BÖLÜM 24

Pulmoner Hipertansiyon

Mustafa YETİŞEN [1]

GİRİŞ

Pulmoner arterler normal koşullarda düşük basınçlı yüksek debinin hâkim olduğu, kompliyansın çok fazla olduğu bir yapıya sahiptir. Etiyopatogenez net olarak aydınlatılamamakla birlikte pulmoner arterlerin intimal tabakasındaki endotel hücrelerinde proliferasyon, düz kas hücrelerinde hipertrofi ve artmış damar içi trombozis suçlanmaktadır. Sonuç olarak antiproliferatif ajanlar olan prostasiklin ve nitrik oksit (NO) lehine olan denge tromboksan A2 ve endotelin-1 gibi vazokonstriksiyona ve proliferasyona neden olan ajanlar lehine bozulmaktadır. Bu durum vazodilatasyonun azalmasına neden olmaktadır.

TANI VE SINIFLANDIRMA

Tanım

İlk olarak 1973 yılında birinci pulmoner hipertansiyon sempozyumunda istirahat halinde sağ kalp kateterizasyonu sonucu ortalama pulmoner arter basıncı (oPAB) değerinin 25 mmHg'nin (SKK) üzerinde olması pulmoner hipertansiyon (PHT) olarak tanımlandı (1). Ancak normal sağlıklı insanlarda istirahat halindeyken oPAB değerinin 14 ± 3 mmHg olduğu ve oPAB değerinin 20 mmHg'nin üzerine çıkmasının mortalite üzerine önemli ölçüde olumsuz katkısının olduğu fark edildi. Son olarak 2022 ESC/ERS PHT Kılavuzunda 20 mmHg değeri referans kabul edilmiş, istirahat halinde SKK ile ölçülen oPAB değerinin 20 mmHg'nin üzerine çıkması PHT olarak tanımlanmıştır (2).

[1] Uzm. Dr., Erzurum Oltu Devlet Hastanesi, m.yetisen@hotmail.com, ORCID iD: 0000-0002-9027-8908

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