BÖLÜM

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SELÜLİT VE DİĞER BAKTERİYEL DERİ ENFEKSİYONLARI

Ebru SOYDAN YAPRAK[1]

GİRİŞ

Selülit ve veya apse lezyonları en sık deri ve yumuşak doku enfeksiyonları (YDE) arasındadır (1,2). Selülit (erizipel dahil), ciltte kızarıklık, ödem ve ısı artışı ile seyreder; cilt bariyerindeki açıklıklardan bakterilerin girişi ile oluşmaktadırlar (3). Cilt apseleri, dermis veya subkutan dokuda yerleşimli püy birikimidir. Her iki durumunda yanlış tanı konulması sıktır ve ayrıcı tanılar dikkatle düşünülmelidir.

EPİDEMİYOLOJİ

Selülit en sık orta yaşlı ve yaşlı erişkinlerde, erizipel ise küçük çocuklarda ve yaşlı erişkinlerde görülmektedir (4,5). Selülit insidansı yılda yaklaşık 200/100.000 vakadır ve sıklıkla sıcak aylarda artar(6). Selülit ve/veya cilt apsesi riski ile ilişkili predispozan faktörlerden bazıları şunlardır (7–15).

1. Travma nedeniyle cilt bariyerinin bozulması (dermabrazyon, penetran yara, bası yarası, venöz bacak ülseri, böcek ısırığı, intravenöz ilaç kullanımı gibi)
2. Dermatozlar ve dermatitler (egzama, radyasyon tedavisi, sedef hastalığı gibi)
3. Lenfatik drenajın bozulmasına bağlı ödem
4. Venöz yetmezliğe bağlı ödem
5. Obezite
6. İmmünosupresyon (diyabet veya HIV enfeksiyonu gibi)
7. Önceden var olan deri enfeksiyonu (tinea pedis, impetigo, suçiçeği gibi)
8. Koroner arter by-pass greft cerrahisi için safen ven grefti alınması

Cerrahi prosedürleri (safenöz venektomi veya lenf nodu diseksiyonu gibi) takiben veya konjenital olarak lenfatik ödem oluşabilmektedir, 2012-2017 yıllarında ABD'de yapılan bir retrospektif bir araştırmada selülit nedeniyle 165.000'den fazla hastane başvurularının analizinde, vakaların çoğu (%92) lenfödem ile ilişkilendirilmiştir(16).

Pürülan cilt ve YDE'nın için ek bir risk faktörü, metisiline dirençli S. aureus enfeksiyonu (MRSA) veya taşıyıcısı olan diğer kişilerle yakın temastır.

ETİYOLOJİ

Selülitin en sık nedenleri beta-hemolitik streptokoklardır (A, B, C, G ve F grupları), en sık A grubu S. pyogenes; S. aureus (MRSA dahil) daha az

[1] Uzm. Dr., Özel Gözde Hastanesi Sıtmapınarı, Deri ve Zührevi Hastalıkları Kliniği, dr.ebru_91@hotmail.com

dikloksasilin tedavileri metsiline duyarlı S.aureus infeksiyonları için uygundur. Eritromisin ve klindamisin impetigo da kullanılabilmekle birlikte, bazı S. aureus ve GAS suşları bu tedavilere dirençli olabilir. Yaygın impetigo veya ektimada yalnızca beta-hemolitik streptokoklar saptanırsa, tercih edilen tedavi oral penisilindir(83). Streptokoksik infeksiyonlarda, yapılan bir çalışmada oral trimetoprim-sülfametoksazolün enjektabıl penisilin tedavisine etkili bir alternatif olabileceği bildirilmiştir(100).

Metisiline dirençli olmayan S.aureus'a bağlı impetigoda birinci basamak tedavi olmasa da, kısa bir oral trimetoprim-sülfametoksazol kürü, stafilokoksik ve streptokoksik impetigo için ucuz bir oral alternatif olabilir(48).

Şüpheli veya doğrulanmış MRSA infeksiyonu olan hastalar, izole etkenin duyarlı olması koşuluyla trimetoprim-sülfametoksazol, klindamisin veya doksisiklin ile tedavi edilebilir (83).

Tetrasiklinler, sekiz yaşından küçük çocuklarda kalıcı diş renklenmesine neden olabilse de doksisiklin diğer tetrasiklinlere göre kalsiyuma daha az bağlanır(101). Bu nedenle, bu yaş grubunda kısa süreli (<21 gün) doksisiklin verilebilir(102).

Hastalar doksisiklin ile ilişkili fotosensitivite konusunda uyarılmalı ve aşırı güneşe maruziyetten kaçınmalıdırlar(103).

Kabuklu lezyonlar nazikçe ılık-sabunlu su yıkanabilir. El yıkama, çocuklar arasında yayılımı azaltmak için çok önemlidir, drene olan akıntılı lezyonlar mutlaka kapalı pansuman edilmelidir.

İmpetigo infeksiyonunun hem bulaştırıcı olması, hem de okul çağı çocuklarında görülmesi nedeniyle okula dönüş süresi ve eğitimin devamı da çok önemli olduğundan hastalar, etkili bir antimikrobiyal tedaviden 24. saat sonra okula dönebilirler.

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