ

İMMÜNİZASYON
- Paliviumab (ilk 2 yaşta RSV sezonunda)
- İnfluenza aşısı (6 aylıktan sonra yılda bir)
- Pnömokok aşısı (yıllık)

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Ciddi solunum yetmezliği	• *SEPN1*, *COL6*, *LMNA* ilişkili KMD	• Konjenital miyastenik sendrom (CHAT), Pompe hastalığı, nemalin miyopatisi
Kardiyak tutulum	• αDG: Hızlı gelişen dilate kardiyomiyopati (FKRP, FKTN) • Kor Pulmonale: *COL6*, *SEPN1* • Ani kardiyak olay/ölüm: *LMNA*	• OR titinopati, MYH7 ilişkili miyopati
Kreatin kinaz düzeyi	• Normal veya hafif yüksek • *SEPN1* ilişkili KMD • *LMNA* ilişkili KMD • Ullrich-KMD	• Konjenital miyopatiler, konjenital miyastenik sendromlar, nörojenik hastalıklar
	• Yüksek • *LAMA2* ilişkili KMD'de sürekli yüksek • αDG'de genellikle yüksek	• Musküler distrofiler, inflamatuar miyopatiler, Pompe hastalığı
	• *CK düzeyi doğum sonrası ilk birkaç gün yüksek olabilir, 10. günden sonra normale döner.*	
Beyin MRG	• Beyin MRG Normal • Ullrich KMD • *SEPN1* ilişkili KMD • *LMNA* ilişkili KMD • İntegrin alfa 7 ilişkili KMD • Hafif spektrumda olan bazı αDG ilişkili KMD	• Konjenital miyopatiler, konjenital miyastenik sendromlar, nörojenik hastalıklar
	• Beyin MRG Anormal • *LAMA2* ilişkili KMD: Ak maddede sinyal değişikliği, daha nadir olarak oksipital pakigiri, serebllar tutulum, ak maddede kistler	• Miyelinizasyon bozuklukları, lökodistrofiler
	• αDG ilişkili KMD: Hidrosefali, pakigiri, lisensefali, serebellar kistler, beyin sapı hipoplazisi (kraniyal grüntüleme normal de olabilir)	• Migrasyon bozuklukları (*LIS1, DCX, ARX, RLn, TUBA1*..), pontoserebellar hipoplaziler (CDG1A, PCH1-8)
Kas biyopsisi	• Normal veya normale yakın olabilir • Orta- ağır miyopatik bulgularla beraber değişken distrofik bulgular, lif tipi dağılımnda değişiklikler: *SEPN1, COL6* • Yer-yer inflamatuar bulgular, ama lif invazyonu yok	• Nöomusküler olmayan hastalıklar, konjenital miyastenik sendomlar, kanalopatiler, bazı metabolik miyopatiler • Konjenital lif tipi disproporsiyon (actin, *RYR1*, *TPM3*)
Kas MRG	• *SEPN1*, *LMNA*, *RYR1* ve Ullrich KMD tiplerinde tanıda yardımcı • Ullrich KMD: Uylukta sartorius, grasilis ve adduktor longus kaslarınn korunduğu, yaygın yağlı infiltrasyon . Vastus kaslarında merkezin göreceli korunduğu, periferin yağlı infiltrasyonu sonucu çember şeklinde hipointensite görüşebilir. • *SEPN1* ilişkili KMD: Sartorius, semimembranöz ve büyük adduktor kaslar tutulur, grasilis kası göreceli olarak korunur. *RYR1* ilişkili kas hastalıklarında da benzer tutulum vardır; ama *SEPN1*'de aksiyel tutulum, özellikle ense fleksörlerinin belirgin hipotrofisi görülür. • *LMNA* **ilişkili KMD:** Ambulasyonun korunduğu hastalarda vastus lateralis ve gastrocnemius medialis tutulur. Ağır formlarında sadece kraniyal, psoas ve ön kol kasları korunur.	

KMD, konjenital musküler distrofi; αDG,alpha distroglikanopati; LGMD, limb girdle musküler distrofi; SMA, spinal musküler atrofi; OR, otozomal resesif; MRG, manyetik rezonans görüntüleme. KMD alt tipleri için gen adları kullanılmıştır.

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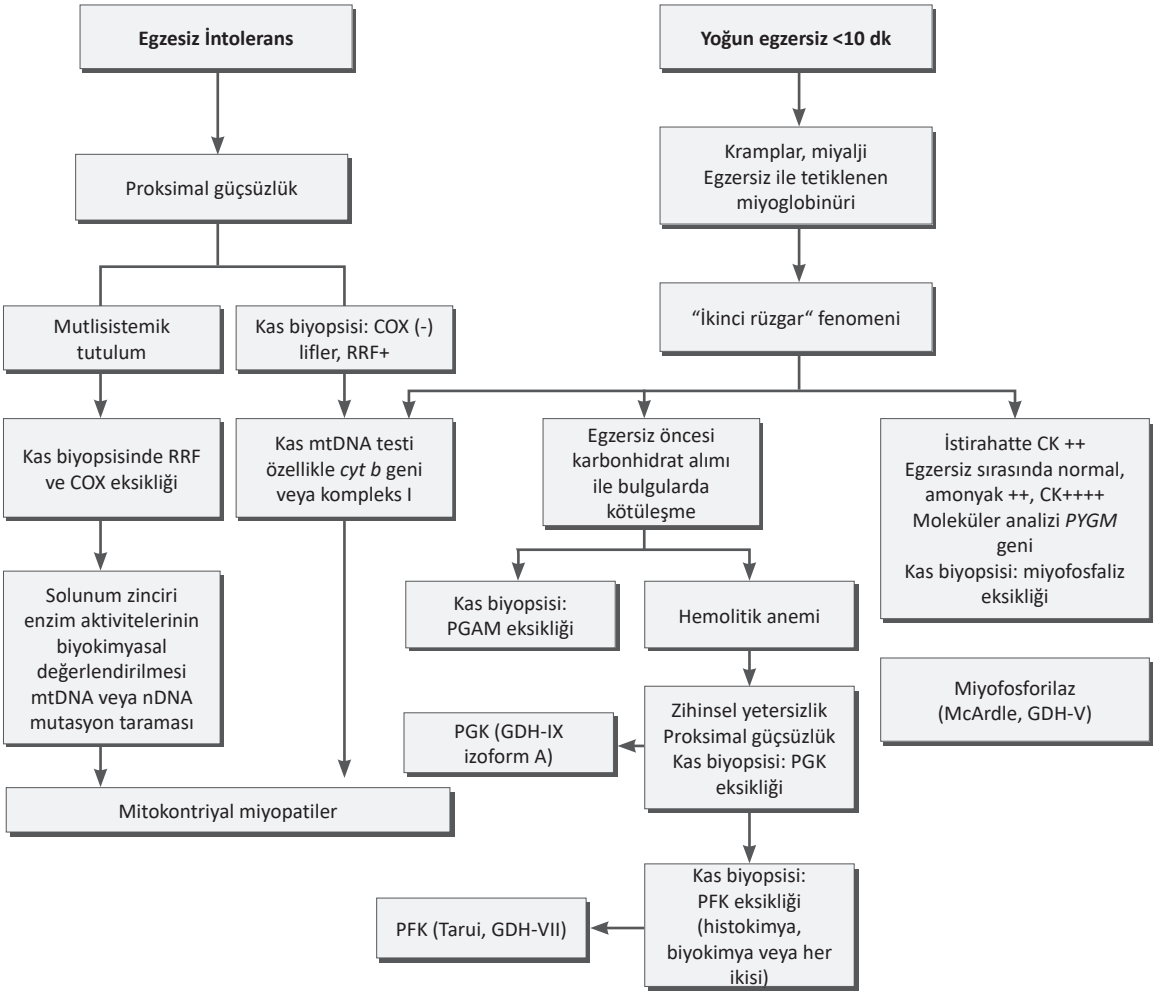

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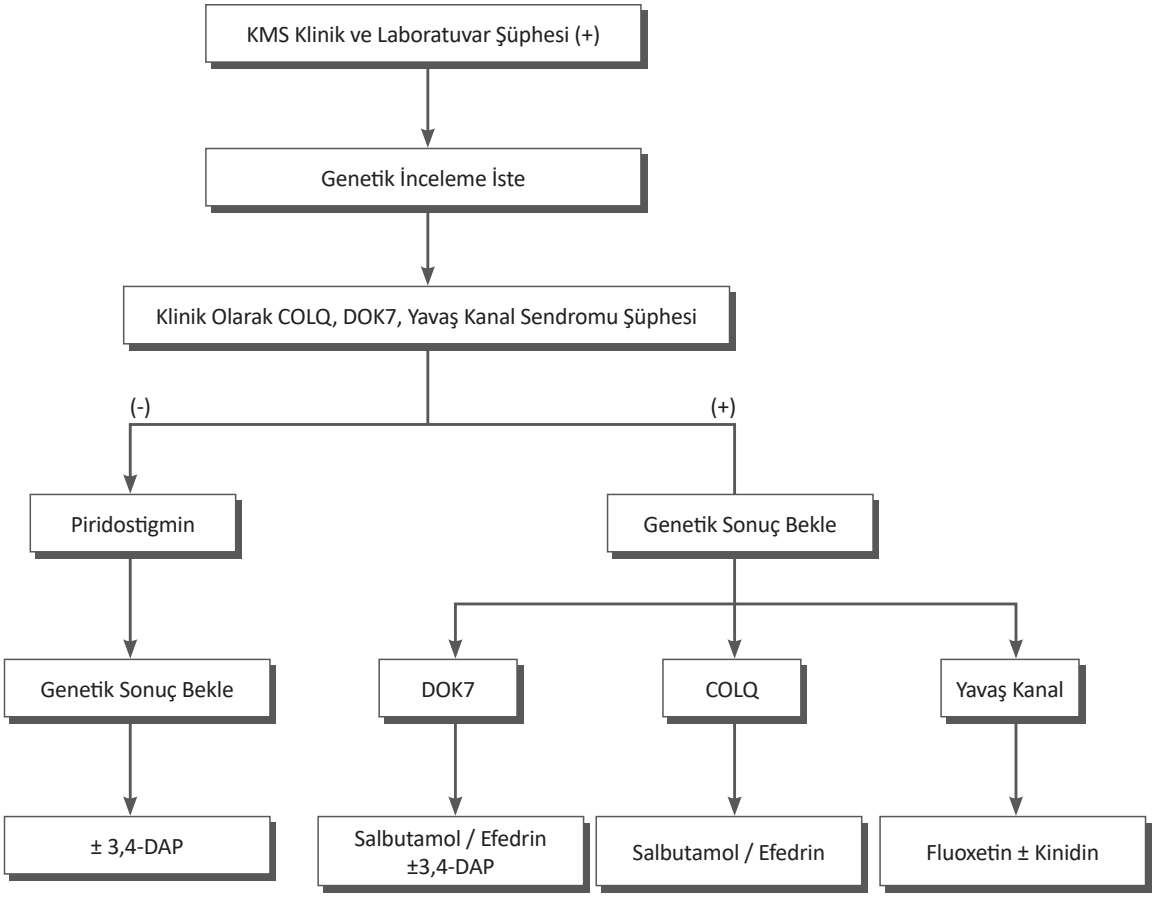
KONJENİTAL MİYASTENİK SENDROM (KMS) – TEDAVİ ALGORİTMASI



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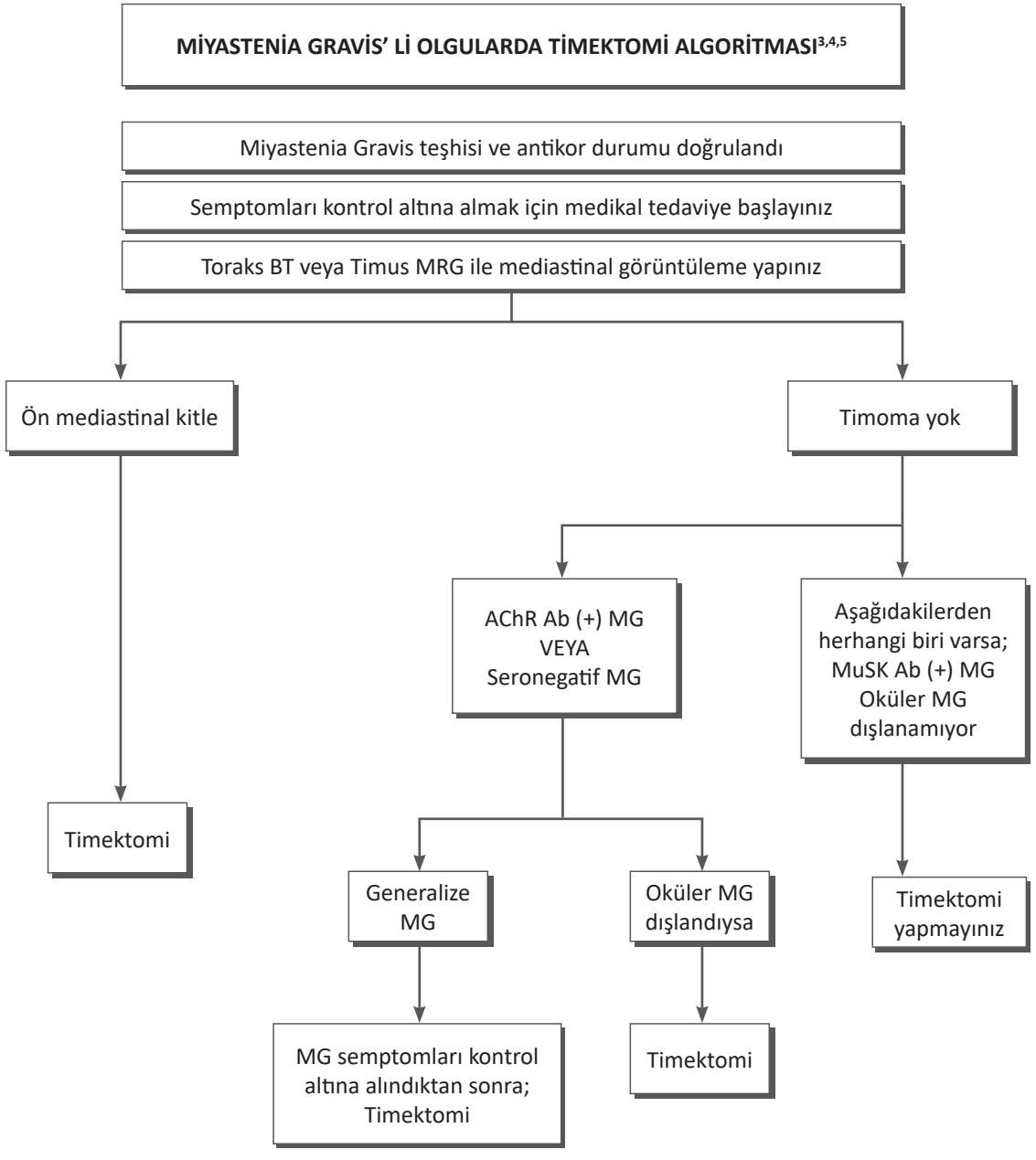

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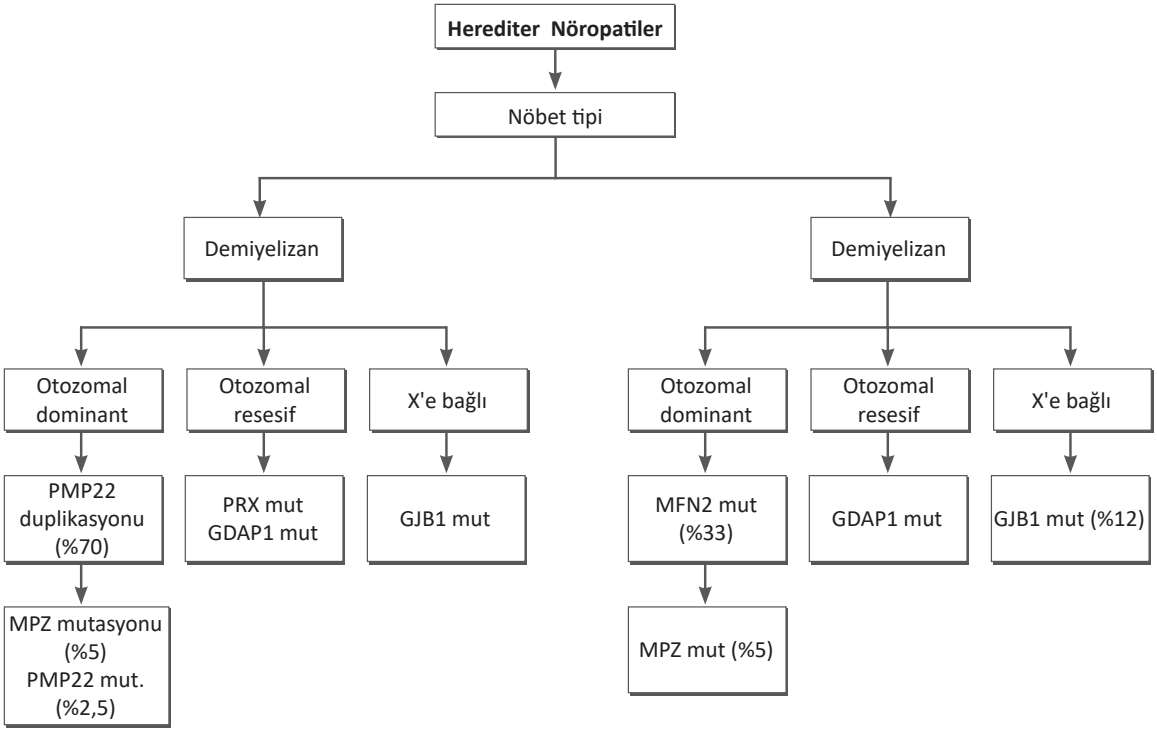

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5. Tutulum paterninin belirlenmesi (Flask paralizi ve…)
+ *miyelopati bulguları;* seviye veren duyu kusuru, mesane/barsak disfonksiyonu → ATM / Spinal kord basısı + *simetrik tutulum, arefleksi* (± *bulbar disfonksiyon, solunum yetmezliği)* → GBS + *febril, pür motor tutulum, asimetrik paralizi (± meningismus)* → Polio ve non-polio (örn enteroviral) AFP/AFM + *motor-duyusal alt ekstremite monoparezisi (im injeksiyon sonrası)* → İnjeksiyon ilişkili nörit + *oftalmopleji, pitoz, bulbar* güçsüzlük → Miller-Fischer varyantı, botulizm, myastenia gravis + *proksimal güçsüzlük, kas hassasiyeti, refleksler korunmuş* → Viral miyozit, dermatomiyozit
6. Planlanan Tetkikler
Tüm olgulara kranial + spinal, kontrastlı + kontrastsız, sagittal + aksiyel kesitler içeren MRG önerilir AFP/AFM düşünülen tüm olgulardan surveyans çalışmaları için gaita ve nazofarenks örneği alınması önerilir
Serum biyokimyasal analizler (eletrolitler, magnezyum, fosfat, kreatin kinaz) X-ray omurga; atlantoaksiyel dislokasyon, vertebral tüberküloz EMG & NCS; GBS BOS analizi (BOS'da hücre sayımı, protein, kültür); meningoensefalit, GBS, AFP/AFM, infeksiyöz miyelopatiler EKG; Hipokalemi İdrar; Porfiri'de porfobilinojenler, Toksik olaylarda idrar
7. Tedavi
Tüm olgulara destek tedavisi (solunum yetmezliği, bulbar güçsüzlük için), *vasküler tonusta azalma ve şok tablosuna yönelik tedavi, otonom instabiliteye* yönelik destek tedavi, yeterli *nutrisyon ve beslenme, nozokomiyal enfeksiyonların* önelenmesi*, immobilizasyon ilişkili komplikasyonların önlenmesi*
Spesifik tedavi; GBS; IVIG 2 gr/kg 2-5 gün ATM; iv metilprednizolon 10-30 mg/kg/gün 3-5 gün (max 1 gr/gün) Spinal kord basısı; Spinal immobilizasyon, cerrahi müdahele, steroidler (akut travmatik miyelopati) Dermatomyozit, myastenia gravis; Immunmodülatör tedaviler Hipokalemi; iv potasyum desteği AFP/AFM; IVIG, plazmaferez, steroid (enterovirüsler hariç), fluoksetin (enterovirüsde)

(DTR derin tendon reflkesleri, AFP akut flask paralizi, AFM akut flask miyelit, ATM akut transvers miyelit, GBS Guillain Barre sendromu,VZV varisella zoster virüs, EMG elektromiyograf, NCS sinir ileti çalışmaları-nerve conduction studies, EKG elektrokardiyografi, IVIG intravenöz immunglobulin, iv intravenöz, gr gram, kg kilogram)

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Akut Güçsüzlük Tedavisi	
Hastalık	**Tedavi**
Akut poliomiyelit	Analjezi, destek tedavisi
Guillain-Barre Sendromu	IV Ig PLEX
Miyastenia Gravis	IV Ig PLEX Neostigmin/pridostigmin Steroid, Azotiopürin Timektomi
Transvers Miyelit	P. MPZ PLEX
Akut Dissemine Ensefalomiyelit	P. MPZ PLEX IV Ig
Porfiri	Glukoz IV hematin
Botulizm	Destek tedavisi
Viral Miyozit	Destek tedavisi
Dermatomiyozit/Polimiyozit	Steroid İmmünsüpresan ilaçlar
Hipokalemik periyodik paralizi	Oral/IV potasyum Asetazolamid

IV Ig: IV immunglobulin, PLEX: Plazmaferez,
P. MPZ: Pulse metilprednizolon

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KRONİK GÜÇSÜZLÜK TANI VE TEDAVİ ALGORİTMASI

BÖLÜM 123

Rojan İPEK[1] Mustafa KÖMÜR[2]

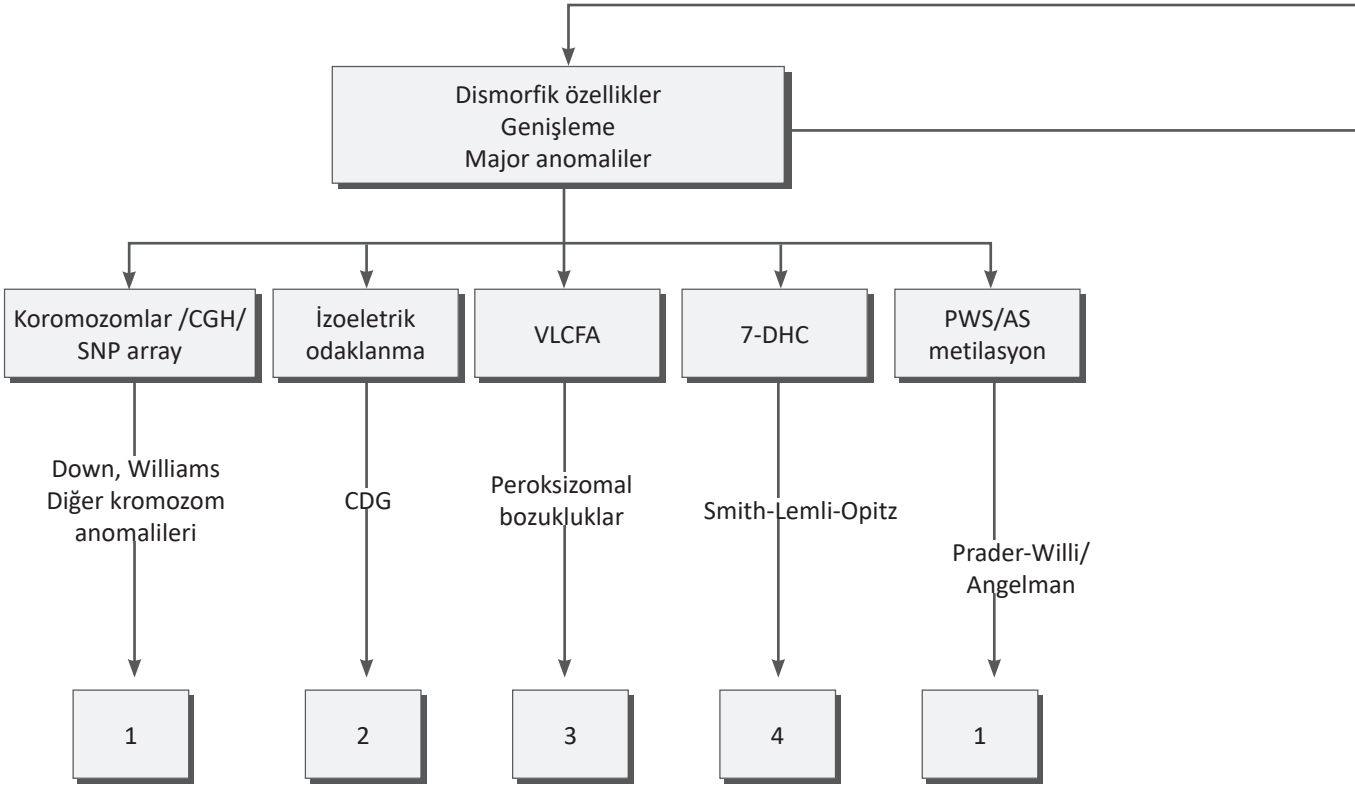
Güçsüzlük, dirence karşı oluşan istemli hareket etme yeteneğinin azalması ya da aktif kas kasılmasında meydana gelen yetersizlik olarak tanımlanır.[1] Güçsüzlük, akut veya kronik güçsüzlük şeklinde görülebilir. Sekiz haftadan uzun süren güçsüzlük kronik güçsüzlük olarak bilinmektedir. Bu bölümde kronik güçsüzlüğe yaklaşımda tanı ve tedavi algoritmaları vurgulanmaktadır.

Hipotoni ve güçsüzlük sıklıkla ilişkili olmasına rağmen aynı anlama gelmemektedir. Hipotoninin değerlendirilmesinde perinatal olaylar önemli olmakla birlikte fetal yutmada azlık, polihidroamnios, hareket azlığı ve malprezentasyon (özellikle makat geliş) yenidoğan değerlendirilmesinde yardımcıdır.[2] Zor doğum veya travma (vakum aleti ve forseps kullanma) öyküsü intrakraniyal kanamalar için risk faktörüdür. Ayrıca yüz ve diğer fizik muayene bulguları (kromozom bozuklukları) tanıya yardımcı olmaktadır. Yapılan muayene bulgularıyla kas gücünün şiddeti ve güçsüzlüğün dağılımı hakkında bilgi sağlanabilmektedir. Hipotonik infantta genellikle spontan hareketlerde azalma gözlenmektedir. Traksiyon cevabı, ventral ve vertikal süspansiyonda görülen anormal postural reflekslerin varlığı şüphe uyandıran işaretlerdir. İlkel reflekslerin olması gereken zaman aralığında yokluğu veya olmaması gereken zaman aralığında devam etmesi ve artmış derin tendon refleksleri birlikteliği serebral hipotoniyi düşündürür. Hipotonik infantta zayıf emme, bilinçte kötüleşme, irkilme, konvülziyon, dismorfik bulgular ve organ malformasyonlarının varlığı metabolik hastalıkların da eşlik edebileceği santral nedenleri akla getirmelidir. Yürümeye başlayan çocuklarda ise oturduğu yerden kalkma, merdiven çıkma, düğme ilikleme ve saçlarını taramada güçlük ile takılma ve sık düşme gibi bulguların varlığı kas gücü değerlendirmesinde çok kıymetlidir.

Kreatin kinaz (CK) ile birlikte sinir iletim hızı/elektromiyografi çalışmaları kas hastalıklarını tanısında yardımcı bulgulardır. Kas biyopsisi nörojenik veya miyojenik nedenlerin ayırımında değerlidir. Ayrıca kas görüntüleme yöntemleri (USG, BT, MRG gibi) günümüzde kullanılmaktadır. Metabolik hastalık şühesinde metabolik taramalar yapılabilir. Kardiyak değerlendirme (EKG, EKO), biyokimyasal çalışmalar ve moleküler genetik analiz yöntemleri planlanabilir.[3] Kronik güçsüzlükle gelen hipotonik infant ve daha büyük çocuklarda tanı ve tedavi algoritması Şekil 1, 2 ve 3'de gösterilmiştir.[4,5] (Şekillerde verilen sayılar önerilen tedavi yöntemi açıklamaları olup bu açıklamalar şekillerden sonra belirtilmiştir.)

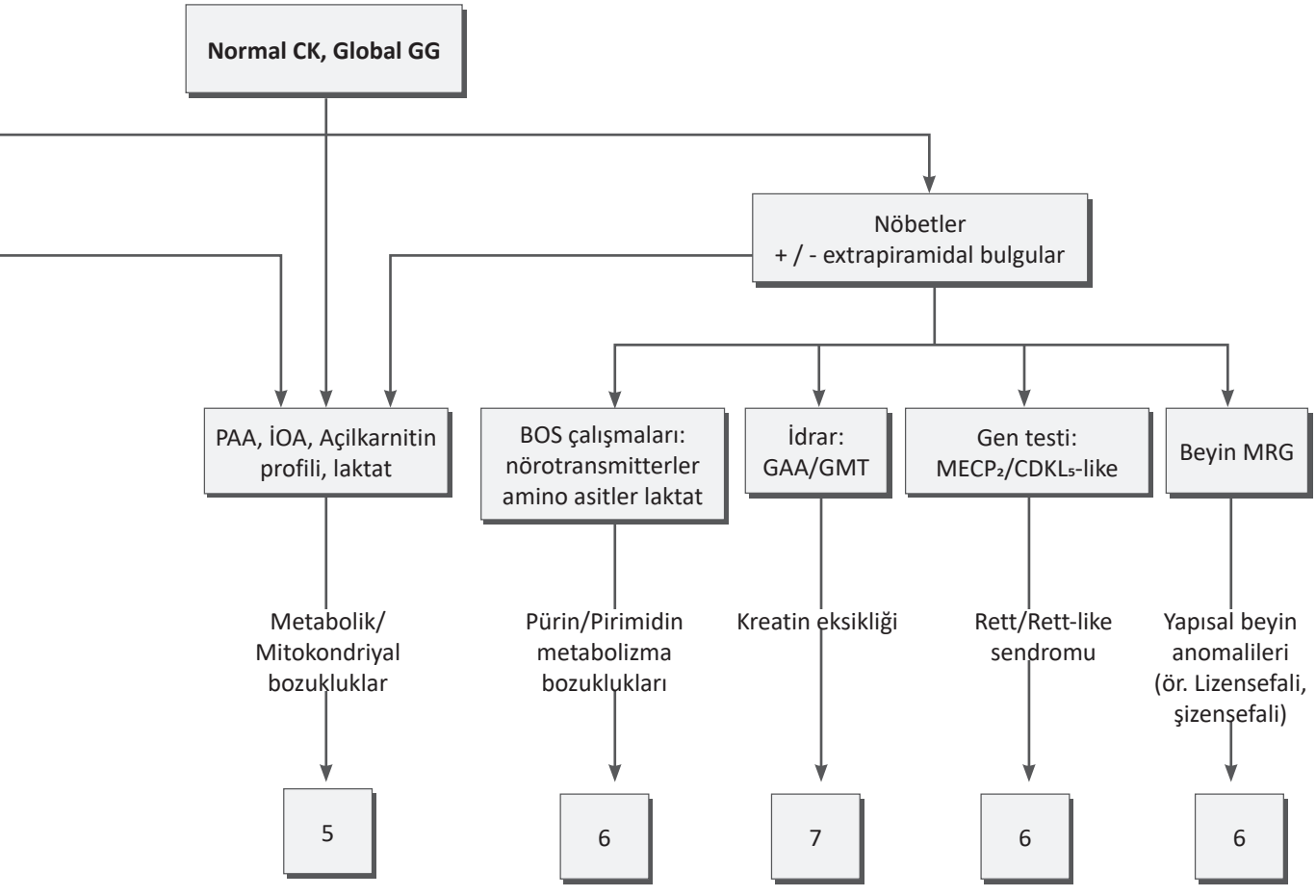
[1] Uzm. Dr., Adıyaman Eğitim ve Araştırma Hastanesi Çocuk Nörolojisi Kliniği, rjnipek@hotmail.com
[2] Doç. Dr., Mersin Üniversitesi Tıp Fakültesi Çocuk Nörolojisi BD., drmustafakomur@yahoo.com

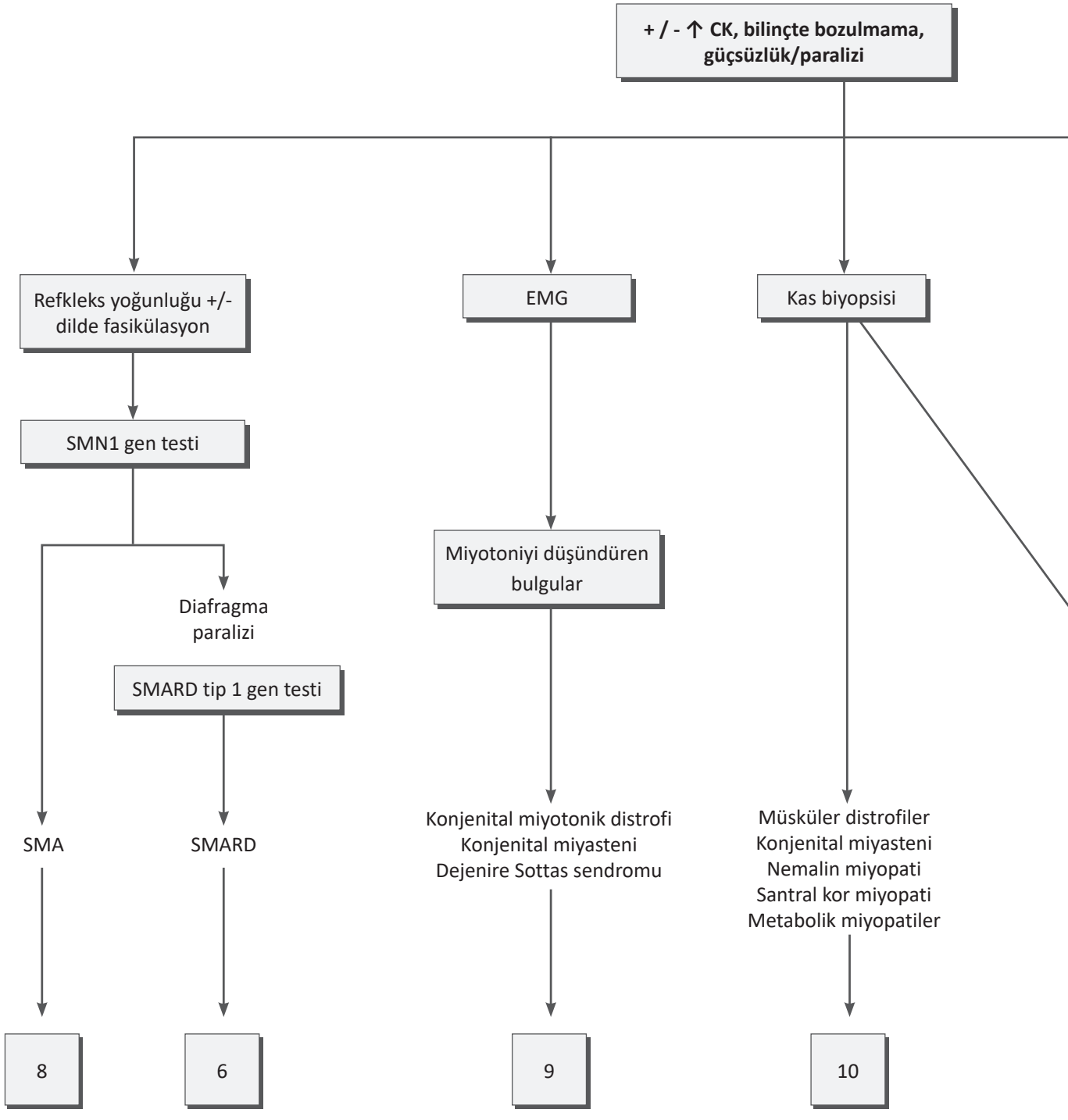
KRONİK GÜÇSÜZLÜK TANI VE TEDAVİ ALGORİTMASI



Şekil 1. Santral hipotoni

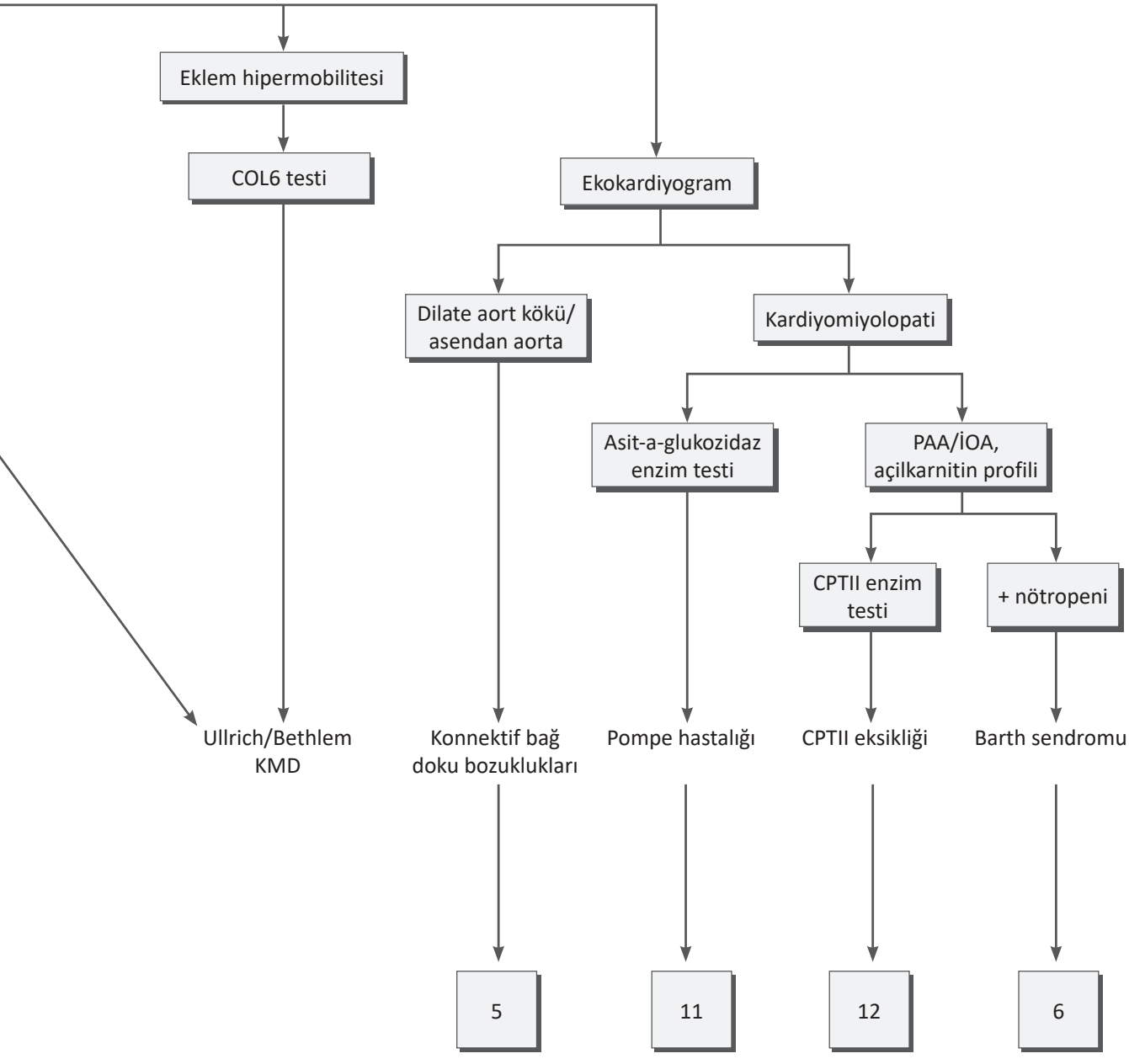
CK: kreatin kinaz; CDG: konjenital glikozilasyon bozuklukları; DHC: dehidrokolesterol; GAA: guanidinoasetat; GG: gelişim geriliği; GMT: guanidinoasetat metil transferaz; PAA: plazma aminoasitleri; İOA: idrar organik aminoasitleri; VLCFA: çok uzun zincirli yağ asitleri

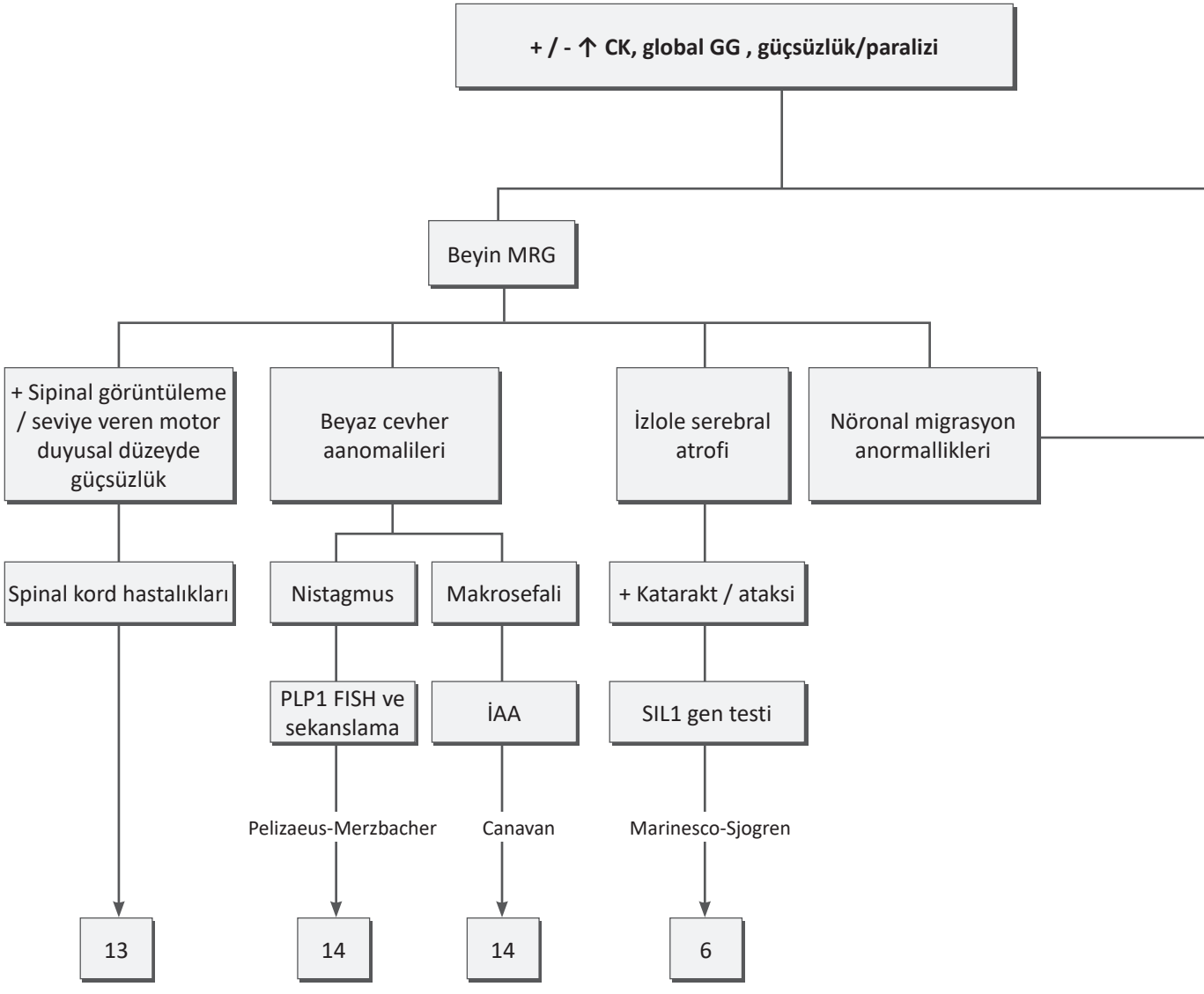

Normal CK, Global GG
Nöbetler
+ / - extrapiramidal bulgular
PAA, İOA, Açilkarnitin profili, laktat
BOS çalışmaları: nörotransmitterler amino asitler laktat
İdrar: GAA/GMT
Gen testi: MECP2/CDKL5-like
Beyin MRG
Metabolik/ Mitokondriyal bozukluklar
Pürin/Pirimidin metabolizma bozuklukları
Kreatin eksikliği
Rett/Rett-like sendromu
Yapısal beyin anomalileri (ör. Lizensefali, şizensefali)
5
6
7
6
6



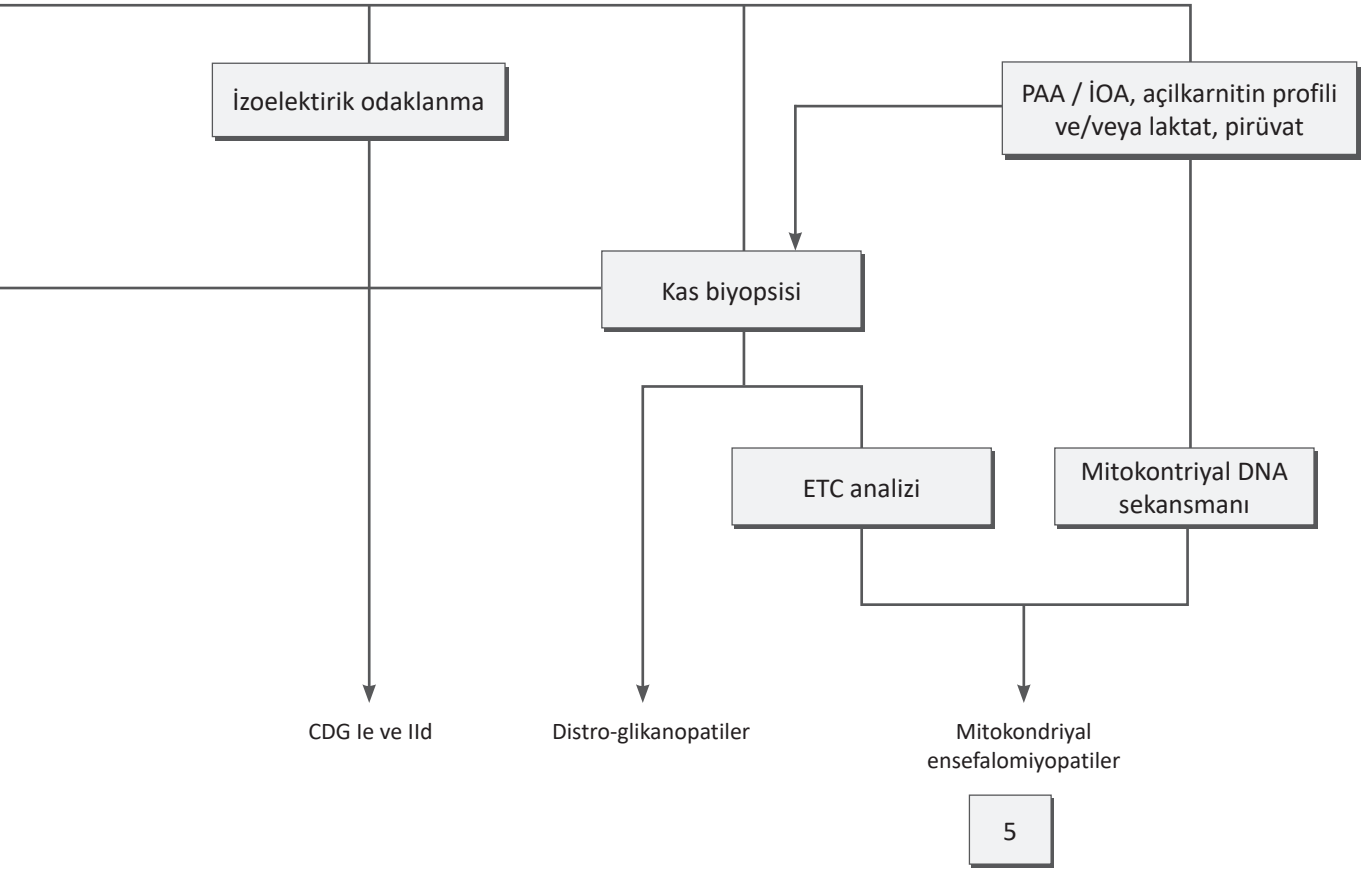
Şekil 2. Periferal hipotoni

CPT II: Karnitin palmitoil transferaz II; EMG: elektromiyografi; KMD: konjenital müsküler distrofi; SMARD: spinomüsküler atrofi ile respiratuvar distres


Eklem hipermobilitesi
COL6 testi
Ekokardiyogram
Dilate aort kökü/
asendan aorta
Kardiyomiyolopati
Asit-a-glukozidaz
enzim testi
PAA/İOA,
açilkarnitin profili
CPTII enzim
testi
+ nötropeni
Ullrich/Bethlem
KMD
Konnektif bağ
doku bozuklukları
Pompe hastalığı
CPTII eksikliği
Barth sendromu
5
11
12
6



Şekil 3. Kombine hipotoni (İAA: idrar aminoasit)


İzoelektirik odaklanma
PAA / İOA, açilkarnitin profili ve/veya laktat, pirüvat
Kas biyopsisi
ETC analizi
Mitokontriyal DNA sekansmanı
CDG Ie ve IId
Distro-glikanopatiler
Mitokondriyal ensefalomiyopatiler
5

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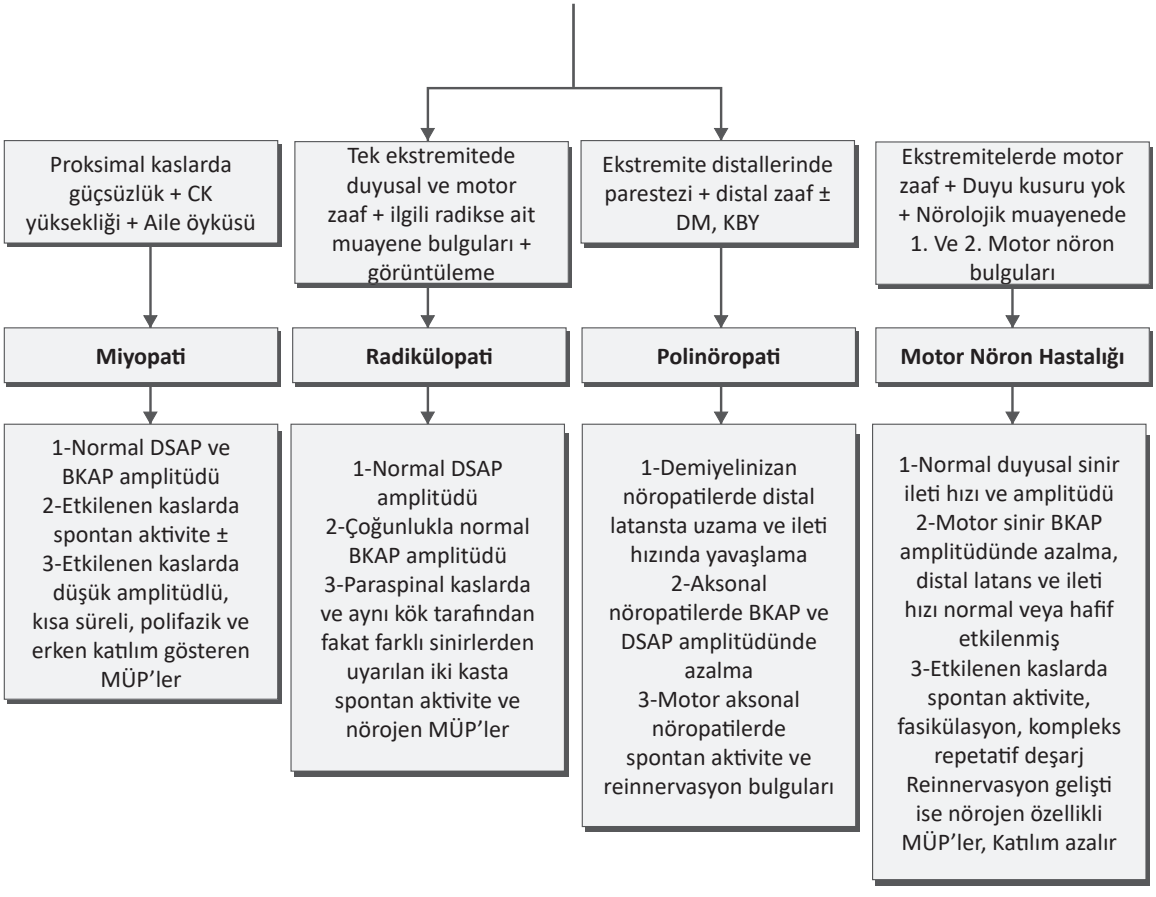

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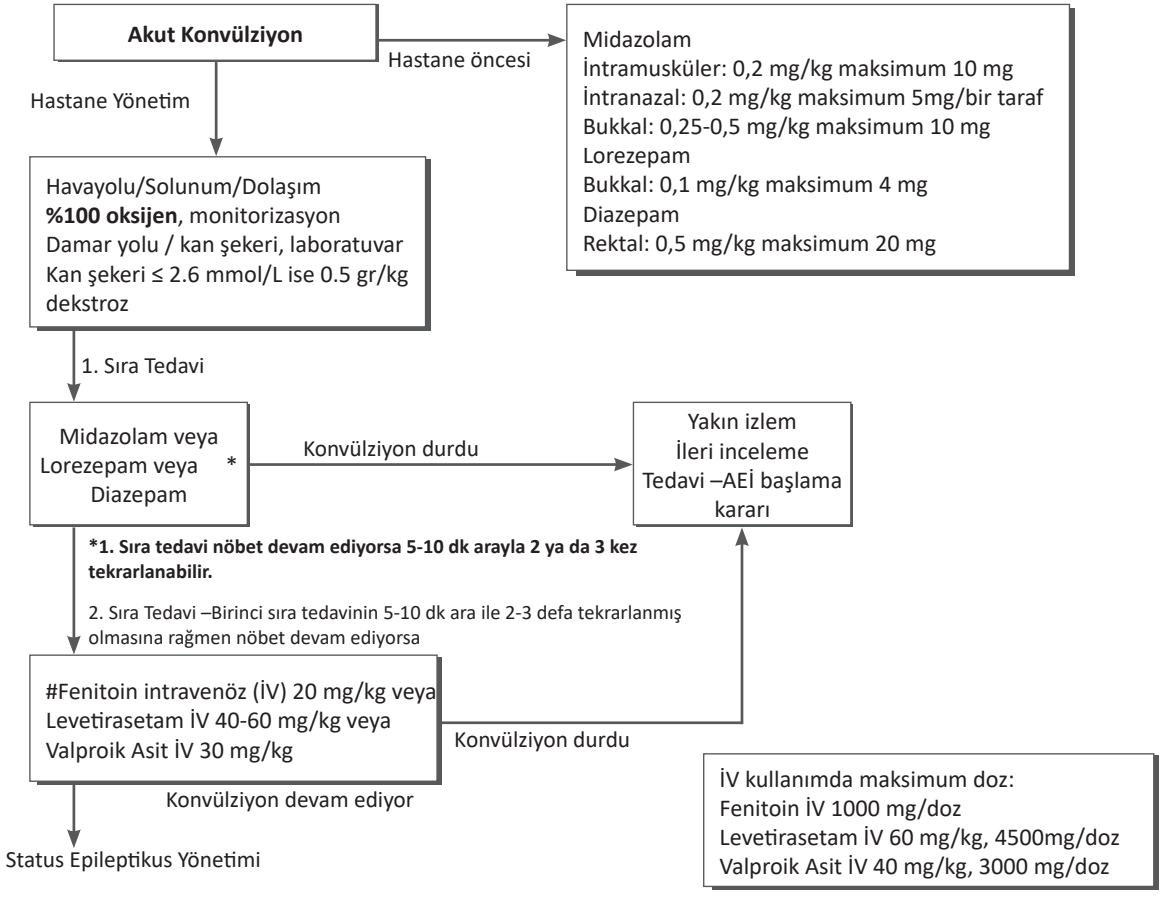

#İnfüzyonu 1 mg/kg/dk hızında başla, %0.9 NaCL ile sulandır, glukoz içeren sıvılar ile aynı damar yolunu tercih etme, aritmi varsa tercih etme

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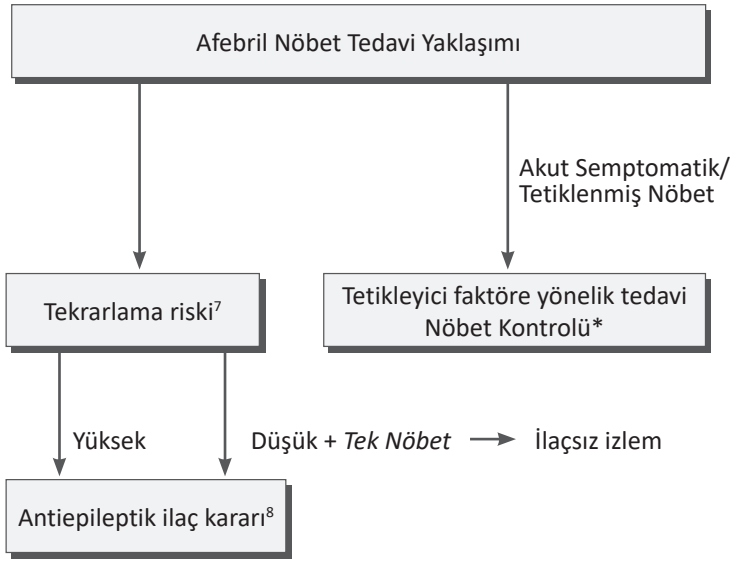
6. İlk afebril nöbetten sonra EEG çekilmesi önerilmektedir. Nöbet tipi, epilepsi sendromu, nöbet tekrar riski, tedavi seçeneklerinin belirlenmesinde ve prognoz tahmininde yardımcı bir tetkiktir. EEG'de epileptiform bulguların yokluğu nöbeti ekarte ettirmez.

EEG çekimi için ideal zamanlama hakkında veriler net olmamakla birlikte anormalliğin en çok nöbetten sonra ilk 24 saat içinde görüldüğü bildirilmektedir. Ayrıca eğer hasta acil servise başvurduysa taburculuk öncesi EEG çektirilmesi gerektiğine dair kanıtlar yetersiz olup hastaya göre düzenleme yapılabileceği bildirilmektedir.



*Konvülziyonla gelen çocuğa akut yaklaşım konusuna ayrı bir algoritma ile değinilmiştir.

7. Herhangi bir tetikleyici faktör olmadan 24 saatten daha uzun arayla en az iki kez afebril nöbet geçirilmesi antiepileptik ilaçların başlanması için gerekli ve yeterli kriter olarak kabul edilmektedir. EEG'de epileptik aktivite görülmesi, altta yatan nörolojik hastalık, aile öyküsü, epileptik sendromla uyumlu bulgular veya anormal nörogörüntüleme bulguları nöbet tekrar riskinin yüksek olduğu durumlardır.
8. İlk afebril nöbetten sonra nöbet tekrar riski ve ilaç yan etkisi göz önünde bulundurularak hastaya ait özellikler değerlendirilerek aile ile birlikte karar verilmelidir.

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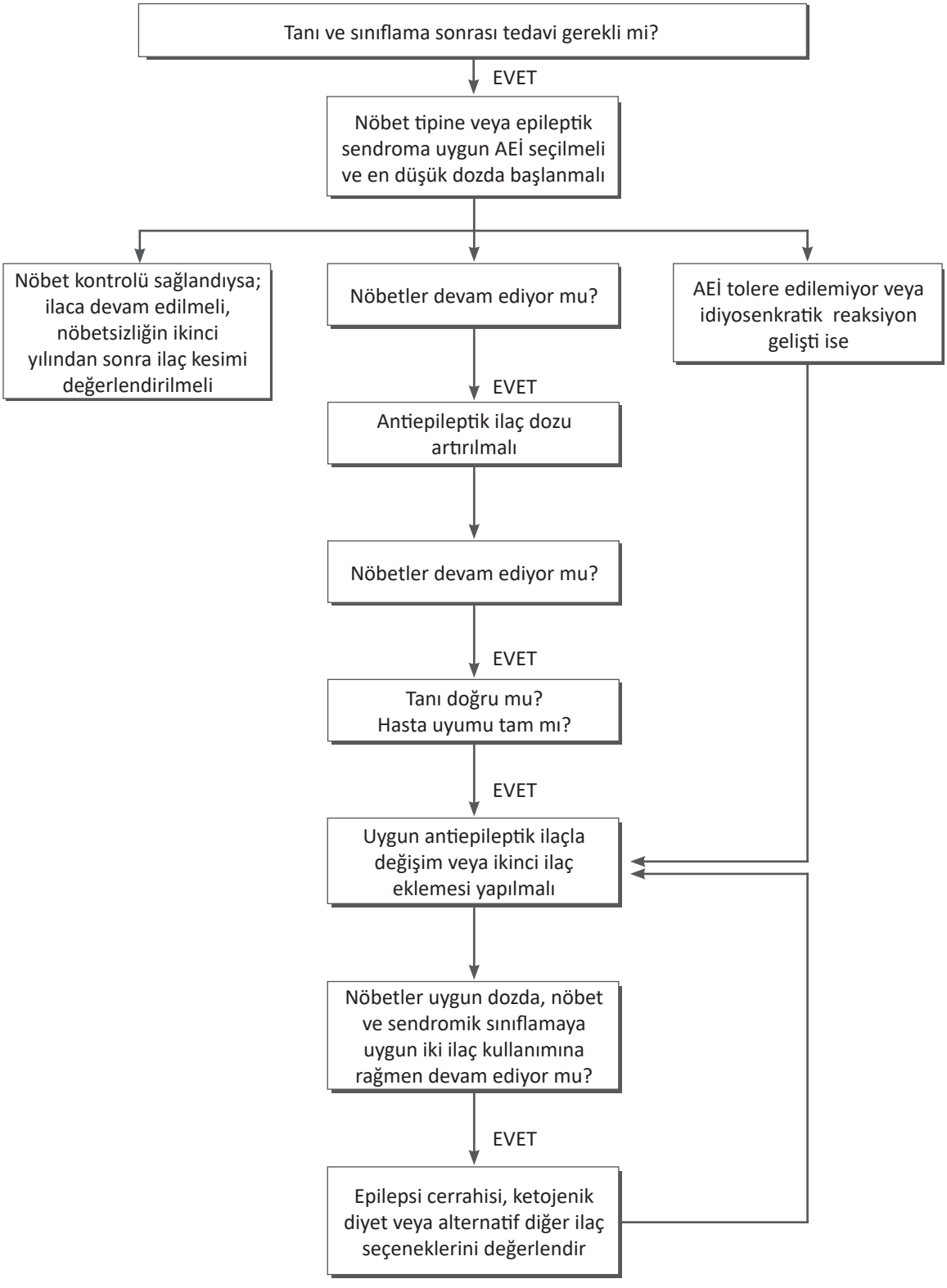

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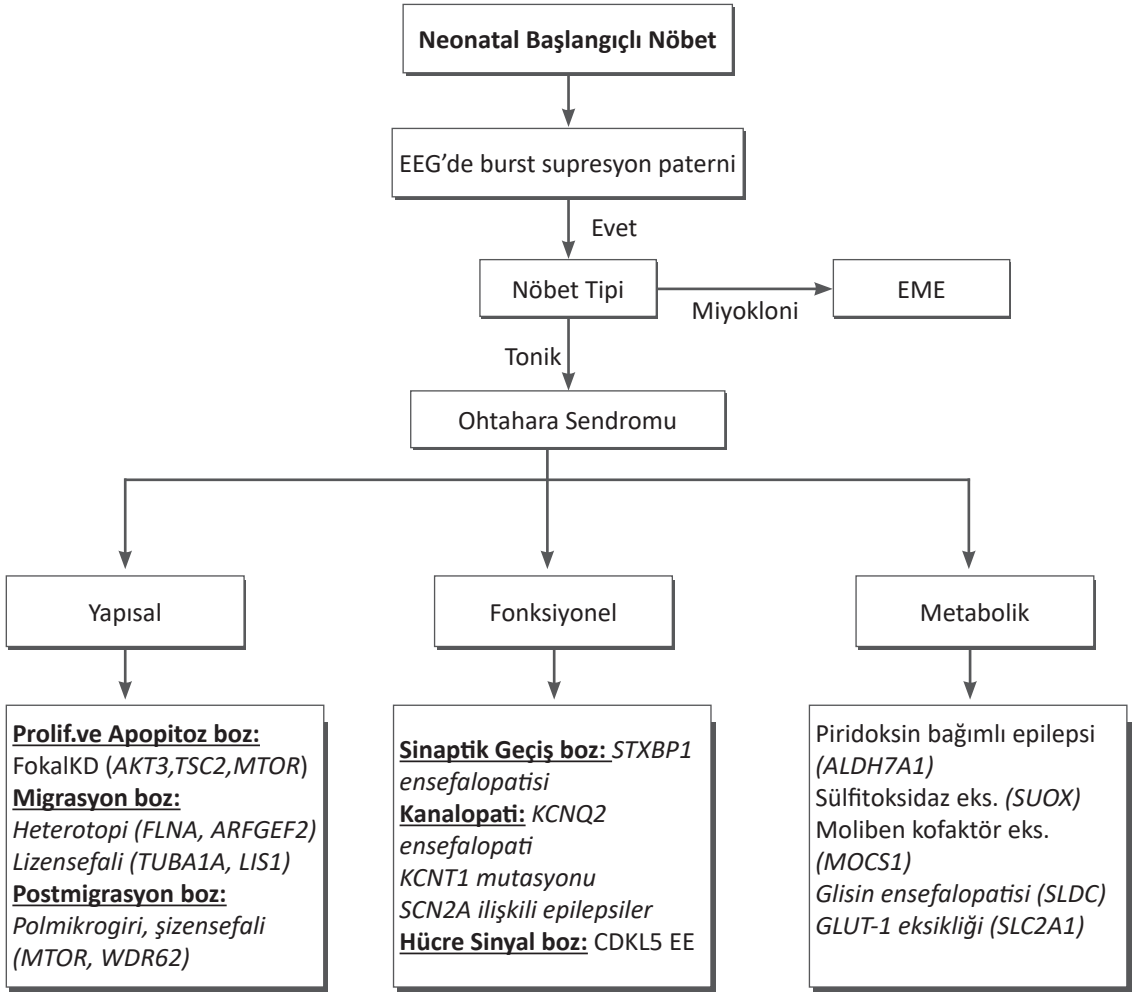

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EIEE/ DEE ve Klinik İpuçları (1) Anormal genitalya • ARX gen mutasyonları [EIEE-1][1,2,4] (2) Awake bruksizm • STXBP1(MUNC18-1) [EIEE-4][13-15] (3) Anormal hareket (Distoni/ataksi/diskinezi/kore/tremor/ataksi/parkinsonyen semptomlar) • STXBP1 [EIEE-4][14-18] • KCNQ2 [EIEE-7][19-22] • SCN8A [EIEE-13][23-25] • GNAO1 [EIEE-17][28,29] (4) Anormal ERG • SLC25A22 [EIEE-3][13] (5) Allerjik hava yolu hastalığı • ST3GAL3 [EIEE-15][26,27] (6) Anormal tonus (sıklıkla konnatal hipotoni) • ST3GAL3 [EIEE-15][26,27] (7) Ani açıklanamayan ölüm • SCN mutasyonları [EIEE-13/6][23-25] (8) Anormal baş çevresi (sıklıkla makrosefali) • SZT2 [EIEE-18][30,31]	↓ HEDEFE YÖNELİK SEKANSLAMA ↓ Array CGH ↓ SNP ↓ WES ↓ KESİNLEŞTİRİLMİŞ GENETİK SONUÇ ↓ FONKSİYONEL GENETİK ÇALIŞMA ↓ HAYVAN MODELLERİNİN GELİŞTİRİLMESİ ↓ HEDEFE YÖNELİK TEDAVİ STRATEJİSİ YAKLAŞIMI

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Tablo 1: İnfantil epileptik spazm sendromu için tanı kriterleri[6]

	Kesin	Uyarılar	İstisnalar
Nöbetler	Sıklıkla kümeler halinde ortaya çıkan fleksör, ekstansör veya miks epileptik spazmlar		
EEG	interiktal: Hipsaritmi, multifokal veya fokal epileptiform deşarjlar (spazmlar başladıktan sonra hızlı bir şekilde görülebilir)	İnteriktal: Normal EEG Burst-Supresyon paterni	İktal: Şüpheli spazmların klinik kayıtları sırasında normal EEG
Başlangıç yaşı	1-24 ay (epileptik spazmlar daha sonra başlayabilir, ancak onlar ISS olmaz)	Başlangıç yaşı 1-2 ay	
Komorbiditeler	Spazmlar başladıktan sonra gelişimsel yavaşlama olur, ancak başlangıçta olmayabilir (önemli gelişimsel bozuklukları olan bir çocuklarda ayırt etmek zor olabilir)		

Tanı için MRG veya iktal EEG gerekli midir?: Tanı için MRG gerekli değildir, ancak altta yatan nedeni değerlendirmek için tavsiye edilir. İnteriktal çalışma, hipsaritmi veya epileptiform anormallikler veya gelişimsel gecikme gösteriyorsa, tanı için iktal EEG gerekli değildir. Hipsaritmi veya epileptiform anormalliklerin yokluğunda iktal kayıt gereklidir.

Muhtemel gelişen sendrom: Beyin hasarı, gelişimsel beyin malformasyonları veya spesifik genetik durumları olan bebekler, önemli interiktal EEG anormallikleri (yüksek amplitüd, zemin ritminde yavaşlama ve/veya multifokal deşarjlar) klinik açıdan epileptik spazmların gelişimi için dikkatle izlenmelidir. Bununla birlikte, kesin nöbet tipinin başlangıcından önce ISS sendromu tanısı konulamaz.

Laboratuvar ile desteklenmeyen sendrom: Tanı kaynaklarının sınırlı olduğu bölgelerde interiktal EEG şiddetle tavsiye edilir. Ancak, eğer EEG yoksa, deneyimli bir klinisyen tarafından (şahsen veya video kaydında)tipik epileptik spazm kümelerine tanık olunursa, diğer klinik zorunlu ve dışlayıcı kriterler ile ISS tanısı konulabilir.

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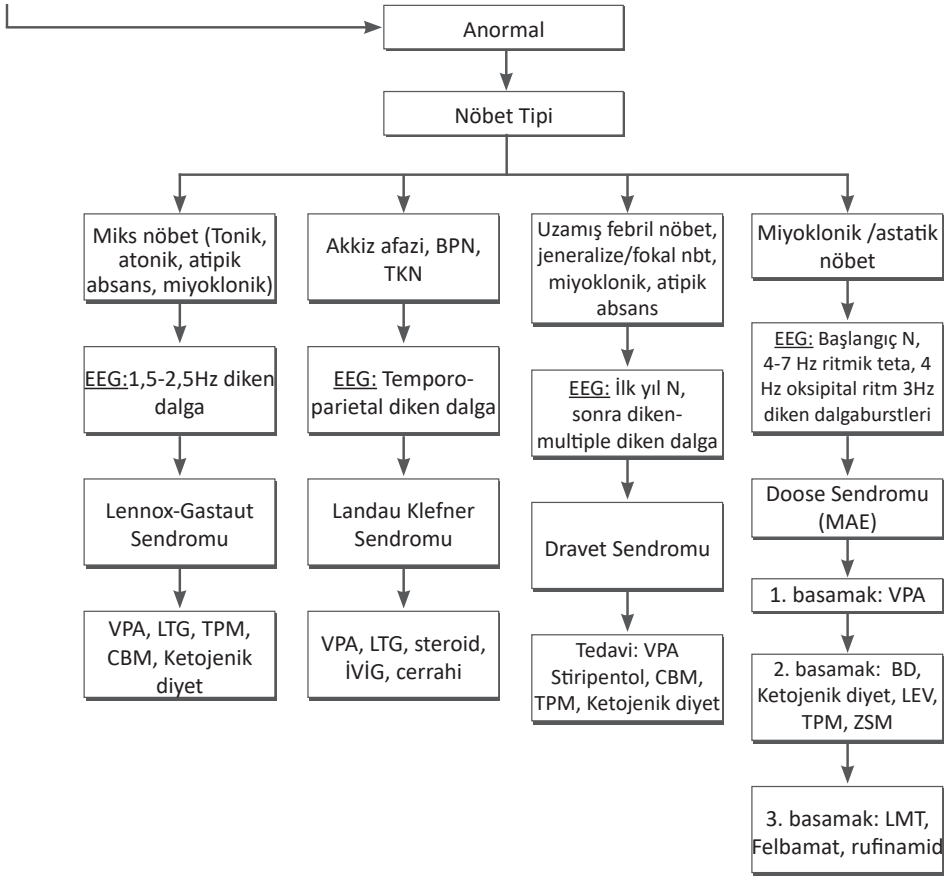

Şekil 1. BPN: basit parsiyel nöbet, KPN: kompleks parsiyel nöbet, TKN: tonik klonik nöbet, KBZ: karbamazepin, OXC: okskarbazepin, VPA: valproik asit, LTG: lamotrijin, ESM: etosüksimid LEV: levatirasetam, TPM: topiramat, CBM: Klobazam, İVİG: intravenöz immunglobulin, ZSM: zonisamid, BD: benzodiazepinler.

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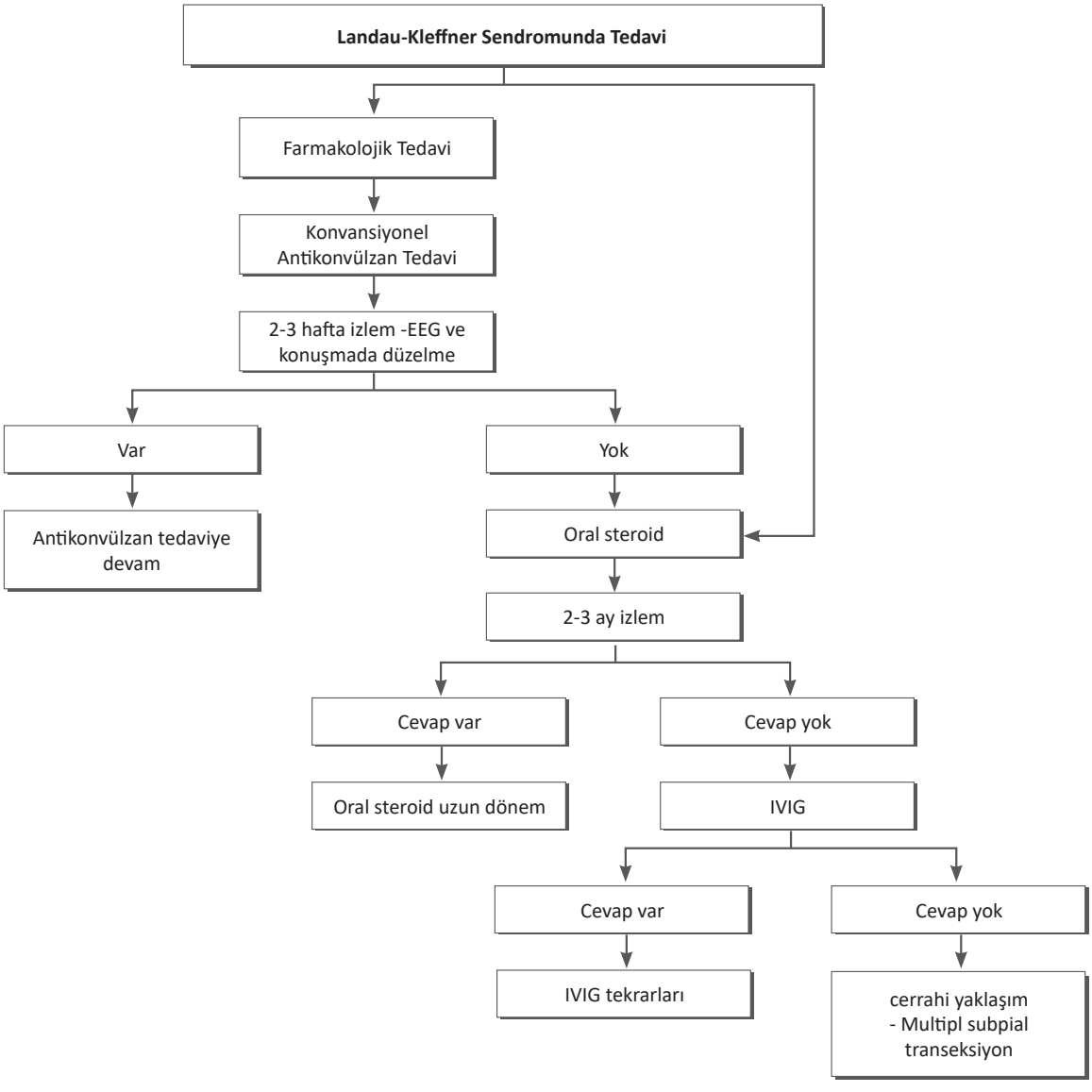
değerlendirilmesi önerilmektedir. Tedavinin takibinde izlenecek önemli parametrelerden biri dil ve nöropsikolojik değerlendirmedir. Bu değerlendirmeler 2-3 ayda bir tekrarlanmalı ve etkinliği görülmeyen tedavilerin yerine yeni tedavi seçenekleri denenmelidir. Bununla birlikte LKS'de dalgalı bir klinik seyir olması ve öngörülemeyen remisyonların olması nedeniyle tedavi etkinliğinin belirlenmesi zorlaşabilmektedir (Şekil 2).[1-3]



Şekil 2. Landau-Kleffner sendromunda tedavi yaklaşımı

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Tablo 1. Uykuda diken-dalga aktivasyonu (SWAS) ile birlikte gelişimsel ve epileptik ensefalopati ve SWAS ile birlikte epileptik ensefalopati[8]

	Kesin	Uyarılar	İstisnalar
Nöbetler		Uykuda tonik nöbetler	Epileptik spazmlar
EEG	N-REM uykusunda yavaş (1,5–2 Hz) diken-dalga anormallikleri. Anormallikler uykuda belirgin şekilde aktive olur.	Uykuda jeneralize paroksismal hızlı aktivite (Lennox-Gastaut sendromunu düşünün) Hem uyanık hem de uyku halindeyken <2,5 Hz'lik jeneralize yavaş diken-dalga kompleksleri (Lennox-Gastaut sendromunu düşünün)	
Başlangıç yaşı		>1 ve <2 yaş	<1 yaş veya >12yaş
Başlangıçta gelişim	EEG'de SWAS bulguları ile birlikte bilişsel, davranışsal veya motor regresyon veya geçici duraklama		
Prognoz	EEG genellikle anormal kalsa da, ergenliğin ortalarında EEG'de SWAS paterninde remisyon gözlenir.		

Tanı için MRG gerekli değildir, ancak genellikle altta yatan etiyolojiyi değerlendirmek için yapılır.

Tanı için uyku EEG'si zorunludur.

Laboratuvar desteği olmayan sendrom: Kaynakların sınırlı olduğu bölgelerde, bu sendroma uyku EEG'si olmadan varsayımsal olarak tanı konulamaz.

Not: Vakaların büyük çoğunluğunda uyarı kriterleri yoktur, ancak nadiren görülebilir. Onların varlığı, sendromun tanısında ve diğer durumların değerlendirilmesinde dikkatli olunması ile sonuçlanmalıdır.

Kısaltmalar: EEG, elektroensefalogram; MRG, manyetik rezonans görüntüleme; N-REM, hızlı olmayan göz hareketi; SWAS, uykuda ani dalga aktivasyonu

[8] Specchio N, Wirrell EC, Scheffer IE, Nabbout R, Riney K, Samia P, et al. International League Against Epilepsy classification and definition of epilepsy syndromes with onset in childhood: Position paper by the ILAE Task Force on Nosology and Definitions. Epilepsia. 2022;63:1398– 1442.

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Tablo 1. Lennox–Gastaut sendromu tanı[1]

	Kesin	Uyarılar	İstisnalar
Nöbetler	Tonik nöbetler, Tonik nöbetlere ek olarak aşağıdakilerden herhangi biri: Atipik absans, Atonik, Miyoklonik, fokal farkındalığı bozulmuş nöbet, jeneralize tonik–klonik, Nonkonvulzif status epileptikus, Epileptik spazmlar		
EEG	1)<2,5 Hz'lik jeneralize yavaş diken-dalga kompleksleri (veya önceki EEG'de bu bulgunun geçmişi) 2)Uykuda jeneralize paroksismal hızlı aktivite (veya önceki EEG'de bu bulgunun geçmişi)	Düşük frekanslarda fotoparoksismal yanıt (CLN2 hastalığını düşünün)	Jeneralize diken-dalga olmadan kalıcı fokal anormallik paterni
Başlangıç yaşı	<18 yaş	>8 yaş	
Prognoz	İlaca dirençli epilepsi, Hafiften ileri dereceye kadar değişen entellektüel yetersizlik		

Tanı için MRG gerekli değildir, ancak genellikle altta yatan etiyolojiyi değerlendirmek için yapılır.

Tanı için iktal EEG gerekli değildir. Bununla birlikte, miyoklonik atonik nöbetlerle giden epilepsiyi düşündürebilecek uyarıları olan veya klinik özellikleri olan bir çocukta ayırıcı tanı açısından düşünülmelidir.

Gelişmekte olan sendrom: Şiddetli DEE'si olan bebeklerin yaklaşık %50'si, örn., IESS veya erken infantil DEE, zamanla Lennox Gastaut sendromuna dönüşür.

Laboratuvar desteği olmayan sendrom: Tanı için yardımcı kaynakların sınırlı olduğu bölgelerde, interiktal EEG'de uyanıklık sırasında jeneralize yavaş diken-dalga paterninin görülmesi gereklidir.

[1]Specchio N, Wirrell EC, Scheffer IE, et al. International League Against Epilepsy classification and definition of epilepsy syndromes with onset in childhood: Position paper by the ILAE Task Force on Nosology and Definitions. Epilepsia. 2022 Jun;63(6):1398-1442.

Not: Vakaların büyük çoğunluğunda uyarı kriterleri yoktur, ancak nadiren görülebilir. Onların varlığı, sendromun teşhisinde ve diğer durumların değerlendirilmesinde dikkatli olunması ile sonuçlanmalıdır.

Kısaltmalar: CLN2, seroid lipofuscinosis tip 2; IESS, infantil epileptik spazm sendromu; DEE, gelişimsel ve/veya epileptik ensefalopati; EEG, elektroensefalogram; MRI, manyetik rezonans görüntüleme

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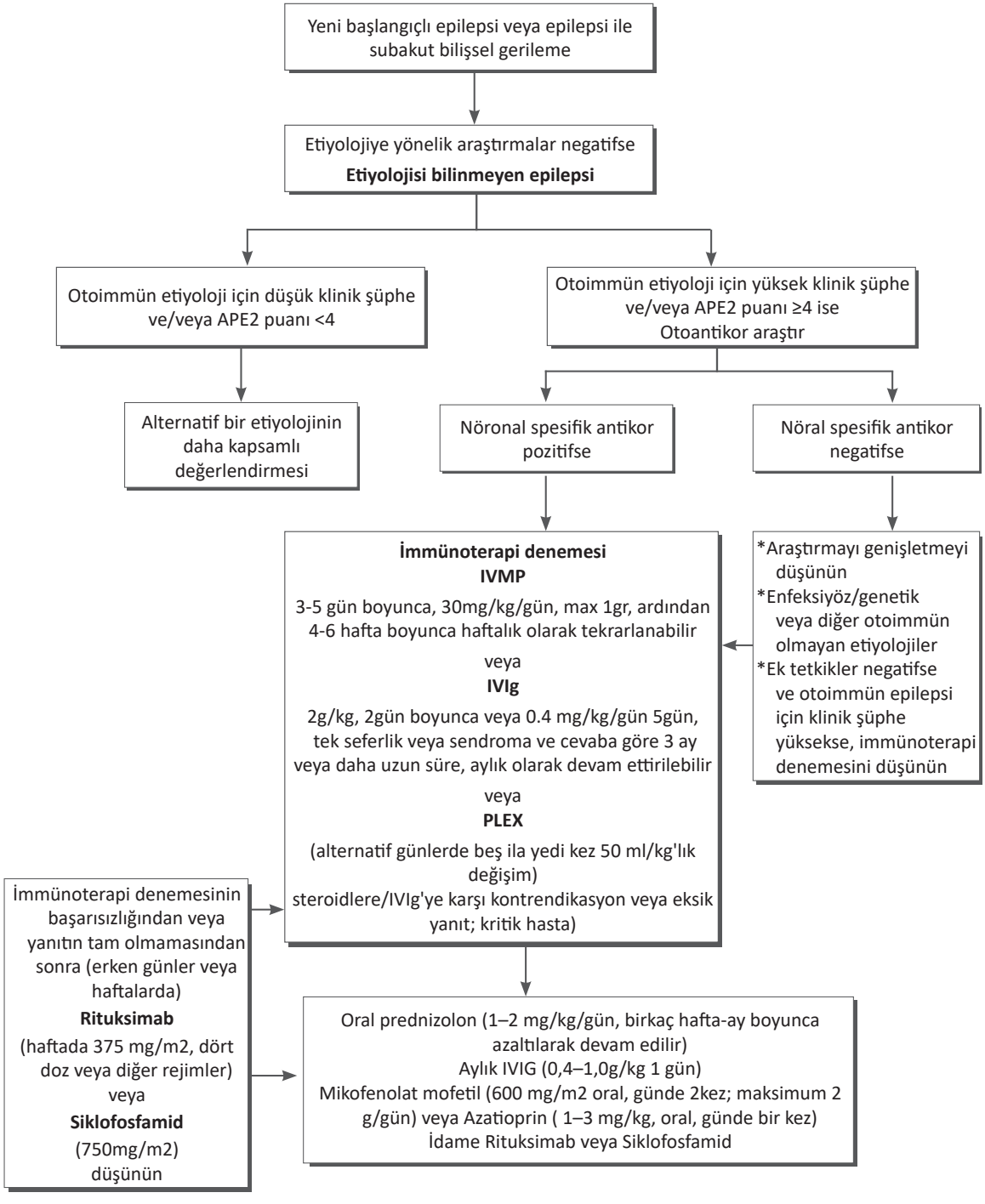

Şekil 2. Otoimmün epilepsi için yönetim algoritması[2]

APE2: Epilepsi ve Ensefalopatide Antikor Prevalansı; IVIg: intravenöz immünoglobulin; IVMP: intravenöz metil prednizolon; PLEX: plazmaferez

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II. Kriptojenik Sebepler (Bilinmeyen)

Tablo 1: Nonkonvulzif status epileptikus tedavi algoritması [4,5,6,7]

Alt tipler	Tedavi Stratejisi
Tipik absans status epileptikus	• PO ya da İV benzodiazepin 4 mg İV lorazepam; ihtiyaç halinde 10 dk sonra tekrarlanabilir. Benzodiazepin etkili olmazsa; PO valproik asit ya da İV valproat • Refraktör olgularda İV levetirasetam, PO topiramat ya da İV lakozamid düşünülebilir (nöroloji konsültasyonu ile karar verilir).
Fokal NKSE (bilinç bozukluğu yok)	• Altta yatan nedenin tedavisine ek olarak; • İV benzodiazepin 4 mg İV lorazepam; ihtiyaç halinde 10 dk sonra tekrarlanabilir • Etkili olmazsa ikinci basamakta; İV fenitoin/fosfenitoin, valproat ve levetirasetam • İkinci basamak tedaviye de refraktör olgularda PO topiramat ve İV lakozamid düşünülebilir (nöroloji konsültasyonu ile karar verilir).
Fokal NKSE (bilinç bozukluğu var)	• Altta yatan nedenin tedavisine ek olarak; • İV benzodiazepin 4 mg İV lorazepam; ihtiyaç halinde 10 dk sonra tekrarlanabilir • Etkili olmazsa ikinci basamakta; İV fenitoin/fosfenitoin, valproat ve levetirasetam • İkinci basamak tedaviye de refraktör olgularda PO topiramat ve İV lakozamid düşünülebilir (nöroloji konsültasyonu ile karar verilir) • Kritik hastalarda ya da uzamış olgularda entübe edip propofol ve midazolam infüzyonu uygulamayı düşün
Komayla beraber NKSE	• İV benzodiazepine ek olarak İV fenitoin/fosfenitoin, valproat ya da levetirasetam gibi ikinci bir ilaç uygula • Entübe edip üçüncü ilaç olarak propofol ve midazolam uygulamayı düşün • PO topiramat, İV lakozamid, İV ketamin ya da nörolojist/ yoğun bakım uzmanı tarafından önerilen tedavi stratejisini izle • Birçok olgu yoğun bakımda izleme ihtiyaç duyar, yoğun bakımda izlemeyi düşün

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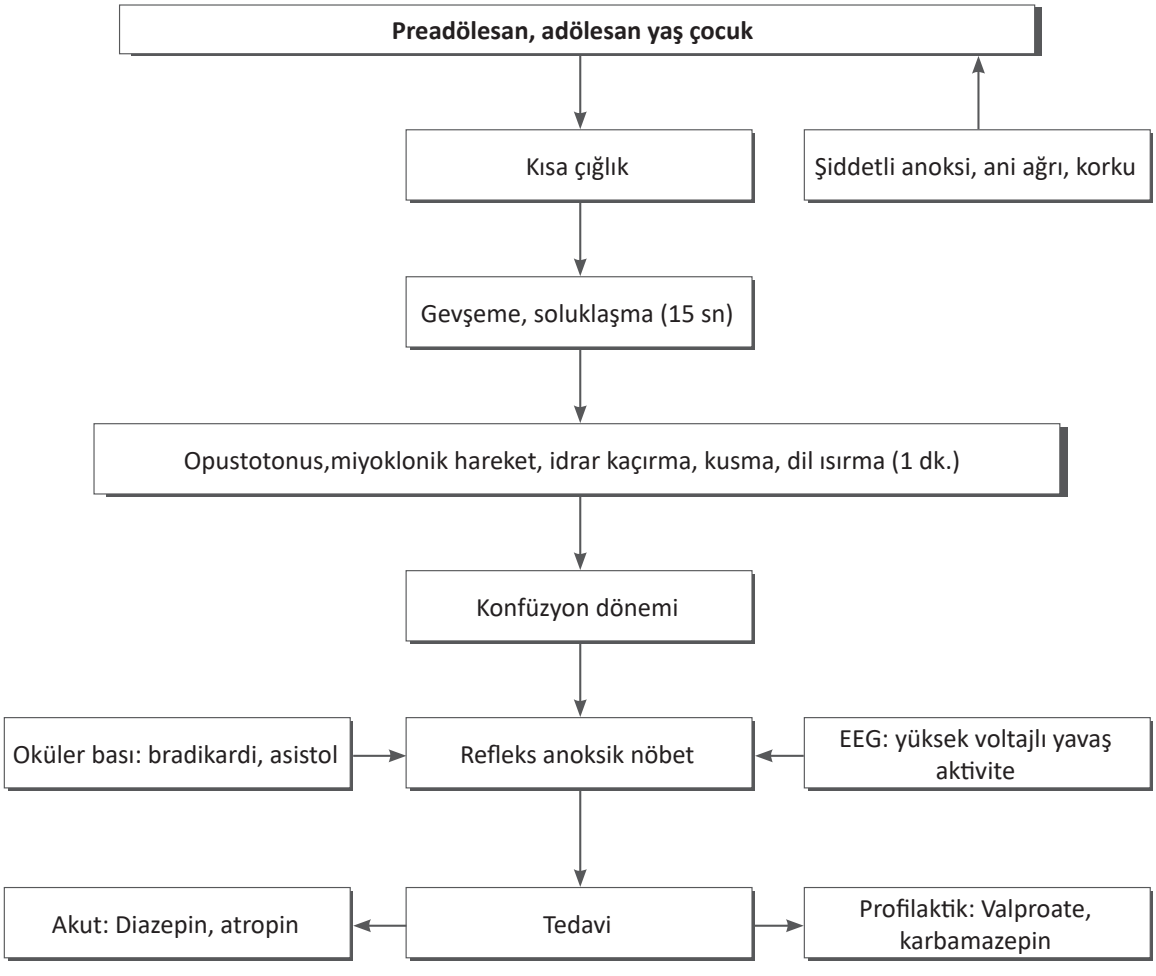
BÖLÜM 170

Gülşen KÖSE [1]



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Erişkin yaş grubunda belirgin semptoma göre veya altta yatan fizyopatolojiye göre bir farmakolojik tedavi algoritması oluşturulabilir iken pediatrik yaş grubunda sadece birkaç ilaç için randomize kontrollü çalışma bulunması nedeni ile zordur.

Propranolol; POTS' ta en yaygın semptomun çarpıntı olması ve bu ilaç ile randomize kontrollü çalışma bulunması nedeni ile ilk tercih olabilir.[18] Ayrıca hipovoleminin ön planda oluğu hastalarda pediatrik hastalarda diğer endikasyonlarda yaygın kullanımı nedeni ile fludrokortizon ve desmopressin kullanılabilir. Midodrinin pediatrik POTS hastalarında özellikle nöropatik fizyopatolojiye sahip olanlarda randomize kontrollü çalışmalarda etkin olduğu gösterilmiştir. Ancak bu ilaç ülkemizde bulunmamaktadır ve yurt dışından bakanlık onayı ile getirilebilmektedir. Diğer ilaçlar için ise randomize kontrollü çalışmalara ve tecrübelere ihtiyaç vardır.

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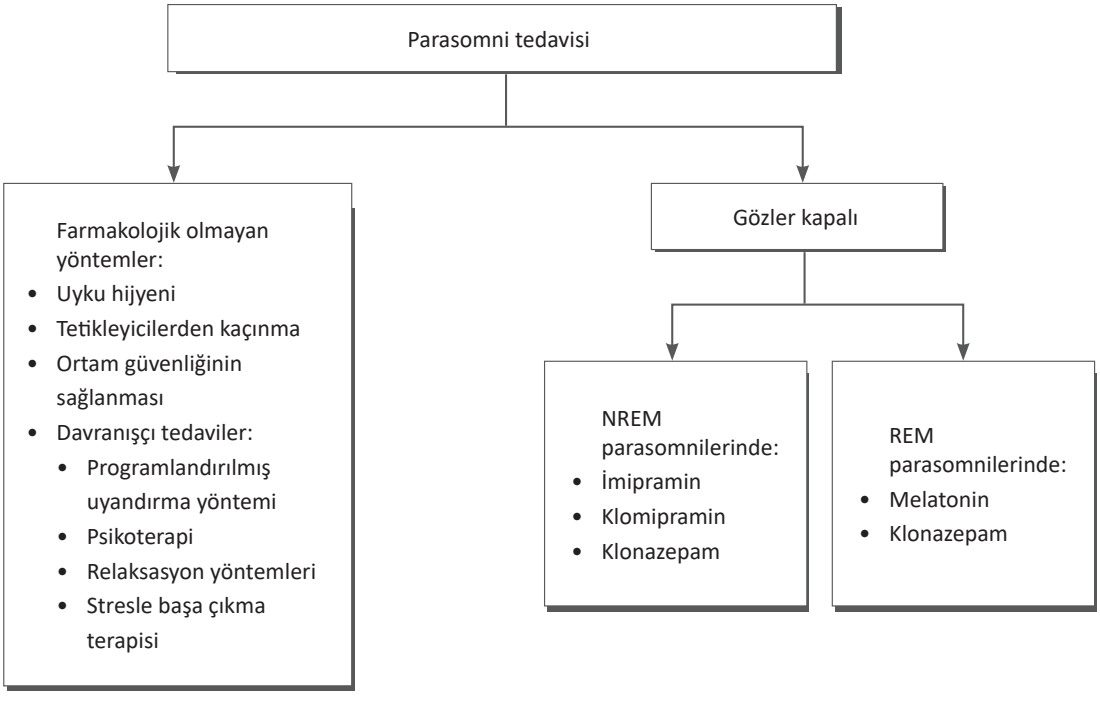
ÇOCUKLARDA PARASOMNİ TEDAVİ ŞEMASI[12]



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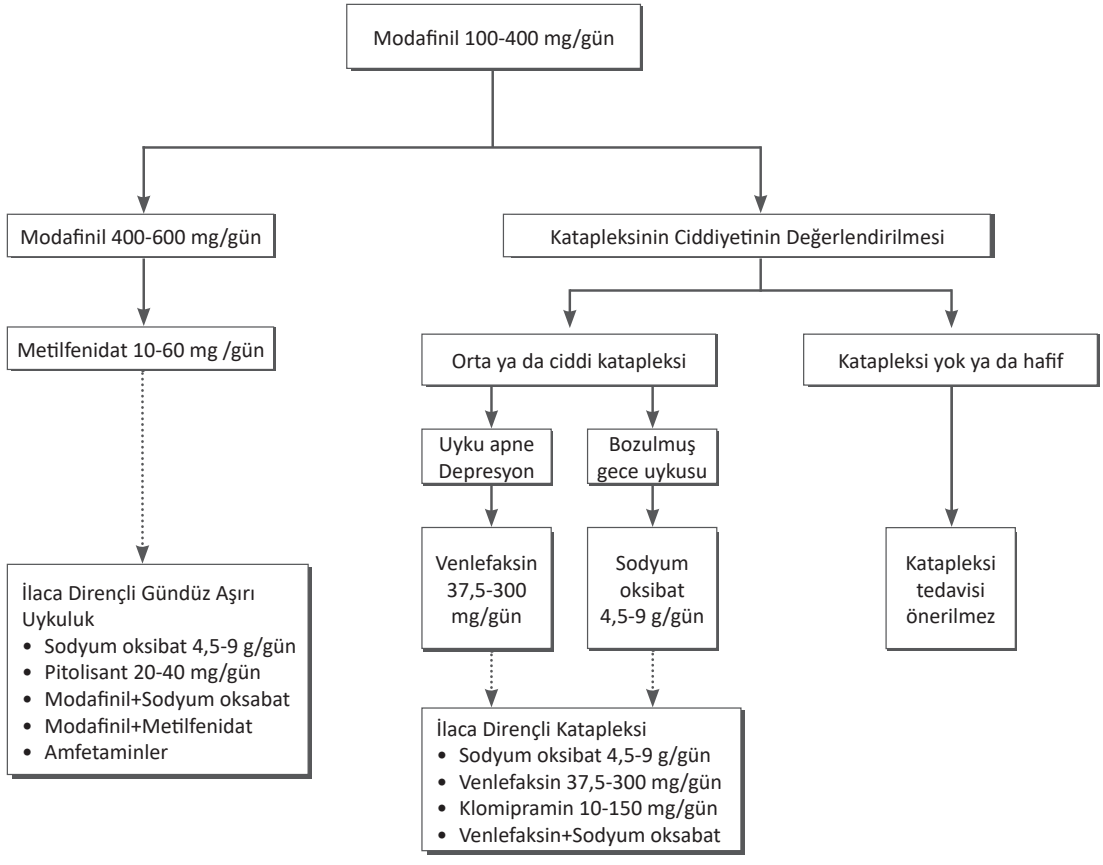

Algoritma 3. Narkolepsi Tip 1 Tedavi Algoritması[3]

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Doku Tanısı

Beyin tümörünün tanısı ve tedavisi cerrahidir. Arzu edilen kitlenin cerrahi ile total rezeksiyonudur.

TEDAVİ

Cerrahi

Kemoterapi

Radyoterapi

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Cerrahi prosedürlerin seçimi, displastik lezyonun boyutuna ve nöbet başlangıç zonuna bağlı olarak değişecektir ve fokal, lober, multilobar ve hemisferik rezeksiyonları veya bağlantı kesilmelerini içerir. [21,22,23] MCD ile ilişkili epilepsiye stereotaktik cerrahi ve lazer interstisyel tedavi (LTT) gibi yeni cerrahi yaklaşımlar uygulanmış ve umut verici sonuçlar elde edilmiştir. [24] LTT, açık cerrahiye kıyasla daha kısa işlem süresi, daha kısa hastanede kalış süresi ve daha düşük komplikasyon oranları ile ilişkilidir.[24,25] Büyük veya multigyral lezyonlar yeterli ablasyon için birden fazla yörüngeye ihtiyaç duyabilir ve bu bireysel vakaya göre ayarlanmalıdır.[25] Bununla birlikte, LTT kesin bir histopatolojik tanıya izin vermez, bu nedenle şüpheli FCD'si olan hastalarda klinik uygulamasını zorlaştırır

Cerrahi sonrasında nöbetsizlik için en önemli faktörün, lezyonun tam rezeksiyonu olduğu bildirilmektedir. Tam rezeksiyon, radyolojik görüntüleme yönteminde saptanan lezyonun ya da invaziv monitorizasyonda saptanan iktal alanın rezeksiyonu olarak tariflenmektedir. Tam rezeksiyon sağlanmış hastalarda nöbetsizlik oranı ortalama %77 saptanırken, kısmi rezeksiyon yapılmış hastalarda nöbetsizlik oranı %20'de kalmaktadır. [26,27] Tam rezeksiyon sağlanamamasının ardındaki en önemli nedenler; Kortikal displazi'nin yeri genellikle MR'da bulgu vermediği için tam olarak saptanamaması ve Kortikal displazinin hassas alanlarda yerleşim göstermesi sebebiyle fonksiyonel nörolojik defisitten kaçınmak amacıyla cerrahi ekibin bilerek kısıtlı rezeksiyon yapmasıdır. [28]

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1. Original figure modified for this publication. Elbers J, Wainwright MS, Amlie-Lefond C. The Pediatric Stroke Code: Early Management of the Child with Stroke. J Pediatr 2015; 167:19. Illustration used with the permission of Elsevier Inc. All rights reserved
2. Medley TL, Miteff C, Andrews I, et al. Australian Clinical Consensus Guideline: The diagnosis and acute management of childhood stroke. International Journal of Stroke 2019, 14: 94–106.
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Tablo 1: Şüpheli bakteriyel menenjit tablosunda ampirik başlangıç antibiyotik tedavisi[3]

Yaş	Olası bakteriyel etkenler	Ampirik başlangıç antibiyoterapi¥	Antibiyotik dozu*
< 1 ay	Group B streptokoklar, *E. coli*, diğer enterobakteriler, *S. pneumoniae*, *Listeria monocytogenes*	Ampisilin *ve* Sefotaksim *veya* Gentamisin	300 mg/kg/gün (6 dozda) 200 mg/kg/gün (4 dozda) 7.5 mg/kg/gün (3 dozda)
≥ 1 ay	*Neisseria meningitidis*, *S. pneumoniae*, *H. influenza*	Sefotaksim *veya* Seftriakson *ve* Vankomisin†	225-300 mg/kg/gün (4 dozda) 100 mg/kg/gün (2 dozda) 60 – 80 mg/kg/gün (4 dozda)

¥ H. influenza tip b'den şüphelenilmesi durumunda tedavi başlangıcında deksametazon da verilmelidir.
*Hastanın yaşı ve renal fonksiyonlarına göre doz düzenlemesi yapılması gerekebilir. BOS Gram boyama ve kültür sonuçlarına göre tedavi revize edilmelidir.
†Adolesanlarda vankomisin 60 mg/kg/gün (3 dozda) başlanabilir. Penisilin dirençli pnömokok varlığında tedaviye rifampisin eklenebilir.
Tablo, Cherry JD, Harrison GJ, Kaplan SL, Steinbach WJ, Hotez PJ editors. Feigin and Cherry's Textbook of Pediatric Infectious Diseases 'dan yararlanarak oluşturulmuştur.

Tablo 2: Etkene göre antibiyotik tercihi[4]

Etken bakteri	Tedavi tercihi
Grup B *Streptococcus*	Ampisilin *ve* aminoglikozit
Escherichia coli	Sefotaksim *ve* aminoglikozit
Listeria monocytogenes	Ampisilin *ve* aminoglikozit
Streptococcus pneumoniae sefotaksim-duyarlı (MİK ≤ 0.5 µg/ml)	Sefotaksim
Streptococcus pneumoniae penisilin-dirençli (MİK ≥ 0.1 µg/ml) sefotaksim-dirençli (MİK ≥ 1.0 µg/ml)	Sefotaksim *ve* vankomisin ± rifampisin
Neisseria meningitidis penisilin MİK ≤ 0.1 µg/ml	Penisilin G *veya* ampisilin
Neisseria meningitidis penisilin MİK 0.1 – 1.0 µg/ml	Sefotaksim
Haemophilus influenzae	Sefotaksim

Tablo, Long SS, Prober CG, Fischer M editors. Principles and practice of pediatric infectious diseases yararlanarak oluşturulmuştur.

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Tablo 2: Lyme nöroborelyoz klinik bulgulara göre tedavi önerileri[2,3]

Antibiyotik	Doz	Süre (gün)
Kranial sinirleri veya periferik sinir sistemini etkileyen Lyme hastalığı		
< 9 yaş		
Amoksisilin (oral)	30 mg/kg, günde 3 defa	21
9 – 12 yaş		
Doksisiklin (oral) (<45 kg ağırlığında)	5 mg/kg/gün 1. gün, ardından; 2.5 mg/kg/gün, günde 1-2 dozda (ciddi enfeksiyonda, 5 mg/kg/gün)	21
Amoksisilin (oral)	30 mg/kg, günde 3 defa	21
> 12 yaş		
Doksisiklin (oral)	100 mg/doz, günde 2 defa veya, 200 mg/doz, günde tek doz	21
Amoksisilin (oral)	1 g, günde 3 defa	21
Merkezi sinir sistemini etkileyen Lyme hastalığı		
< 9 yaş		
Seftriakson (IV) (<50 kg ağırlığında)	80 mg/kg/doz (maks. 4 gr), günde 1 defa	21
9 – 12 yaş		
Seftriakson (IV) (<50 kg ağırlığında)	80 mg/kg/doz (maks. 4 gr), günde 1 defa	21
> 12 yaş		
Seftriakson (IV) (oral tedaviye geçişte doksisiklin kullanılabilir)	2 gr/doz, günde 2 defa veya, 4 gr/doz, günde 1 defa	21
Doksisiklin (oral)	200 mg/doz, günde 2 defa veya, 400 mg/doz, günde 1 defa	21

Tablo, Neurokids - Child Neurology Workbook ve Lyme Disease: Diagnosis and Management; National Institute for Health and Care Excellence kullanılarak oluşturulmuştur.

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İlaçlar	Dozlar	Süre	Yan etkiler
Plekonaril	3x5 mg/kg/doz, oral	7-10 gün	Bulantı, ishal
Asiklovir	0-3 ay: 3x20 mg/kg/doz, intravenöz (İV) >3 ay: 3x10 mg/kg/doz, İV	14-21 gün	Böbrek fonksiyonlarında bozulma
Gansiklovir	2x5 mg/kg/doz, İV	14-21 gün (Tam iyileşme oluncaya kadar)	Anemi, lökopeni, trombositopeni Böbrek fonksiyonlarında bozulma
Foskarnet	2-3x60mg/kg/doz, İV	En az 21 gün (Tam iyileşme oluncaya kadar)	Böbrek fonksiyonlarında bozulma Elektrolit dengesinde bozulma
İVİG*	1 gram/kg/doz	2 gün	İnfüzyon ilişkili reaksiyonlar: ateş, titreme

*İmmün yetmezliği bulunan hastalarda antiviral tedavilere ek olarak kullanılabilir.

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Antiviral Tedavi

- COVID-19 enfeksiyonunda mortalite ve prognoz üzerine etkinliği kanıtlanmış bir antiviral tedavi bugüne kadar bulunamamıştır.
- Hafif, orta hastalık bulguları olan çoğu çocuk hastada destek tedavi (sıvı gıda alımının arttırılması, gerekli olduğunda antipiretik tedavi) yeterlidir.
- Şu an için ağır hastalık bulguları olan veya ağır hastalık riskine sahip, oksijen ihtiyacı olan 12 yaş üstündeki hastalara remdesivir kullanımı B3 düzeyinde, ani başlayan veya giderek artan oksijen ihtiyacı olan tüm çocuklara ise çocuk enfeksiyon hastalıkları değerlendirmesiyle remdesivir CIII düzeyinde önerilmektedir.
- Sağlık Bakanlığı COVID-19 rehberinde ise her çocuk hastanın tedavi açısından ayrı ayrı değerlendirilmesi, ağır pnömoni bulguları olan veya risk faktörü (serebral palsi, nörolojik gelişim sorunları, obezite, genetik sendromlar (trizomi 21), kardiopulmoner hastalık ve immünkompromize çocuklar ve ergenler) olup, hafif ancak ilerleyen pnömoni bulguları olan çocuklara favipravir başlanılması önerilmektedir.

SONUÇ

- Yeni koronavirüs SARS-Cov-2'nin neden olduğu şiddetli enfeksiyonda daha çok akciğer tutulumu ön planda olsa da, MSS ve PSS gibi birçok başka organ sistemi etkilenebilir.
- Çocuklarda COVID-19'a bağlı akut nörolojik tutulum; tat/koku kaybı, baş ağrısı, konfüzyon, febril konvülziyon, status epileptikus, ensefalopati ve inme şeklinde karşımıza çıkabilir.
- Yenidoğanlarda ve çocuklarda COVID-19'un nörolojik tutulumu hala oldukça nadirdir ancak son vaka bildirimleri, gelişebilecek nörolojik komplikasyonlar yönünden dikkatli olunmasının faydalı olacağını düşündürmektedir.

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Yaşa Göre Önerilen Tedavi Rejimleri

- * < **8 yaş:** Rifampisin + Trimetoprim-sülfometaksazol + Seftriakson ±Streptomisin
- * ≥ **8 yaş:** Rifampisin + Doksisiklin + Trimetoprim-sülfometaksazol / Seftriakson ± Streptomisin

İlaç	Doz	Maksimum
Rifampisin	15-20 mg/kg/gün	2x300 mg, oral
Trimetoprim **Sulfometaksozol**	10 mg/kg/gün 40 mg/kg/gün	2x160 mg, oral / intravenöz (iv) 2x800 mg, oral
Seftriakson	100 mg/kg/gün	2x2 gram, iv
Doksisiklin	2,2-4,4 mg/kg/gün	2x100 mg, oral
Streptomisin [1] **Gentamisin** [1]	15 mg/kg/gün 6-7,5 mg/kg/gün	1x1 gram, **intramuskuler (im)** 2x120 mg (iv, im)

[1] Tedavinin ilk 2 haftasında diğer antibiyotik tedavileri ile birlikte.

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Tedavi

- Nörosistiserkozis tedavisi parankimal veya ekstraparankimal bölge tutulumuna, parazit sayısı ve evresine, dejenerasyon ve ilişkili inflamasyona göre bireyselleştirilir.
- KİBAS bulgusu olmayan **canlı kisti** olan tüm hastalara 10 gün AP tedavi verilmelidir.

Tedavi	**Antiparaziter (AP) tedavi**[a]	**Antiepileptik (AE) tedavi**	**Kortikosteroid**	**Cerrahi**
Endikasyonlar	Canlı kisti olan tüm hastalar **Albendazol** 15mg/kg/gün (maksimum 1200 mg/gün, 10 gün) **Prazikuantel**[b]**:** 50mg/kg/gün (maksimum 3600 mg/gün, 10 gün)	Nöbeti olan tüm hastalar **Fenitoin** **Karbamazepin** **Levetirasetam**	AP tedavi alacak tüm hastalar Perilezyonel ödemi bulunan kalsifiye kist Diffüz serebral ödem **Deksametazon** 0,15-0,6 mg/kg/gün	Dirençli nöbete yol açan kalsifiye kist İntraoküler sistiserkoz Medikal tedaviye yanıt alınmayan semptomatik büyük kist varlığında

[a] İntrakraniyal basınç artışı, gözde sistiserkoz, diffüz serebral ödeme eşlik eden ensefalit varlığında antipaziter (AP) tedavi kontrendikedir. Kalsifiye kistlerde AP tedavi verilmesi önerilmemektedir. [b] Çoklu lezyona sahip veya albendazole yeterli yanıt alınmayan çocuk hastalarda PZQ tedaviye eklenebilir.

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- Kesin tanıya sahip hastalarda ise , tüberkülomun cerrahi eksizyonu, genellikle medikal tedaviyi takiben klinik kötüleşme olduğunda gerçekleştirilir. Tüberkülomun total eksizyonu, yalnızca tüberkülomun ön frontal lob, anterior temporal lob veya serebellum gibi beynin elequan olmayan bir bölgesinde olduğu durumlarda denenmelidir.[28]
- Tüberkülom elequan alanda ve derin yerleşimli ise, kısmi eksizyon veya biyopsi
- yeterli olabilir.
- Talamus, bazal ganglionlar ve beyin sapındakiler gibi derin yerleşimli tüberkülom şüphesi olan çocuklarda stereotaktik biyopsi iyi bir seçenek olabilir.[30]
- Beyin tüberkülomunun total eksizyonundan sonra bile antitüberküloz ilaçlar en az 12 ay boyunca uygulanmalıdır. Bazı hastalar 4 yıla kadar antitüberküloz tedavi gerektirebilir. [25,28]

Tüberküloz Beyin Apsesi[31]

- Tedavi seçenekleri arasında basit ponksiyon, sürekli drenaj, fraksiyonel drenaj, burrhole den tekrarlanan aspirasyon ve apsenin total eksizyonu yer alır.
- Posterior fossa yerleşimli lezyonlarda ve multiloküle lezyonlarda total eksizyon önerilmektedir.
- Motor veya konuşma bölgesinde yerleşimli lezyonlarda tercih edilen tedavi apsenin sürekli drenajıdır.
- Kesin tedavi, 12-18 aylık tam bir tüberküloz tedavisi kürü ile desteklenen total eksizyondur.

Kalvaryal Tüberküloz

- Cerrahi eksizyon ve antitüberküloz tedavi kombinasyonu tercih edilen tedavidir.

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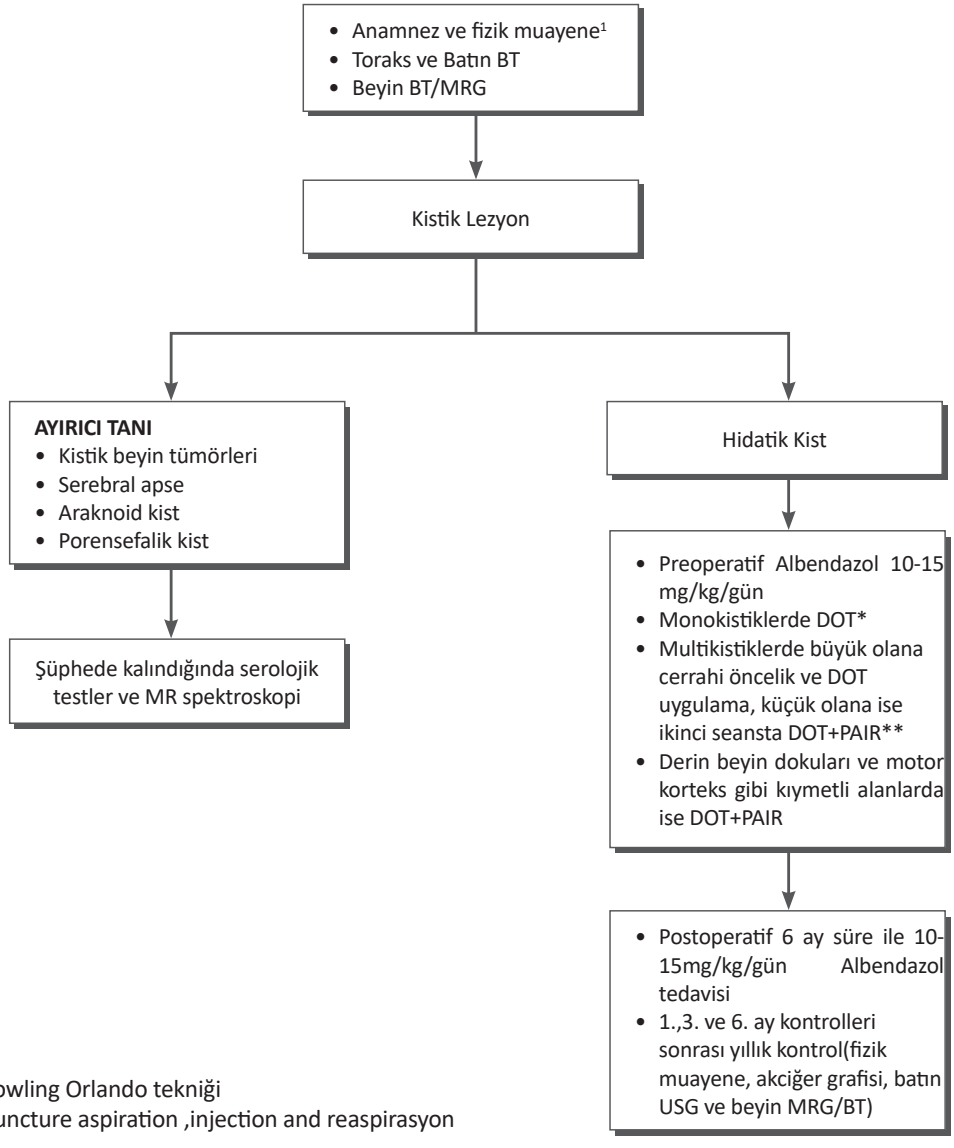
KİST HİDATİK: MEDİKAL VE CERRAHİ TEDAVİ ALGORİTMASI

BÖLÜM 197

Merve Türkay KARAAVCI[1] Mehmet Kürşat KARADAĞ[2] Hakan HADİ KADIOĞLU[3]



* DOT :Dowling Orlando tekniği
**PAIR:Puncture aspiration ,injection and reaspirasyon

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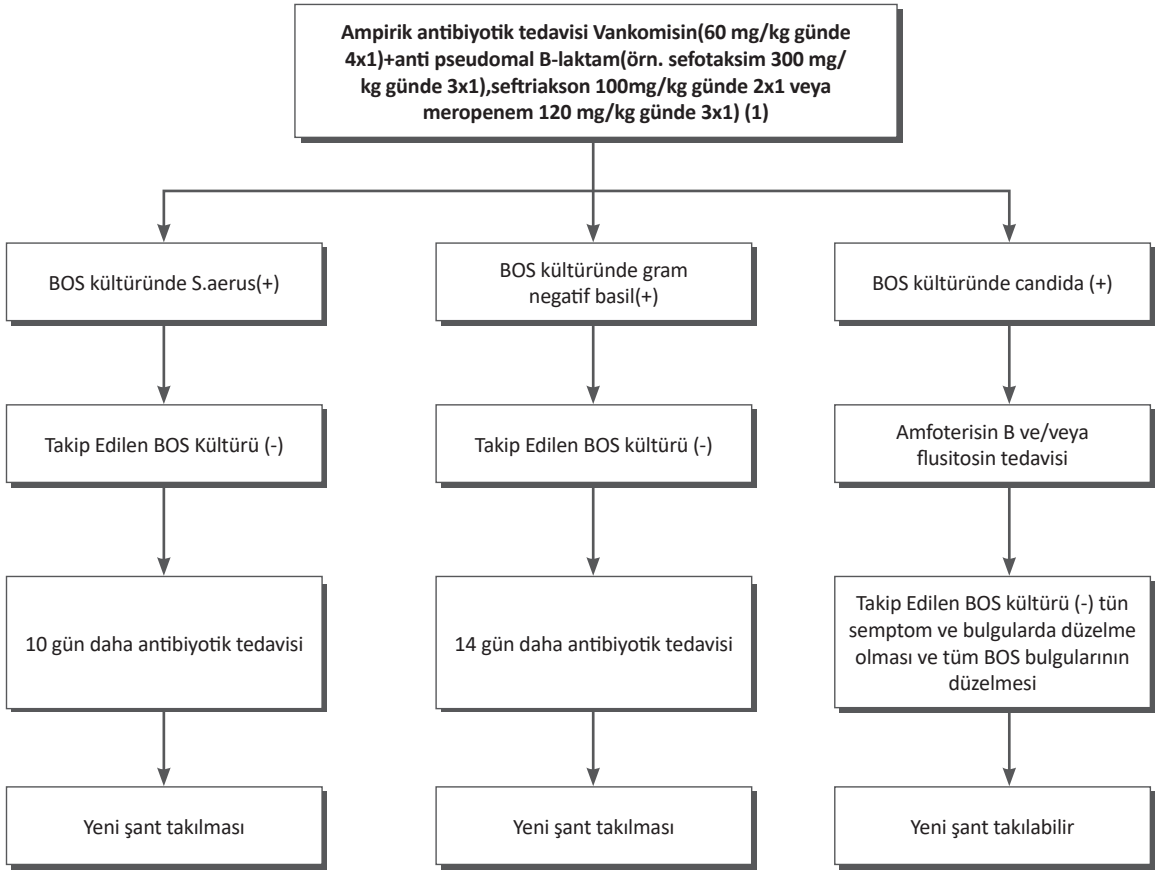

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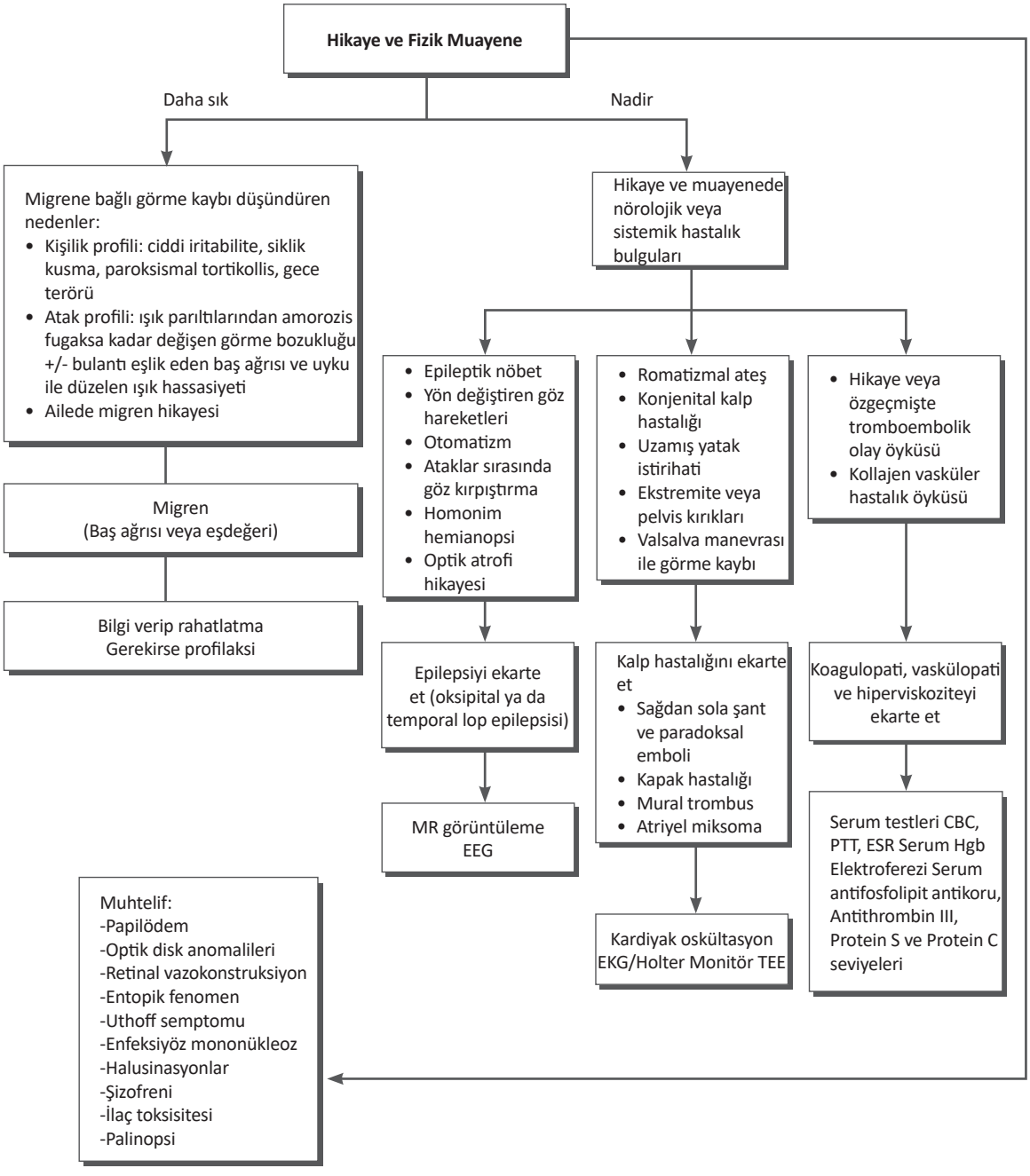

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Tablo 1: Dizartri tiplleri, etyoloji ve semptomlara yaklaşım şeması

Dizartri Tipi	Etkilenen bölge	Etyoloji	Eşlik eden klinik semptom	Konuşma bozukluğu
Spastik	piramidal sistem	Serebrovasküler olaylar Travma Bulber paralizi	Hemiparezi, tetraparezi	Boğuk, genizden, yükselen alçalan tonda
Ataksik	Serebellum	Enfeksiyon, tümör, kanama	Ataksi, dismetri, disdiadokinezi	Patlayıcı, arada tiz ve kalın karışık yanlış yerde vurgulu
Flask	7,9,10,12. Kafa sinir çiftleri Üst motor nöron hasarı	Tümör, kanama, enfeksiyon, ALS, SMA	Kafa sinirlerinin özgül parazileri Kas ve dilde fasikülasyonlar, güçsüzlük	Nazone konuşma, genelde hece bölünmesi ve gramer hatası yok
Hipokinetik Dizartri	Bazal ganglionlar	Parkinson hastalığı	Mimiklerin kaybolması, yavaş hareketler, tremor	Tek düze, monoton, alçak sesle, düşük tonda, sürekli ama anlaşılmaz yutulan heceleri ile konuşma
Hiperkinetik Dizartri	Bazal ganglionlar	Syndenem koresi, SLE, Wilson hastalığı, herediter distoniler, nörometabolik hastalıklar v.b.	Distoni, kore, atetoz, ballismus, tremor	Konuşma başlarken zorlanma, heceler ve kelimeler bölük bölük, uzun duraklamalar, ses uzatmalar şeklinde olan konuşma

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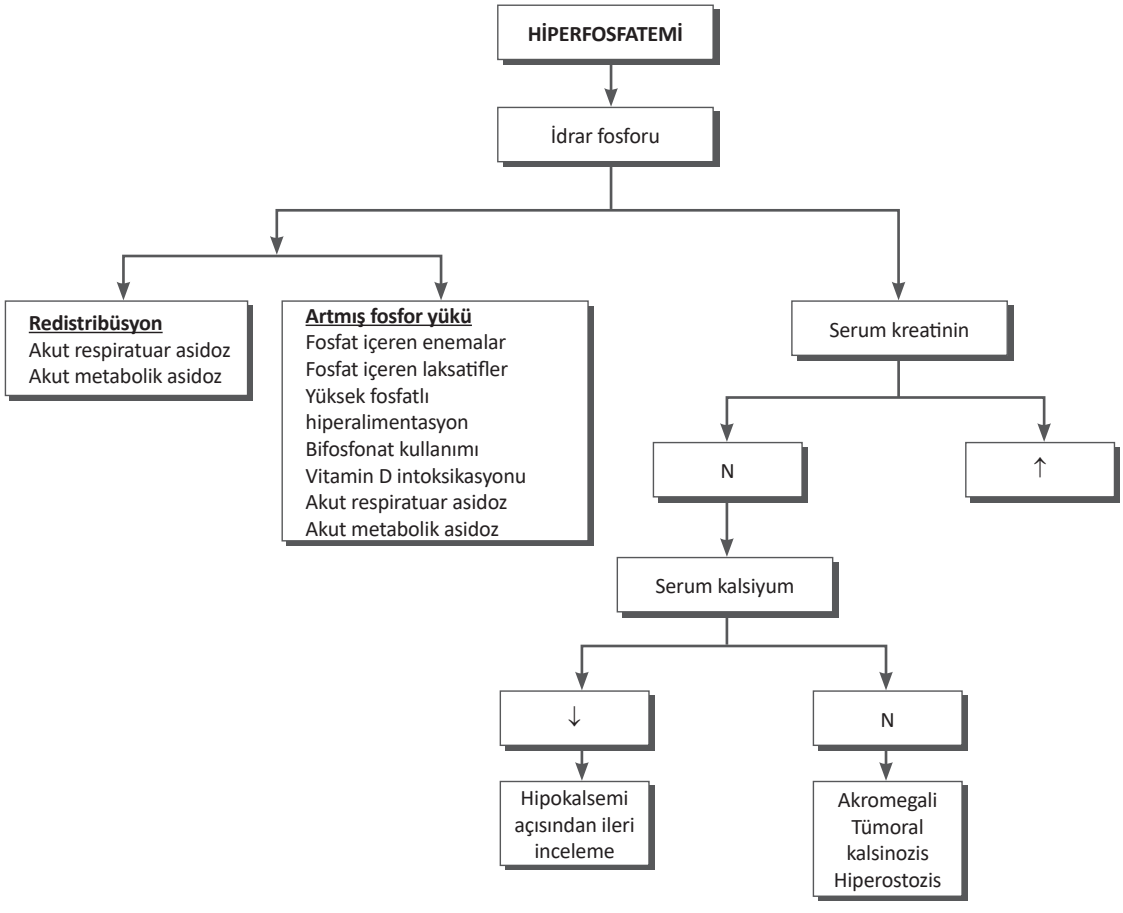
HİPERFOSFATEMİYE YAKLAŞIM [9]



ABH: akut böbrek hasarı, KBH: kronik böbrek hastalığı, ↑: artmış, ↓:azalmış, N:normal

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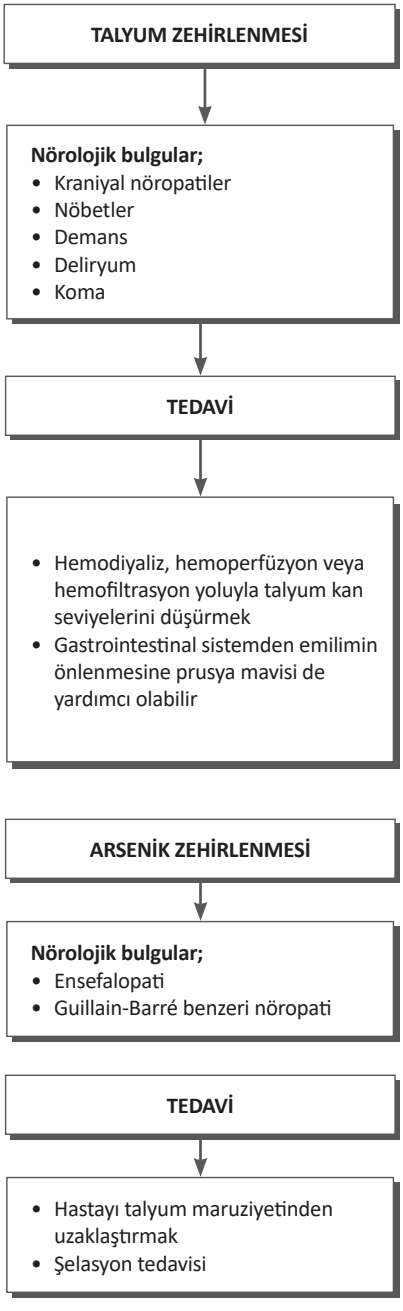

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Adjuvan Ajanlar[15,16]	İlaç Dozları	
Gabapentin	İlk 3 gün 10-15 mg/kg/gün doz üçe bölünerek kullanılmalı Etkili dozlar ise 3-4 yaş arası çocuklarda 25 mg/kg/gün 5-12 yaş arası 25-35 mg/kg/gün (doz üçe bölünerek) 12 yaşından büyüklerde oral 300 mg günde 3 kez	Türkiye'de kullanımı prospektif bilgisi olarak 6 yaşından küçüklerde yeterli deneyim yoktur olarak belirtilmektedir.
Pregabalin	4 ≥ ve <17 yaş aralığında <30 kg, 2,5 mg/kg/gün/doz günde 2 veya 3'e bölünerek kullanılmalı ≥30 kg'da ise başlangıç 3,5 mg/kg/gün/ doz günde 2 veya 3'e bölünerek kullanılmalı	Türkiye'de kullanımı prospektif bilgisi olarak 18 yaşından küçüklerde yeterli deneyim yoktur olarak belirtilmektedir.
Trisiklik Antidepresanlar (Amitriptilin)	Başlangıç dozu yatmadan önce, ilk 4 gün 0,2 mg/kg (maksimum 10 mg), sonrasında 0,4 mg/kg çıkarılır	Türkiye'de kullanımı, prospektif bilgisi olarak 12 yaşından küçüklerde kullanımı önerilmemektedir.

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PEDİATRİK NÖROLOJİ PRATİĞİ İÇİN İLAÇ REHBERİ

BÖLÜM 221

Ayten GÜLEÇ[1] Mehmet CANPOLAT[2]

İlaç Adı:

- **İlaç Formu:** Tablet (hızlı salınım, yavaş salınım, kapsül, efervesan) /şurup/ enjektabl formları ve formların mg-mg/ml cinsinden formunu içerir.

Endikasyonu

- **Pediatrik Nörolojik Endikasyon**
- **Diğer endikasyon:** Pediatrik nöroloji dışı ya da erişkin kullanım endikasyonları

Mekanizma:

Doz:

Örn: 10 kg hasta

20 mg/kg/gün/2 dozda: Toplam doz 2 doza bölerek uygulanır: 2x100mg

20mg/kg/doz x2 dozda: doz başına yapılan hesap 2 kez verilir: 2x200mg

Başlangıç: Artış: Kademeli artırılması gereken ilaçlarda ayrıntılı doz anlatımı verilmiştir.

İdame:

- **Max:** Ulaşılabilen doz başına ve günlük maksimumlar belirtilmiştir. Genellikle erişkin dozlarından daha yüksek dozlara çıkılmaz.
- **Yan etkiler ve kontrendikasyonlar:** Öncelikle kontrendike durumlar ve dikkat edilmesi gerekenler vurgulanmıştır. Ardından önem ve sıklığa göre yan etkiler ve yapılması gereken değişiklik ya da destek tedavi seçeneği bilgisine ulaşılabilenler verilmiştir.
- **Atılım yolu:** İlacın ön planda eliminasyon ve metabolize olma yolları verilmiştir. Böbrek ve karaciğer yetmezliği konusunda doz ayarlaması yönünden öneriler için ilgili kılavuzlar incelenmelidir. Ulaşılabilen doz ayarlaması gerekliliği durumu kısa özet olarak bildirilmiştir**.**
- **Yarı ömrü:** Eliminasyon için t ½ süresidir**.**

İlaç adı: Asetazolmid (AZA)

- **İlaç Formu:** Tablet 125-250mg suspansiyon: 25mg/ml yavaş salınımlı, kapsül: 500mg, Enjektabl:500mg/ml (2.05 meq/500mg Na içerir)

Endikasyonu

- **Pediatrik Nörolojik Endikasyon:** Kafa içi basınç artışında; serebrospinal sıvı üretimini azaltma yoluyla basıncı azaltma

  Parsiyel, generalize ya da absans epilepside alternatif tedavi seçeneği olarak

  Tip 2 epizodik ataksi

  Hipoksiye eşlik eden santral solunumsal bozukluk

Diğer Endikasyon:

İdrar alkalinizasyonu

İntraokuler basınç artışı

- **Mekanizma:** Karbonik anhidraz inhibisyonu, diüretik

Doz:

Başlangıç:

- **İnfant:** 8mg / kg / 3 dozda (maks. 100 mg / kg / gün)

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renç oluşturarak hem de salgılanmasını azaltarak hipokalseminin yaygın bir nedenidir.

*Hipomagnezemili hastalarda, hipokalsemi dirençli olabilir. Bu nedenle, serum magnezyum konsantrasyonu düşükse, magnezyum sülfat infüzyonu verilmelidir(bakınız Magnezyum sülfat)

Hafif semptomatik veya kronik hipokalsemi

Daha hafif derecelerde akut hipokalsemisi olan hastalar (serumla düzeltilmiş kalsiyum konsantrasyonu 7.5-8 mg/dl'nin [1.9-2.0 mmol/L] üzerinde veya serum iyonize kalsiyum konsantrasyonu 3.0 mg/dL'nin [0.8 mmol/L] üzerinde]) veya kronik hipokalsemi için oral kalsiyum(kalsiyum karbonat ya ada kalsiyum sitrat) takviyesi tercih edilir Hastala sıklıkla asemptomatiktir veya hafif semtomları olabilir

(Örn.: oral paresteziler).

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