

Bölüm 22

VENA KAVA SUPERİOR SENDROMU

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GİRİŞ

İnce duvarlı Superior Vena Kavanın (SVK) sıkışmasından kaynaklanan klinik bir durum olan Superior Vena Kava Sendromu (SVKS) ilk olarak 1757'de İskoçya'lı bir hekim olan William Hunter tarafından sifilitik aortitli bir hastada tanımlandı.(1) Amerika Birleşik Devletlerinde yılda 15.000'den fazla hastada SVKS görülmektedir.(2) Antibiyotiklerin ortaya çıkmasından önce, SVC sendromunun en yaygın iki nedeni SVC kompresyonuna neden olan sifilitik torasik anevrizma ve mediastinal adenopatiye yol açan tüberkülozdu.(1-4) Bununla birlikte, tıbbi gelişmelere bağlı olarak SVC sendromunun etiyojisi de değişti. Mediastinal lenfadenopati ile birlikte olan metastatik akciğer kanseri ve primer mediastinal maligniteler en sık nedendir (vakaların% 60'ı).(2,5) Malign olmayan nedenler ise santral venöz hatların ve kalp pillerinin daha sık kullanılması nedeniyle artmaktadır. Diğer nedenler arasında mediastinal fibrozis; histoplazmoz gibi granülo-matöz mantar hastalığı; mediastene önceki radyasyon; retrosternal guatr; ve aort diseksiyonudur.

Anatomi ve Fiziopatoloji

Superior Vena Kava, baş, boyun, üst ekstremiteler ve toraks üstünden kalbe dönen kanı taşıyan ince duvarlı, düşük basınçlı bir damardır. Sağ mediastende bulunduğu yerde, trakea, sağ bronş, aort, pulmoner arter veya perihilar ve paratrakeal lenf nodları gibi ilişkili yapılardaki anormallikler ile kolayca sıkıştırılabilir.

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