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Kitap Adı
Granülomatöz Akciğer Hastalıkları

Yazar
Aysun ŞENGÜL
ORCID iD: 0000-0002-8084-1322

Yayın Koordinatörü
Yasin DİLMEN

Sayfa ve Kapak Tasarımı
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UYARI

Bu üründe yer alan bilgiler sadece lisanslı tıbbi çalışanlar için kaynak olarak sunulmuştur. Herhangi bir konuda profesyonel tıbbi danışmanlık veya tıbbi tanı amacıyla kullanılmamalıdır. Akademisyen Kitabevi ve alıcı arasında herhangi bir şekilde doktor-hasta, terapist-hasta ve/veya başka bir sağlık sunum hizmeti ilişkisi oluşturmaz. Bu ürün profesyonel tıbbi kararların eşleniği veya yedeği değildir. Akademisyen Kitabevi ve bağlı şirketleri, yazarları, katılımcıları, partnerleri ve sponsorları ürün bilgilerine dayalı olarak yapılan bütün uygulamalardan doğan, insanlarda ve cihazlarda yaralanma ve/veya hasarlardan sorumlu değildir.

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Akademisyen Kitabevi, üçüncü bir taraf tarafından yapılan ürüne dair değişiklikler, tekrar paketlemeler ve özelleştirmelerden sorumlu değildir.

GENEL DAĞITIM
Akademisyen Kitabevi A.Ş.

Halk Sokak 5 / A
Yenişehir / Ankara
Tel: 0312 431 16 33
siparis@akademisyen.com

www.akademisyen.com

ÖNSÖZ

Granülomatöz inflamasyon, tanı yöntemleri, tedavileri, seyirleri farklılık gösteren hem enfeksiyon hem enfeksiyon dışı onlarca hastalık nedeniyle gelişebilmektedir. Granülomatöz akciğer hastalıkları, birçok klinisyen için ayırıcı tanıda zorlanılan hastalıklardandır. Granülomatöz Akciğer Hastalıkları kitabının Göğüs Hastalıkları uzmanları başta olmak üzere tüm klinisyenlere, granülomatöz inflamasyon ile seyreden hastalıkların tanısı ve tedavi yaklaşımı konusunda katkısı olmasını umuyorum.

Aysun ŞENGÜL
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Göğüs Hastalıkları Ana Bilim Dalı

İÇİNDEKİLER

şumuna neden olabilmektedirler. Dikkatli klinik değerlendirme, laboratuvar testleri, solunum fonksiyon testi ve yüksek çözünürlüklü bilgisayarlı tomografi (HRCT) dahil olmak üzere radyolojik görüntüleme, granülomatöz akciğer hastalıklarına tanısal yaklaşımda çok önemli adımlardır. Çoğu durumda, akciğer dokusu örneklerinin patolojik incelemesi için akciğer biyopsisi gereklidir.

Bu kitapta enfeksiyöz ve nonenfeksiyöz granülomatöz akciğer hastalıklarından özellikle sık görülen hastalıkların klinik, laboratuvar, radyolojik özelliklerinin, tanı yöntemlerinin ve tedavilerinin tartışılması amaçlandı.

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maz. RD/ ÇİD-TB saptandığında, hastanın birinci seçenek ilaçlarla TB tedavisi sonlandırılarak ikinci seçenek ilaçlarla tedaviye geçilir. Moleküler ve fenotipik yeni ilaç duyarlılık sonuçlarına göre tedavi rejimi yenilenir. Dirençli olgularının tedavisinde temel prensip, basilin duyarlı olduğu, üçten az olmamak şartıyla, olabildiğince fazla sayıda ilacı yan etkiler, ilaç etkileşimleri göz önünde bulundurularak uzun süre boyunca uygulama şeklindedir (13). İlaç direnci olan olgularda, kinolonlar (levofloksasin, moksifloksasin, gatifloksasin), parenteral ilaçlar (amikasin, kapreomisin, kanamisin), diğer temel ikinci seçenek ilaçlar (etyonamid/protionamid, sikloserin/terizidon, linezolid, klofazimin) ve bedakuilin, delamanid, paraaminosalisilik asit, imipenem-silastatin., meropenem, amoksisilin-klavulanat, thioasetazon gibi ilaçlardan oluşturulan tedavi rejimleri kullanılmaktadır (13).

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iyi tanımlanmıştır. Genel olarak, cerrahi akciğer biyopsisinde pulmoner fibroz kanıtı olan hastaların prognozu, bu tür değişiklikleri olmayanlara göre daha kötüdür (96-98). Fibrotik akciğer hastalığı olan büyük bir hasta kohortunda, hem kronik fibrotik HP'li hem de idiyopatik pulmoner fibrozlu hastaların yaklaşık %60'ında hastalıkta ilerleme görülmüştür (99) Kötü prognozla ilişkili diğer özellikler arasında sigara içme, başlangıçta daha düşük VC, BAL'da lenfositoz olmaması, tetikleyici ajana sürekli maruziyet ve/veya tetikleyici ajanın tanımlanamaması yer almaktadır.

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Sigara kullanılıyorsa bırakma danışmanlığı, pnömokok ve grip aşısı, gerekliyse oksijen kullanılması ve pulmoner rehabilitasyonu da diğer tedavi önerileridir (23).

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İndüksiyon için rituksimab alan hastalar için, başka bir ajana geçmek yerine rituksimab tedavisine devam edilmesini önerilir.

Mepolizumab ile remisyona ulaşan hastalarda, bir immünosupresif ajana geçmek yerine mepolizumab tedavisine devam edilmesini önerilir. Glukokortikoid dozu, aktif EGPA semptom ve bulgularının kontrolü için gereken en düşük doza kademeli olarak azaltılır.

İdame immünosüpresif tedavi 12 ila 18 ay devam eder. Çok sayıda relapsları olan hastalarda daha uzun süreli veya süresiz idame tedavisi düşünülmelidir (29).

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normaldir. Göğüs görüntüleme çalışmaları tipik olarak, ağırlıklı olarak akciğer tabanlarında olmak üzere çok sayıda kötü tanımlanmış nodüler opasiteler gösterir. Lenfadenopati tipik olarak yoktur. PLG'nin histopatolojik tanısı tipik olarak bir polimorfik lenfoid infiltrat üçlüsü, lenfoid hücreler tarafından arterlerin ve damarların transmural infiltrasyonu ve lenfoid infiltratlar içindeki fokal nekroz alanları izlenir, iyi biçimli olmayan granülomlar şeklinde görülür. Ek özellikler, in-situ hibridizasyon çalışmalarında EBV-pozitif B hücrelerinin varlığı, T hücrelerinin poliklonalitesi ve genellikle B hücrelerinin monoklonalitesidir. Tedavi seçimi, semptomların varlığına, tetikleyici bir ilaç kullanma öyküsüne, ekstrapulmoner tutulumun derecesine ve lezyonun histopatolojik derecesinin dikkatli bir şekilde değerlendirilmesine dayanmalıdır. Akciğerlerle sınırlı düşük dereceli (derece 1 ve 2) hastalığı olan asemptomatik hastalar için, bu hastalardan bazıları spontan remisyon yaşayabileceğinden tedavi yerine gözlem önerilir. Semptomatik hastalar, daha yaygın hastalığı olan hastalar (özellikle nörolojik tutulumu olanlar) ve yüksek dereceli (3. derece) lezyonları olan hastalar, immünkemoterapi ile tedavi konusunda konsültasyon için bir hematoloji onkoloji uzmanına yönlendirilmelidir (38,39). Genel olarak, yüksek dereceli PLG için tedavi seçenekleri diffüz büyük B hücreli lenfoma (DLBCL) için olanlara benzerlik göstermektedir.

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