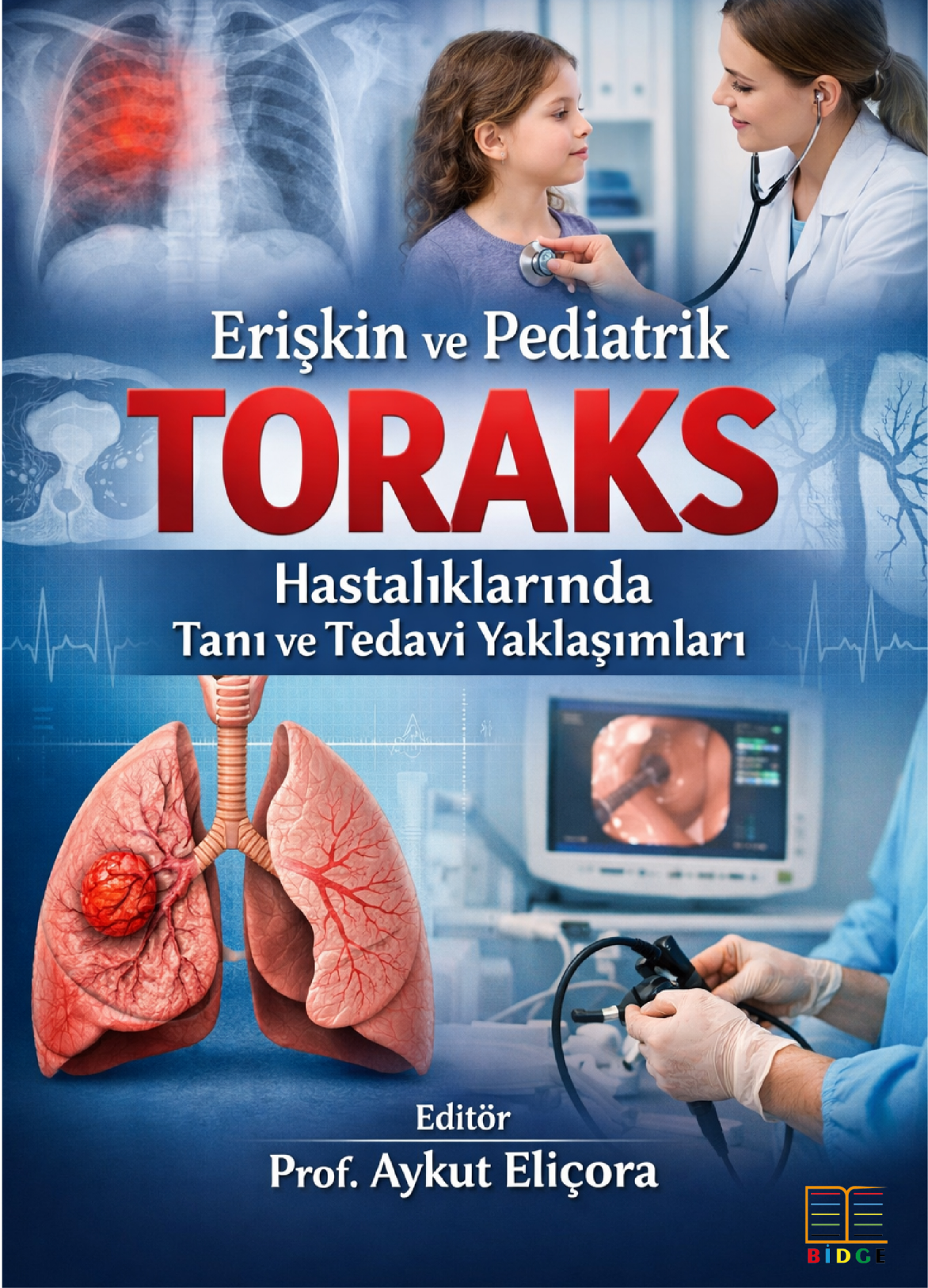
Erişkin ve Pediatrik
TORAKS
Hastalıklarında
Tanı ve Tedavi Yaklaşımları
Editör
Prof. Aykut Eliçora
BİDGE

BİDGE Yayınları

Erişkin ve Pediatrik Toraks Hastalıklarında Tanı ve Tedavi Yaklaşımları

Editör: PROF. DR. AYKUT ELİÇORA

ISBN: 978-625-8673-19-7

1. Baskı
Sayfa Düzeni: Gözde YÜCEL
Yayınlama Tarihi: 2025-12-25
BİDGE Yayınları

Sertifika No: 71374
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İÇİNDEKİLER

BÖLÜM 1

ÇOCUK AKCİĞER APSESİNDE KLİNİK ÖZELLİKLER, TANI VE TEDAVİ STRATEJİLERİ

HIDIR ESME[1]

Giriş

Çocukluk çağı akciğer apsesi nispeten nadir görülen bir durumdur ve bu nedenle bu konudaki veriler oldukça azdır [1]. Akciğer apsesi, toplum kaynaklı pnömoninin olası bir lokal komplikasyonudur. Ciddi hastalığa ve uzun süreli hastanede kalışa yol açabilse de, hastaların çoğu genellikle tam olarak iyileşir [2]. Akciğer apsesi, akciğer parankiminin geniş çaplı tahribatına neden olan bir akciğer enfeksiyonundan kaynaklanır ve doku iltihabı ve nekrozu nedeniyle pürülan materyal içeren sınırlı, kalın duvarlı bir boşluğun gelişmesine yol açar [3].

Sınıflandırma

Akciğer apseleri, etiyoloji, süre veya ekstrapulmoner bölgelerden yayılma gibi çeşitli faktörlere göre sınıflandırılabilir. Akut apseler 4-6 haftadan az sürerken, kronik apseler daha uzun süre devam eder [4].

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Etiyoloji açısından, akciğer apseleri predispozan faktörlerin varlığına bağlı olarak primer veya sekonder olarak sınıflandırılabilir. Primer apse önceden sağlıklı olan çocuklarda görülürken, sekonder apse altta yatan lokal veya sistemik nedenlere bağlı olarak ortaya çıkabilir. Sekonder akciğer apsesi, konjenital pulmoner hava yolu malformasyonu, kistik fibrozis veya primer siliyer diskinezi gibi kalıtsal akciğer hastalıkları olan hastalarda ve immün yetmezlik gibi immün sistemi zayıflamış hastalarda gelişebilir [1,4].

Bronşial yabancı cisim veya pulmoner metastaz gibi nedenlerlede ortaya çıkabilir [5,6]. Ek olarak, akciğer apseleri, septik tromboembolilerde olduğu gibi hematojen yayılım yoluyla ekstrapulmoner bölgelerden akciğere gelebir. Bazende abdominal veya orofaringeal bölgelerden doğrudan yayılma yoluyla da ortaya çıkabilir [4,7].

Nörogelişimsel gecikmesi olan çocuklarda, bozulmuş klirens ve zayıf koordinasyonlu yutma nedeniyle pulmoner aspirasyon akciğer apselerinin oluşumunda önemli bir rol oynar. Bazı durumlar belirli mikroorganizmalarla daha sık ilişkili olduğundan, bu predispozan faktörleri tanımlamak önemlidir [1,7].

Etiyoloji ve Patogenez

Anaerobik bakteriler geçmişte akciğer apselerinin primer patojenleri olarak tanımlanmıştır; Peptostreptococcus türleri, Bacteroides türleri ve Fusobacterium türleri en yaygın olanlarıdır [2]. Son zamanlarda yapılan çalışmalar, bakteriyolojik özelliklerde bir değişiklik olduğunu göstermektedir. Staphylococcus aureus ve Streptococcus türleri, özellikle primer apselerde en yaygın olanlardır ve bunları Klebsiella pneumoniae gibi bazı Gram-negatif basiller izlemektedir [8–10]. Sekonder apseler daha sıklıkla anaeroblar ve Gram-pozitif koklar tarafından neden olmaktadır [2,7]. Parazitler, mantarlar ve Mycobacterium türleri gibi bakteriyel

olmayan ve atipik bakteriyel patojenler, genellikle bağışıklık sistemi zayıflamış kişilerde akciğer apselerine yol açabilir [11].

Akciğer apseleri, enfeksiyöz materyalin yerçekimi akışı ve sağ bronşun dikey yönelimi nedeniyle genellikle sağ akciğerde, özellikle sağ üst lobun arka segmentinde veya sağ alt lobun üst bölgesinde gelişir [4].

Distal bronşlarda yerleşen enfeksiyöz materyal, akciğer dokusunda lokal inflamasyonu başlatır. Saatler ila birkaç gün içinde, eksüda, kan hücreleri ve nekrotik dokunun birikmesi nedeniyle bu inflamasyon şiddetlenir ve sıvılaşan nekroza ilerleyerek apse oluşumuyla sonuçlanabilir. Nekrotik materyal bronşlardan dışarı atıldıkça, hava-sıvı seviyesi içeren nekrotik bir boşluk gelişir. Enfeksiyon, plevral boşluğa yayılabilir ve apse kavitesi plevral aralığa açılmadan ampiyeme neden olabilir [4].

Klinik Özellikler

Çocuklarda pulmoner apsenin klinik görünümü, çocuğun yaşına, altta yatan etiyolojiye ve akciğer tutulumu derecesine bağlı olarak büyük ölçüde değişebilir. Hastalar tipik olarak, yüksek ateş ve öksürük ile başvururlar; öksürük balgamlı veya pürülan olabilir ve sıklıkla iştahsızlık ve kilo kaybı ile birliktedir. Nadiren göğüs ağrısı, nefes darlığı ve hemoptizi görülür. Yenidoğanlarda, bebeklerde, bağışıklık sistemi zayıflamış hastalarda veya uzun süreli kortizon tedavisi gören hastalarda ateş görülmeyebilir. Akciğer apsesi olan çocuklar, özellikle yenidoğan ve bebekler, apne, halsizlik, kusma, ishal ve hematemez gibi nonspesifik semptomlar gösterebilir [4].

Ağır vakalarda, taşipne, burun kanatlarının solunuma katılması, interkostal veya subkostal çekilmeler ve siyanoz şeklinde kendini gösteren solunum sıkıntısı gelişebilir. Bu durum, akciğer fonksiyonlarında önemli bir bozulmaya işaret eder ve acil tıbbi müdahale gerektirir [12,13].

Çocuklarda akciğer apselerinin klinik görünümü, pnömoni ve bronşiyolit gibi diğer solunum rahatsızlıklarına benzeyebilir. Bu nedenle, ayrıntılı öykü, fizik muayene ve görüntüleme ve mikrobiyolojik çalışmalar gibi uygun tanı testlerini içeren kapsamlı bir değerlendirme, doğru tanı ve etkili tedavi için gereklidir [5,6,14].

Tanı Değerlendirmesi

Görüntüleme, pulmoner apselerin tanısında temel taştır; ancak, tam kan sayımı, inflamatuar belirteçler ve bakteriyel kültürler gibi ek testler, kapsamlı bir tanı değerlendirmesi için gereklidir. Laboratuvar testlerinde genellikle nötrofili ile birlikte lökositoz, yüksek akut faz reaktantları (C-reaktif protein, fibrinojen ve prokalsitonin gibi), düşük hemoglobin seviyesi, hipoalbüminemi ve yüksek D-dimer seviyeleri görülür. Kan kültürleri etiyolojiyi belirleyebilir ve ateşsiz yenidoğanlarda, küçük bebeklerde ve bağışıklık sistemi yetersizliği olan çocuklarda bile yapılmalıdır, ancak pozitif sonuçların verimi düşüktür [15].

Etken organizmayı belirlemek için balgam kültürü ve duyarlılık testi kullanılabilir. Ancak, üst solunum yolu florası kaynaklı bakterilerle kontaminasyon riski yüksek olduğundan, bu testler nadiren güvenilirdir. Etken organizmayı tanımlamak ve uygun antimikrobiyal tedaviyi yönlendirmek için, özellikle tedavi başarısızlığı veya tekrarlayan apseler durumunda, bronkoalveolar lavaj veya akciğer biyopsisi gerekli olabilir [16].

Standart mikrobiyolojik yöntemler, pulmoner apse vakalarının neredeyse yarısında etiyolojik nedeni tespit edememektedir [9,17]. Ancak, akciğer apsesinde tanı görüntüleme çalışmalarına dayalıdır. Göğüs radyografisi genellikle tercih edilen ilk görüntüleme yöntemidir ve sıklıkla hava-sıvı seviyesi içeren sınırlı opasite gibi karakteristik bulguları ortaya çıkarır. Konsolidasyon ve buna bağlı plevral efüzyon tespit edilebilir.

Radyografiler, hastalar dik pozisyonda iken çekilmelidir, çünkü sırtüstü pozisyonda çekilen filmlerde hava-sıvı seviyesi görünmeyebilir [18,19]. Göğüs ultrasonu, akciğer apselerini plevral efüzyondan veya parankimal anormalliklerden ayırt etmede etkilidir ve takip için yararlı olabilir [20].

Bilgisayarlı tomografi (BT), göğüs radyografisinden daha ayrıntılı anatomik bilgiler sağlar, komplikasyonları tanımlamak için çok önemlidir ve atipik vakalarda daha fazla ayrıntı sağlayabilir. BT her biri farklı bir tedavi yaklaşımı gerektiren parankimal lezyonlar ile plevral efüzyonları birbirinden ayırt edebilir [20]. Ayrıca, apsenin altta yatan nedeninin, enfekte kist, konjenital anomali, hava yolu tıkanıklığı veya komşu yapı enfeksiyonu olup olmadığını belirlemeye yardımcı olur.

Toraks BT rezeksiyon kararının alınmasında yol gösterici olarak cerrahi planlamada önemli bir rol oynar [21]. Dahası, toraks BT ve ultrason, tanı ve drenaj için göğüs tüpü yerleştirme ve iğne aspirasyonu gibi girişimsel prosedürleri planlamada da yol gösterici değerli araçlardır [19].

Tedavi

Akciğer apseleri, apsenin boyutu, nedeni ve ilk tedaviye verdiği yanıt gibi faktörlere bağlı olarak, antibiyotik ve destekleyici tedavi ile konservatif olarak tedavi edilebir veya cerrahi olarak rezeke edilebilir [10].

Konservatif Tedavi

Uzun süreli antibiyotik tedavisi, pediatrik vakaların %90'ında akciğer apsesinin tedavisinde etkilidir ve primer akciğer apsesinin tedavi sonucu genellikle olumludur. Morbidite ve mortalite genellikle sekonder apselere yatkınlık yaratan durumlardan kaynaklanır [7].

İntravenöz antibiyotik tedavisi genellikle 2-3 hafta sürer, ancak hastalığın ilerlemesine bağlı olarak uzatılabilir. Genellikle parenteral tedaviyi oral tedavi izler ve toplam tedavi süresi yaklaşık 3-6 haftadır [22]. Antibiyotik tedavisinin süresi, yerel direnç paternlerine ve deneyime göre değişir. Antimikrobiyal ajanların seçimi, apse türü ve çocuğun herhangi bir altta yatan hastalığı olup olmadığı, organizmayı izole etme ve tanımlama yeteneği, maliyetler ve yerel uygulamalar gibi faktörlere bağlıdır [2,7].

Akciğer apsesi için mevcut antibiyotik rejimleri, Gram-pozitif, Gram-negatif ve anaerobik organizmaları hedef alan geniş spektrumlu antibiyotik tedavisine odaklanmaktadır [1]. Çoğu vaka, genellikle iki antibiyotik ajanla başlanan tedavi ile etkili bir şekilde yönetilebilir. Genellikle, klinik iyileşme yaklaşık bir hafta içinde görülür, ateş genellikle 3-4 günlük antibiyotik tedavisinden sonra düşer ve genel durum 4-7 gün sonra iyileşir. Görüntüleme açısından, radyografik görüntünün tamamen düzelmesi 2-3 ay sonra görülebilir. Hasta 7-10 gün içinde antibiyotik tedavisine yanıt vermezse, dirençli organizmalar veya hava yolu tıkanıklığı açısından yeniden değerlendirme yapılması gerekir [4,23].

Antibiyotikler, özellikle primer apse tedavisinde, üst solunum yolunda tipik olarak bulunan Staphylococcus aureus, Streptococcus pneumoniae ve Gram-negatif basiller gibi yaygın organizmaları hedef almalıdır. Aspirasyon nedeniyle sekonder apse oluşumu riski olan durumlarda, üst solunum yolunda yaygın olarak bulunan anaeroblara karşı etkili antibiyotikleri dahil etmek çok önemlidir. Bağışıklık sistemi zayıflamış hastalarda antibiyotik kapsamı daha geniş olmalı ve mantar patojenleri de dikkate alınmalıdır [1,24].

Geleneksel olarak ilk tercih edilen antibiyotik olan penisilin, streptokok türlerine karşı etkilidir, ancak günümüzde beta-laktamaz üreten organizmalar nedeniyle direnç sorunu ortaya çıkabilmektedir. Hastaların çoğu için kabul edilebilir tedavi

seçenekleri genellikle beta-laktamaz inhibitörü ile kombine karbapenemdir. Gram-pozitif ve Gram-negatif mikropları kapsayan üçüncü nesil sefalosporinler, Staphylococcus aureus ve anaerobları kapsama avantajı sunan klindamisin gibi bir antibiyotiğe ek olarak tercih edilir [10]. Metisiline dirençli S. aureus oranlarının yüksek olduğu bölgelerde, kültür sonuçları elde edilene kadar vankomisin önerilir [1,2].

İlk tedavi planı, kültür sonuçları ve hastanın tedaviye verdiği yanıt dikkate alınarak gerektiğinde değiştirilebilir. Anaerobik organizmaların kültürde üremesinin zor olduğu ve bu nedenle duyarlılık testlerinin nadiren yapıldığı unutulmamalıdır [2].

Cerrahi Tedavi

Bir haftalık uygun antimikrobiyal tedavi sonrasında belirgin bir klinik iyileşme görülmezse, apse veya yeni apseler gibi olası komplikasyonları belirlemek ve cerrahi müdahale öngörülmesi durumunda, tercihen toraks BT taraması olmak üzere ek görüntüleme testleri yapılmalıdır [25].

Yaklaşık %10 oranında hasta antimikrobiyal tedaviye yanıt vermez ve drenaj prosedürü veya cerrahi müdahale gerekir [7]. Cerrahi seçenekler arasında perkütan aspirasyon ve göğüs tüpü yerleştirme, bronkoskopik drenaj, video yardımlı torakoskopik cerrahi (VATS) veya açık cerrahi yer alır [21]. VATS, aspirasyon ve drenaja kıyasla daha yüksek başarı oranları, açık cerrahiye göre daha düşük komplikasyon oranları ve daha hızlı iyileşme oranları nedeniyle tedavi seçeneği olarak giderek daha fazla tercih edilmektedir [22,26]. İğne aspirasyonu veya perkütan kateter drenajı gibi prosedürler hem tanıya yardımcı olur hem de tedavi yaklaşımı olarak işlev görür [27].

Perkütan olarak gerçekleştirilen transtorasik kateter drenajı, görüntüleme ile yönlendirilebilir [28,29]. Kaynaklara bağlı olarak, bu işlem bilgisayarlı tomografi, ultrason veya floroskopi ile

gerçekleştirilebilir [21,30]. Ancak, bu işlemlerde plevral boşluk enfeksiyonu riski ve kanama veya pnömotoraks gibi riskler vardır [31].

Hastaların antibiyotik tedavisine yanıt vermemesi, önemli kanama veya bronkopleural fistül gibi komplikasyonlar gelişmesi durumunda cerrahi müdahale gerekebilir [22]. Buna katkıda bulunan faktörler arasında apse büyüklüğü ve antibiyotik dirençli organizmalar sayılabilir. Bronkopleural fistül veya yaygın pulmoner nekroz şüphesi varsa, açık cerrahi muhtemelen gereklidir. Cerrahi rezeksiyon, lezyonun yaygınlığına göre belirlenir ve en sık uygulanan prosedür lobektomidir [21].

Komplikasyon ve Prognoz

Çocuklarda akciğer apselerinin komplikasyonları, enfeksiyonun yayılması, kalıcı iltihaplanma ve akciğer dokusunda buna bağlı yapısal hasar nedeniyle ortaya çıkabilir [15]. Akciğer apseleri, plevral boşlukta enfekte sıvının birikmesiyle karakterize olan ampiyem oluşumuna yol açabilir. Bu durum solunum sıkıntısının artmasına, plevral göğüs ağrısına ve sistemik sepsis belirtilerine yol açabilir [31].

Kronik veya büyük apseler, komşu bronşlara ve plevral boşluğa yayılabilir ve bronkoplevral fistül oluşumuna yol açabilir. Hava yolu ile plevral boşluk arasındaki bu iletişim, kalıcı hava kaçağı, tekrarlayan pnömotoraks ve ikincil enfeksiyon riskinde artışa neden olabilir [15]. Akciğer apseleriyle ilişkili inflamatuar süreçler, plevral efüzyon gelişmesine yol açarak akciğer fonksiyonunu daha da bozabilir ve solunum yetmezliği ve solunum fonksiyon bozukluğu riskini artırabilir [32].

Şiddetli veya tedavi edilmeyen akciğer apseleri, bakteriyemiye ve enfeksiyonun sistemik yayılmasına neden olarak sepsis, septik şok ve çoklu organ disfonksiyon sendromuna yol

açabilir. Bu, acil tıbbi müdahale gerektiren, yaşamı tehdit eden bir komplikasyondur [9].

Akciğer parankiminde uzun süreli iltihaplanma ve enfeksiyon, doku nekrozu ve kavitasyona yol açarak akciğer apsesinin büyüme riskini, semptomların devam etmesini ve akciğer fonksiyon bozukluğunu artırabilir [33].

Yutma reflekslerinde bozukluk veya nörolojik bozukluklar gibi akciğer apsesi için predispozan faktörleri olan çocuklar, aspirasyon pnömonisi riskinde artışa sahiptir ve bu durum, zamanında tedavi edilmezse akciğer apsesi gelişimine yol açabilir [1,34].

Tekrarlayan veya kronik akciğer apseleri, bronşektazi, fibrozis ve akciğer fonksiyon bozukluğu gibi kronik akciğer hastalıklarına yol açabilir ve çocukları tekrarlayan solunum yolu enfeksiyonlarına ve uzun süreli solunum komplikasyonlarına yatkın hale getirebilir [15].

Çocuklarda akciğer apselerinin nadir ancak ciddi komplikasyonları arasında, beyin, kemik veya eklem gibi uzak organlarda metastatik enfeksiyonlara yol açan septik embolilerin sistemik embolizasyonu yer alabilir [35].

Çocuklarda akciğer apselerinin uzun dönem sonuçları genellikle olumludur ve çoğu çocuk pulmoner sekeller olmadan sorunsuz bir şekilde iyileşir, ancak altta yatan durumların varlığı prognozu etkiler [1]. Komplikasyonları önlemek ve etkilenen çocuklarda sonuçları iyileştirmek için erken tanı ve uygun tedavinin derhal başlatılması çok önemlidir [36]. Sonuçları optimize etmek ve komplikasyon riskini azaltmak için genellikle pediatristler, pulmonologlar, radyologlar, patologlar, enfeksiyon hastalıkları uzmanları ve göğüs cerrahlarının dahil olduğu multidisipliner bir yönetim gereklidir [32].

Kaynaklar

1. Patradoon-Ho, P.; Fitzgerald, D.A. Lung abscess in children. Paediatr. Respir. Rev. 2007, 8, 77–84.

2. De Benedictis, F.M.; Kerem, E.; Chang, A.B.; Colin, A.A.; Zar, H.J.; Bush, A. Complicated pneumonia in children. Lancet 2020, 396, 786–798.

3. Yousef, L.; Yousef, A.; Al-Shamrani, A. Lung Abscess Case Series and Review of the Literature. Children 2022, 9, 1047.

4. Kuhajda, I.; Zarogoulidis, K.; Tsirgogianni, K.; Tsavlis, D.; Kioumis, I.; Kosmidis, C.; Tsakiridis, K.; Mpakas, A.; Zarogoulidis, P.; Zissimopoulos, A.; et al. Lung abscess-etiology, diagnostic and treatment options. Ann. Transl. Med. 2015, 3, 183.

5. Roik, D.; Mosior, T.; Sopyło, B.; Małdyk, J.; Brzewski, M. Abscess mimicking lung metastasis in a 10-year-old boy—Case report. Pol. J. Radiol. 2010, 75, 55–57.

6. Vlahova, A.; Antonova, Z.; Rangelov, E.; Kartulev, N.; Oparanova, V.; Gabrovska, N.; Spasova, A.; Velizarova, S.; Shivachev, H. Surgical Treatment of Lung Abscess Due to an Awn Aspiration in a 9-Year-Old Child: A Case Report. Children 2023, 10, 910.

7. Puligandla, P.S.; Laberge, J.-M. Respiratory infections: Pneumonia, lung abscess, and empyema. Semin. Pediatr. Surg. 2008, 17, 42–52.

8. Nicolini, A.; Cilloniz, C.; Senarega, R.; Ferraioli, G.; Barlascini, C. Lung Abscess Due to Streptococcus pneumoniae: A Case Series and Brief Review of the Literature. Adv. Respir. Med. 2014, 82, 276–285.

9. Takayanagi, N.; Kagiyama, N.; Ishiguro, T.; Tokunaga, D.; Sugita, Y. Etiology and Outcome of Community-Acquired Lung Abscess. Respiration 2010, 80, 98–105.

10. Madhani, K.; McGrath, E.; Guglani, L. A 10-year retrospective review of pediatric lung abscesses from a single center. Ann. Thorac. Med. 2016, 11, 191–196.

11. Redding, G.J.; Carter, E.R. Chronic Suppurative Lung Disease in Children: Definition and Spectrum of Disease. Front. Pediatr. 2017, 5, 30.

12. Breuer, O.; Picard, E.; Benabu, N.; Erlichman, I.; Reiter, J.; Tsabari, R.; Shoseyov, D.; Kerem, E.; Cohen-Cymberknoh, M. Predictors of Prolonged Hospitalizations in Pediatric Complicated Pneumonia. Chest 2018, 153, 172–180.

13. Yen, C.-C.; Tang, R.-B.; Chen, S.-J.; Chin, T. Pediatric lung abscess: A retrospective review of 23 cases. J. Microbiol. Immunol. Infect. 2004, 37, 45–49.

14. Ionescu, M.D.; Popescu, N.A.; Stănescu, D.; Enculescu, A.; Bălgrădean, M.; Căpitănescu, G.M.; Bumbăcea, D. The Challenging Diagnosis of Interstitial Lung Disease in Children—One Case Report and Literature Review. J. Clin. Med. 2022, 11, 6736.

15. Eslamy, H.K.; Newman, B. Pneumonia in Normal and Immunocompromised Children: An Overview and Update. Radiol. Clin. N. Am. 2011, 49, 895–920.

16. Bennett, J.E.; Dolin, R.; Blaser, M.J. Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases: 2-Volume Set; Elsevier Health Sciences: Amsterdam, The Netherlands, 2019; ISBN 978-0-323-55027-7.

17. Mukae, H.; Noguchi, S.; Naito, K.; Kawanami, T.; Yamasaki, K.; Fukuda, K.; Akata, K.; Fukuda, Y.; Kido, T.;

Ishimoto, H.; et al. The Importance of Obligate Anaerobes and the Streptococcus anginosus Group in Pulmonary Abscess: A Clone Library Analysis Using Bronchoalveolar Lavage Fluid. Respiration 2016, 92, 80–89.

18. Klein, J.S.; Schultz, S.; Heffner, J.E. Interventional radiology of the chest: Image-guided percutaneous drainage of pleural effusions, lung abscess, and pneumothorax. Am. J. Roentgenol. 1995, 164, 581–588.

19. Quick, R.D.; Auth, M.J.; Fernandez, M.; Meyer, T.; Merkel, K.G.; Thoreson, L.M.; Hauger, S.B. Decreasing Exposure to Radiation, Surgical Risk, and Costs for Pediatric Complicated Pneumonia: A Guideline Evaluation. Hosp. Pediatr. 2017, 7, 287–293.

20. Odev, K.; Guler, İ.; Altinok, T.; Pekcan, S.; Batur, A.; Ozbiner, H. Cystic and Cavitary Lung Lesions in Children: Radiologic Findings with Pathologic Correlation. J. Clin. Imaging Sci. 2013, 3, 60.

21. Loukeri, A.A.; Kampolis, C.F.; Tomos, P.; Papapetrou, D.; Pantazopoulos, I.; Tzagkaraki, A.; Veldekis, D.; Lolis, N. Diagnosis, treatment and prognosis of lung abscess. Pneumon 2015, 28, 54.

22. Nagasawa, K.K.; Johnson, S.M. Thoracoscopic treatment of pediatric lung abscesses. J. Pediatr. Surg. 2010, 45, 574–578.

23. Shah, S.S.; Srivastava, R.; Wu, S.; Colvin, J.D.; Williams, D.J.; Rangel, S.J.; Samady, W.; Rao, S.; Miller, C.; Cross, C.; et al. Intravenous Versus Oral Antibiotics for Postdischarge Treatment of Complicated Pneumonia. Pediatrics 2016, 138, e20161692.

24. De Benedictis, F.M.; Carloni, I. Management of necrotizing pneumonia in children: Time for a patient-oriented approach. Pediatr. Pulmonol. 2019, 54, 1351–1353.

25. Reimel, B.A.; Krishnadasen, B.; Cuschieri, J.; Klein, M.B.; Gross, J.; Karmy-Jones, R. Surgical Management of Acute Necrotizing Lung Infections. Can. Respir. J. 2006, 13, 369–373.

26. Sueyoshi, R.; Koga, H.; Suzuki, K.; Miyano, G.; Okawada, M.; Doi, T.; Lane, G.J.; Yamataka, A. Surgical intervention for congenital pulmonary airway malformation (CPAM) patients with preoperative pneumonia and abscess formation: “open versus thoracoscopic lobectomy”. Pediatr. Surg. Int. 2016, 32, 347–351.

27. Al-Salem, A.H.; Ali, E.A. Computed tomography-guided percutaneous needle aspiration of lung abscesses in neonates and children. Pediatr. Surg. Int. 1997, 12, 417–419.

28. Saito, T.; Matano, M.; Kodachi, T.; Fukui, K.; Monden, Y.; Fuchimoto, Y. Pulmonary abscess in an infant treated with ultrasound-guided drainage. J. Pediatr. Surg. Case Rep. 2020, 60, 101549.

29. Chiang, P.-C.; Lin, C.-Y.; Hsu, Y.-C.; Huang, L.-T.; Chung, T.-J.; Liu, Y.-S.; Chang, C.-C. Early drainage reduces the length of hospital stay in patients with lung abscess. Front. Med. 2023, 10, 1206419.

30. Hogan, M.J.; Coley, B.D. Interventional radiology treatment of empyema and lung abscesses. Paediatr. Respir. Rev. 2008, 9, 77–84.

31. Wali, S.O.; Samman, Y.S.; Abdelaziz, M.; Shugaeri, A. Percutaneous Drainage of Pyogenic Lung Abscess. Scand. J. Infect. Dis. 2002, 34, 673–679.

32. Macedo, M.; Meyer, K.F.; Oliveira, T.C.M. Pneumonia necrosante em crianças submetidas à toracoscopia por empiema pleural: Incidência, tratamento e evolução clínica. J. Bras. Pneumol. 2010, 36, 301–305.

33. Stockmann, C.; Ampofo, K.; Pavia, A.T.; Byington, C.L.; Sheng, X.; Greene, T.H.; Korgenski, E.K.; Hersh, A.L. Comparative Effectiveness of Oral Versus Outpatient Parenteral Antibiotic Therapy for Empyema. Hosp. Pediatr. 2015, 5, 605–612.

34. Masarweh, K.; Gur, M.; Toukan, Y.; Bar-Yoseph, R.; Kassis, I.; Gut, G.; Hakim, F.; Nir, V.; Bentur, L. Factors associated with complicated pneumonia in children. Pediatr. Pulmonol. 2021, 56, 2700–2706.

35. Cowles, R.A.; Lelli, J.L.; Takayasu, J.; Coran, A.G. Lung resection in infants and children with pulmonary infections refractory to medical therapy. J. Pediatr. Surg. 2002, 37, 643–647.

36. Masters, I.B.; Isles, A.F.; Grimwood, K. Necrotizing pneumonia: An emerging problem in children? Pneumonia 2017, 9, 11.

BÖLÜM 2

ÇOCUKLUK ÇAĞINDA TRAKEABRONŞİAL YABANCI CİSİM ASPİRASYONLARINA YAKLAŞIM

SUZAN TEMİZ BEKCE[1]

1-Giriş

Çocukluk çağında trakeobronşial yabancı cisim aspirasyonları (YCA); sık karşılaşılan, zamanında müdahale edilmediğinde ciddi komplikasyonlara ve mortaliteye neden olabilen acil bir klinik durumdur (Ding & ark., 2022:873182). Dünya genelinde YCA nedeniyle pediatrik olgularda başvuru oranı ortalama 109/100000 olup; bu oran çocuk acil servis başvurularının yaklaşık %0.2-0.4'ünü oluşturmaktadır (Wu & ark., 2023:1235308; Ocaglı & ark., 2021:938).

Aspirasyonlar her yaş grubunda görülebilmekle birlikte, yapılan çalışmalar olguların en sık 3 yaş altı çocuklarda gerçekleştiğini göstermektedir (Ding & ark., 2020:e20480; Chapin & ark., 2013:275). Literatürde en sık ilişkilendirilen cinsiyet erkektir (Chang & ark., 2021:110685; Ulas, Aydın & Eroglu, 2022:1168; Sahin & ark., 2013:658), bazı çalışmalarda her iki

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cinsiyet için de benzer oranlar bildirilmiştir (Huang & ark., 2015:2394; Baram & ark., 2017:2333794; Gregori & ark., 2008:971).

Aspire edilen materyalin türü; toplumun kültürel yapısı, bölgesel farklılıklar ve beslenme alışkanlıklarına göre değişkenlik gösterir (Kısacık & ark., 2004:86; Mise & ark., 2009:1360). Gıda maddeleri, bebekler ve küçük çocuklar tarafından en sık aspire edilen nesnelerken; gıda dışı maddeler (örn. madeni paralar, ataçlar, iğneler, kalem kapakları) daha çok büyük çocuklar tarafından aspire edilmektedir (Rimell & ark., 1995:1763; Lemberg, Darrow & Holinger, 1996:267; Jiaqiang & ark., 2009:1708).

Aspirasyon ilişkili ölümlerin yaklaşık üçte ikisinin, olguların sağlık kuruluşuna ulaşmadan evde gerçekleştiği bildirilmiştir (Bresler, Gren & Holinger, 1999:430). Tanının hızlı konulması ve erken müdahale; komplikasyonların, morbiditenin önlenmesi ve mortalitenin azaltılmasında kritik öneme sahiptir (Baram & ark., 2017:2333794). Sağlık hizmetlerindeki gelişmelere ve toplumsal farkındalığın artmasına rağmen günümüzde halen yaygın olarak görülmektedir.

2. Klinik Değerlendirme

YCA olgularında; tanıya götüren en önemli kriter öyküdür. Semptom ve bulgular; aspire edilen cismin tipi, çocuğun yaşı ve aspirasyon ile başvuru arasındaki zaman gibi faktörlere bağlı olarak değişkenlik gösterir (Musani & ark., 2010:178; Yalçın & ark., 2025:76).

Semptomlar 3 evrede sınıflandırılmaktadır; akut dönem, asemptomatik dönem ve komplikasyon dönemi (Gorelik & Schroeder, 2025:2578).

Akut dönemde: aspirasyonu takiben öksürük, boğulma, öğürme, dispne, göğüs duvarı retraksiyonu, hırıltı, taşipne ve

stridor görülebilir. Literatürde en sık bildirilmiş semptomlar öksürük, boğulma ve hırıltı yer almaktadır (Burton & ark., 1996:195; Tan & ark., 2000:91; Even & ark., 2005:1122). Obstruksiyona bağlı hipoksi bulgularıda bu fazda görülmektedir.

Asemptomatik dönem: Yabancı cismin trakeobronşial sisteme yerleşmesine bağlı olarak refleksler yorulur ve ilk oluşan semptomların şiddeti azalabilir. Semptomların olmaması yanıltıcı olabilir.

Komplikasyon dönemi: Bu fazda enfeksiyona veya kronik obstruksiyona bağlı klinik tablo ön plana çıkmaktadır. Pnömoni, apse, atelektazi, bronşektazi, ateş, balgam çıkarma, hemoptizi, pnömotoraks, pnömomediastinum, genel durum bozukluğu ve öksürük görülebilir.

Semptomların ortaya çıkmasıyla tanı ve müdahale arasında geçen sürenin olgular arasında bir saatten birkaç aya kadar değiştiği bildirilmiştir (Scmidt & Manegold, 2000:644; Mu, He & Sun, 1991:657; Wolach & ark., 1994:1; Paşaoğlu & ark., 1991:95).

Aspirasyona spesifik olmayan semptom ve bulguların olması, aspirasyon öyküsünün net olmaması tanının gecikmesine ya da komplikasyonların gelişmesine sebep olabilir.

Değerlendirmede ilk tercih edilen görüntüleme yöntemi anteroposterior akciğer grafisidir. Yabancı cisim radyolusent özellikte ise direkt seçilebilir. Grafi değerlendirildiğinde; atelektazi, havalanma artışı, mediastinal kayma, kronik dönemde bronşektazi, pnömonik infiltrasyonları içerebilir. Bununla birlikte röntgen bulgularında herhangi bir özellik olmayabilir, doğal olabilir. Gerekli olgularda bilgisayarlı toraks tomografisi planlanabilir. Radyolojik incelemeler aspirasyonu doğrulamaya yardımcı olabilirse de dışlamak için için kullanılmaması önerilmektedir (Rance & ark., 2022:424).

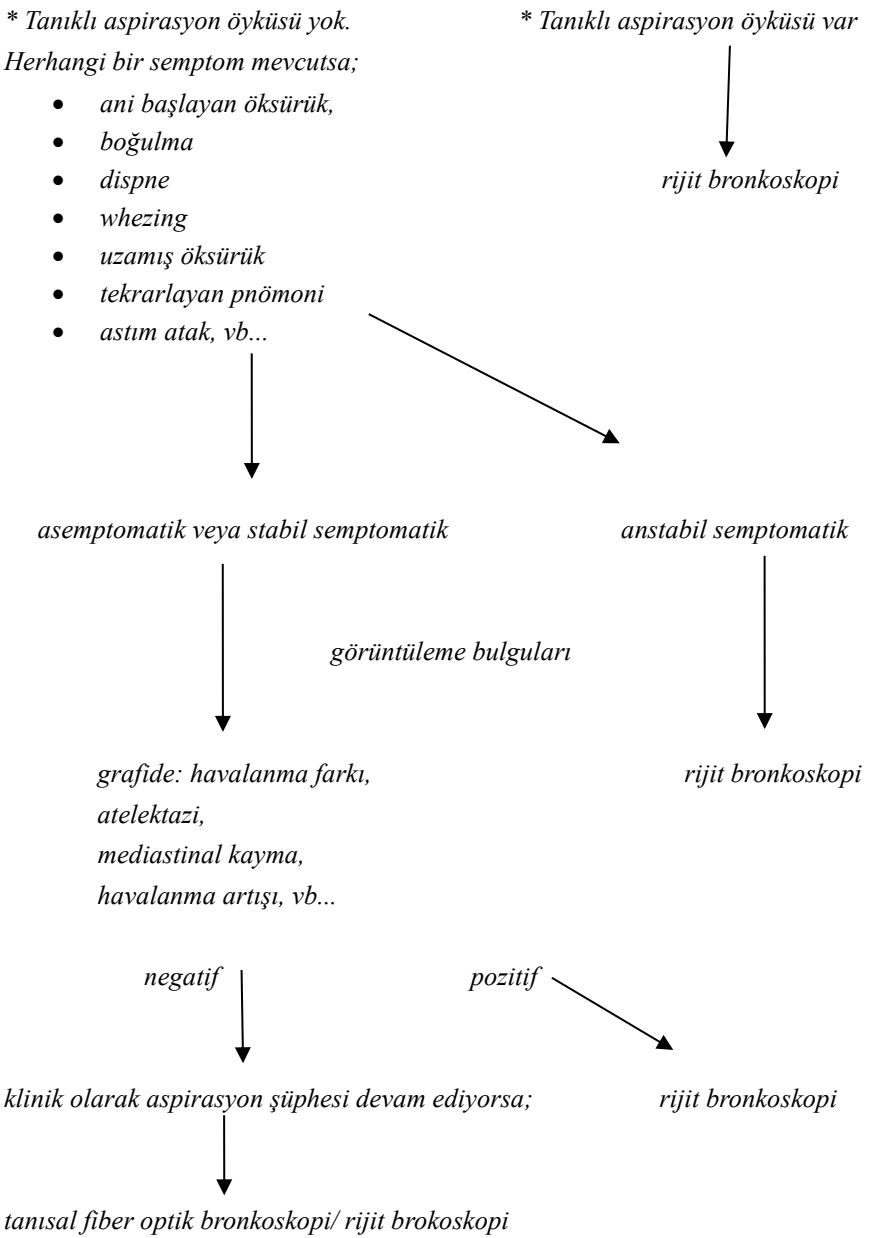
3. Tanı ve Yaklaşım Algoritması

Hızlı müdahale gerektirmesi nedeniyle YCA olgularını tespit etmenin zor olduğu klinik durumlar mevcuttur. Bronş ağacında bir yabancı cisim olmasına rağmen, olguların küçük bir yüzdesinin tamamen asemptomatik olabileceği unutulmamalıdır (Ulas, Aydın & Eroglu, 2022:1168; Laks & Barzilay, 1988:102; Losek, 1990:348).

Broronkoskopinin tanısal ve terapötik olarak ikili rolü mevcuttur. Genel anestezi altında rijit bronkoskopi, yaygın olarak tercih edilen prosedürdür (Eber & ark., 2017:1; Sheehan, Lopez & Elmaraghy, 2018:72; Kısacık & ark., 2004:86; Shao & ark., 2023:1114043).

Sadece tanıklı aspirasyon öyküsü ve fizik muayene bulguları ile acil rijit bronkoskopiye alınan olgular literatürde yer almaktadır. Öykünün belirsiz olduğu veya radyolojik bulgu saptanmayan yabancı cisim aspirasyonlarının geri dönüşümsüz komplikasyonları göz önüne alındığında, en ufak bir şüphe varlığında bronkoskopi yapılması önerilmektedir (Black, Johnson & Matlak, 1994:682; Zerella & ark., 1998:1651; Mu, He & Sun, 1991:657; Sinha, Talagauara Umesh & Jha, 2017:449; Karakoc & ark., 2007:241).

1. YCA şüphesi taşıyan olgulara yaklaşım algoritması



Son yıllarda fiber optik bronkoskopinin tek başına yada rijit bronkoskopiye birlikte başarıyla yürütülen olgular yayınlanmıştır (Reugemer & Perkins, 1999:77; Wood & Gauderer, 1984:693; Kim & ark., 2018:82; Tenenbaum & ark., 2017:21; Tang & ark., 2009:191; Han & ark., 2022:159). Rijit bronkoskopi işlemi sırasında, hava yolunun güvenli bir şekilde korunabilmesi ve forseplerle manipülasyonun geniş çalışma kanalında daha etkin yapılabilmesi, bu yöntemi fiber optik bronkoskopiye kıyasla güvenlik avantajı sağlar (Righini & ark., 2007:1383).

Fiber optik bronkoskopi tanısal amaçlı yüksek şüpheli hastalarda kullanımı önerilmekle birlikte gereğinde rijit bronkoskopinin hazır olması gerektiği, hatta cerrahi gerekebileceği unutulmamalıdır. Rijit bronkoskopi yapılan olgularda daha az komplikasyon görüldüğü yayınlanmıştır (Wiemers & ark., 2023:111474).

Yabancı cismin fiber optik bronkoskopi veya rijit bronkoskopi ile çıkarılamayacağı düşünülen olgularda açık cerrahi gerekebilir (Luna & ark., 2017:50e1; Shlizerman & ark., 2010:320; Tan & ark., 2000:91; Even & ark., 2005:1122).

Trakeabronşial YCA'ya yönelik girişimlerin yanı sıra önleme stratejilerinin geliştirilmesi de desteklenmelidir. Klinik yönetimin standardizasyonu için; özellikle risk altındaki pediatrik popülasyonlar ve karmaşık vakalara yönelik ayrıntılı tedavi kılavuzlarına, rehber ve protokollere ihtiyaç vardır. Hem tanı hem tedavi açısından temel ve güvenilir bir yöntem olarak rijit bronkoskopi öne çıkmaktadır, uygun merkezlerde deneyimli ekip tarafından uygulanması önerilir. Karar verme sürecinde sadece öykü değil; semptom, fizik muayene, radyoloji bulguları ve olası risk-fayda dengesi dikkatle değerlendirilmelidir. Şüpheli olgularda müdahale için geç kalmamak kritik önem taşır, radyolojik veya klinik bulgu yokluğu olsa bile trakeobronşial yabancı cisim aspirasyon olasılığı akılda tutulmalıdır.

Kaynakça

Baram, A., Sherzad, H., Saeed, S., Kakamad, F.H., & Hamawandi, A.M.H. (2017). Tracheobronchial foreign bodies in children: the role of emergency rigid bronchoscopy. *Global Pediatric Health.* 4,2333794x17743663. Doi: 10.1177/2333794X17743663

Black, R.E., Johnson, D.G., & Matlak, M.E. (1994). Bronchoscopic removal of aspirated foreign bodies in children. *J Pediatr Surg.* 29,682-684. Doi: 10.1016/0022-3468(94)90740-90744

Bressler, K.L., Gren, C.G., & Holinger, L.D. (1999). Foreign Body Aspiration. In: Taussig LM, Landau LI (eds.), *Pediatric Respiratory Medicine* (pp. 430-435.), Mosby, St.Louis.

Burton, E.M., Brick, W.G., Hall, J.D., Riggs, Wjr., & Houston, C.S., (1996). Tracheobronchial foreign body aspiration in children. *South Med J.* 89,195-198. Doi: 10.1097/00007611-199602000-00007.

Chang, D.T., Abdo, K., Bhatt, J.M., Huoh, K.C., Pham, N.S., & Ahuja, G.S. (2021). Persistence of Choking Injuries in Children. *Int. J. Pediatr. Otorhinolaryngol.* 144,110685. Doi: 10.1016/j.ijporl.

Chapin, M.M., Rochette, L.M., Annest, J.L., Haileyesus, T., Conner, K.A., & Smith, GA. (2013). Nonfatal choking on food among children 14 years or younger in the United States, 2001-2009. *Pediatrics.* 132,275. Doi: 10.1542/peds.2013-0260

Ding, G., Wu, B., Vinturache, A., Cai, C., Lu, M., & Gu, H. (2020). Tracheobronchial foreign body aspiration in children: A retrospective single-center cross-sectional study. *Medicine (Baltimore).* 99(22),e20480. Doi: 10.1097/MD.0000000000020480.

Ding, L., Su, S., Chen, C., Yao, H., & Xiao, L. (2022). Tracheobronchial Foreign Bodies in Children: Experience From 1,328. *Patients in China.* Front Pediatr. 10,873182. Doi:10.3389/fped.2022.873182

Eber, E., Antón-Pacheco, J.L., de Blic, J., Doull, I., Faro, A., Nenna, R., Nicolai, T., Pohunek, P., Priftis, K.N., Serio, P,. Coleman, C., Masefield, S., Tonia, T., & Midulla, F. (2017). ERS statement: interventional bronchoscopy in children. *Eur. Respir. J.* 50,1-19. Doi: 10.1183/13993003.00901-2017.

Even, L., Heno, N., Talmon, Y., Samet, E., Zonis, Z., & Kugelman, A. (2005). Diagnostic evaluation of foreign body aspiration in children: a prospective study. *J Pediatr Surg.* 40,1122-1127. Doi: 10.1016/j.jpedsurg.2005.03.049.

Gregori, D., Salerni, L., Scarinzi, C., Morra, B., Berchialla, P., Snidero, S., Corradetti, R., & Passali, D. (2008). Foreign bodies in the upper airways causing complications and requiring hospitalization in children aged 0-14 years: results from the ESFBI study. *Eur Arch Otorhinolaryngol.* 265,971–978. Doi: 10.1007/s00405-007-0566-8

Gorelik, M., & Schroeder, J.W. (2025). Foreign Bodies in the Airway. In: Kliegman, R.M., St Geme, J.W., Blum, N.J., Tasker, R.C., Wilson, K.M., Schuh, A.M., Mack, C.L., (Eds.), *Nelson Textbook of Pediatrics* (22nd ed., pp.2578-2578.e1). Philadelphia: Elsevier. https://www.clinicalkey.com/#!/content/book/3-s2.0-B9780323883054004351

Han, L.L., Meng, C., Zhang, Z.X., Tang, Z.D., Ma, J., & Li, C.X. (2022). Clinical analysis of bronchoscope diagnosis and treatment for airway foreign body removal in pediatric patients. *Ital J Pediatr*. 48(1),159. Doi: 10.1186/s13052-022-01347-x.

Huang, Z., Zhou, A., Zhang, J., Xie, L., & Li, Q. (2015). Risk factors for granuloma formation in children induced by tracheobronchial foreign bodies. *Int J Pediatr Otorhinolaryngol.* 79,2394–2397. Doi: 10.1016/j.ijporl.2015.10.057

Jiaqiang, S., Jingwu, S., Yanming, H., Qiuping, Lv., Yinfeng, W. Xianguang, Li., Guanglun, W., & Demin, H. (2009). Rigid bronchoscopy for inhaled pen caps in children. *J Pediatr Surg.* 44,1708. Doi: 10.1016/j.jpedsurg.2008.11.035

Karakoc, F., Cakir, E., Ersu, R., Uyan, Z.S., Colak, B., Karadag, B., Kiyan, G., Dagli, T., & Dagli, E. (2007). Late diagnosis of foreign body aspiration in children with chronic repiratory symptoms. *Int. J. Pediatr. Otorhinolaryngol.* 71,241-246. Doi: 10.1016/j.ijporl.2006.10.006.

Kısacık, E., Gülhan, E., Sırmalı, M., Sarıca, E., Türüt, H., Karasu, S., Gezer, S., & Kaya, S.(2004). Trakeobronşiyal yabancı cisim aspirasyonları: 261 olgunun analizi. *Solunum Hastalıkları.* 15,86-91.

Kim, K., Lee, H.J., Yang, E.A., Kim, H.S., Chun, Y.H., Yoon, J.S., Kim, H.H., & Kim, J.T. (2018). Foreign body removal by flexible bronchoscopy using retrieval basket in children. *Ann Thorac Med.* 13,82–85. Doi: 10.4103/atm.ATM_337_17.

Laks, Y., & Barzilay, Z. (1988). Foreign body aspiration in childhood. *Pediatr Emerg Care.* 4,102–106. Doi: 10.1097/00006565-198806000-00004.

Lemberg, PS. Darrow, DH. & Holinger, LD. (1996) Aerodigestive tract foreign bodies in the older child and adolescent. *Ann Otol Rhinol Laryngol.* 105,267. Doi: 10.1177/000348949610500404

Losek, JD. (1990). Diagnostic difficulties of foreign body aspiration in children. *Am J Emerg Med.* 8,348–350. Doi: 10.1016/0735-6757(90)90094.

Luna, J., Olabarri, M., Domènech, A., Rubio, B., Yagüe, F., Benítez, M.T., Esparza, M.J., & Mintegi, S. (2017). Recommendations for the prevention of foreign body aspiration. *An. Pediatr (Barc).* 86;50e1-50e6. Doi: 10.1016/j.anpedi.2016.04.013.

Mise, K., Savicevic, A.J., Pavlov, N., & Jankovic, S. (2009). Removal of tracheobronchial foreign bodies in adults using flexible bronchoscopy: experience 1995-2006. *Surg Endosc.* 23,1360-1364. Doi: 10.1007/s00464-008-0181-9

Mu, L., He, P., & Sun, D. (1991). Inhalation of foreign bodies in Chinese children: A review of 400 cases. *Laryngoscope.* 101(6 Pt 1), 657–660. Doi: 10.1288/00005537-199106000-00014.

Musani, M.A., Khambaty, Y., Jawed, I., Khan, F.A., & Ashrafi, S.K.A. (2010). An Unusual Foreign Body in Trachea. *J Ayub Med Coll Abbottabad.* 22;178–179. PMID: 21409938.

Ocagli, H., Azzolina, D., Bressan, S., Bottigliengo, D., Settin, E., Lorenzoni, G., Gregori, D., & Dalt, L.V. (2021). Epidemiology and trends over time of foreign body injuries in the pediatric emergency department. *Children.* 8(10),938. Doi: 10.3390/children8100938

Paşaoğlu, I., Doğan, R., Demircin, M., Hatipoğlu, A., & Bozer, A.Y. (1991). Bronchoscopic removal of foreign bodies in children: retrospective analysis of 822 cases. *Thorac Cardiovasc Surg.* 39,95–98. Doi: 10.1055/s-2007-1013940.

Rance, A., Mittaine, M., Michelet, M., Martin Blondel, A., & Labouret, G. (2022). Delayed diagnosis of foreign body

aspiration in children. *Archives de Pediatrie.* 29(6),424-428. Doi: 10.1016/j.arcped.2022.05.006

Reugemer, J.L., & Perkins, J.A. (1999). Combined rigid and flexible endoscopic removal of a BB foreign body from a peripheral bronchus. *Int J Pediatr Otorhinolaryngol.* 47,77-80. Doi: 10.1016/s0165-5876(98)00164-5.

Righini, C.A., Morel, N., Karkas, A., Reyt, E., Ferretti, K., Pin, I., & Schmerber, S. (2007). What is the diagnostic value of flexible bronchoscopy in the initial investigation of children with suspected foreign body aspiration?. *Int J Pediatr Otorhinolayngol.* 71,1383-1390. Doi: 10.1016/j.ijporl.2007.05.012.

Rimell, F.L., Thome Jr, A., Stool, S., Reilly, J.S., Rider, G., Stool, D., & Wilson, C.L. (1995). Characteristics of objects that cause choking in children. *JAMA.* 274,1763. PMID: 7500505.

Sahin, A., Meteroglu, F., Eren, S., & Celik, Y. (2013). Inhalation of foreign bodies in children: experience of 22 years. *J Trauma Acute Care Surg.* 74,658. Doi: 10.1097/TA.0b013e3182789520.

Schmidt, H., & Manegold, B.C. (2000). Foreign body aspiration in children. *Surgical Endoscopy.* 14,644–648. Doi: 10.1007/s004640000142.

Shao, H., Li, S., He, J., Wu, L., & Chen, Z. (2023). A combination of flexible and rigid bronchoscopy in the successful removal of a residual fish bone from a peripheral bronchus: A case report. *Front. Pediatr.* 11,1114043. Doi: 10.3389/fped.2023.1114043

Sheehan, C.C., Lopez, J., & Elmaraghy CA. (2018). Low rate of positive bronchoscopy for suspected foreign body aspiration in infants. *International Journal of Pediatric Otorhinolaryngology.* 104,72–75. Doi: 10.1016/j.ijporl.2017.10.030.

Shlizerman, L., Mazzawi, S., Rakover, Y., & Ashkenazi, D. (2010). Foreign body aspiration in children: the effects of delayed diagnosis. *Am. J. Otolaryngol.* 31,320-324. Doi: 10.1016/j.amjoto.2009.03.007.

Sinha, V., Talagauara Umesh, S., & Jha, S.G. (2017). Rigid Bronchoscopy in Pediatric Patients. *Indian J Otolaryngol Head Neck Surg.* 69(4),449–452. Doi: 10.1007/s12070-017-1222-2

Tan, H.K., Brown, K., McGill, T., Kenna, M.A., Lund, D.P., & Healy, G.B., (2000). Airway foreign bodies (FB): a 10-year review. *Int J Pediatr Otorhinolaryngo*l. 56,91-99. Doi: 10.1016/s0165-5876(00)00391-8.

Tang, L.F., Xu, Y.C., Wang, Y.S., Wang, C.F., Zhu, G.H., Bao, X.E., Lu, M.P., Chen, L.X., & Chen, Z.M. (2009). Airway foreign body removal by flexible bronchoscopy: experience with 1027 children during 2000-2008. *World J Pediatr.* 5(3),191-195. Doi: 10.1007/s12519-009-0036-z.

Tenenbaum, T., Kähler, G., Janke, C., Schroten, H., & Demirakca, S. (2017). Management of foreign body removal in children by flexible bronchoscopy. *J Bronchol Interv Pulmonol.* 24,21–28. Doi: 10.1097/LBR.0000000000000319.

Ulas, A.B., Aydın, Y., & Eroglu, A. (2022). Foreign body aspirations in children and adults. *The American Journal of Surgery.* 224(4),1168-1173. https://doi.org/10.1016/j.amjsurg.2022.05.032

Wiemers. A., Vossen, C., Lücke, T., Freitag, N., Nguyen, TMTL. Möllenberg, L. Pohunek, P. & Schramm, D. (2023). Complication rates in rigid vs. flexible endoscopic foreign body removal in children. *Int J Pediatr Otorhinolaryngol.* 166,111474. Doi: 10.1016/j.ijporl.2023.111474.

Wolach, B., Raz, A., Weinberg, J., Mikulski, Y., Ben Ari, j., & Sadan, N. (1994). Aspirated foreign bodies in the respiratory tract of children: eleven years experience with 127 patients. I*nt J Pediatr Otorhinolaryngol.* 30,1–10. Doi: 10.1016/0165-5876(94)90045-0.

Wood, R.E., & Gauderer, M.W.L. (1984). Fexible fiberoptic bronchoscopy in the manegement of tracheobronchial foreign bodies in children: The value of a combined approach with open tube bronchoscopy. *J Pediatr Surg.* 19,693-698. Doi: 10.1016/s0022-3468(84)80354-1

Wu, Y., Zhang, X., Lin, Z., Ding, C., Wu, Y., Chen, Y., Wang, D., Yi, X., & Chen, F. (2023). Changes in the global burden of foreign body aspiration among under-5 children from 1990 to 2019. *Front Pediatr.* 11,1235308. Doi:10.3389/fped.2023.1235308

Yalçın, G., Özdemir Balcı, Ö., Ter, M., Tonak, B.S., Yalçınkaya, İ.Ş., Neşeli, Z., & Anıl, M. (2025). Evaluation and Management of Airway Foreign Bodies in Emergency Settings: A Case Report. *Turkish Medical Journal.* 10(2),76-80. https://doi.org/10.70852/

Zerella, J.T., Dimler, M., McGill, L.C., & Pippus, K.J. (1998). Foreign body aspiration in children: Value of radiography and complications of bronchoscopy. *J Pediatr Surg.* 11,1651-1654. Doi: 10.1016/s0022-3468(98)90601-7.

BÖLÜM 3

Pleural and Intrapulmonary Solitary Fibrous Tumors: Diagnosis and Surgical Management

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1. Introduction

Solitary fibrous tumors (SFTs) are uncommon mesenchymal neoplasms initially described as localized fibrous pleural tumors. Advances in histopathology, immunohistochemistry, and molecular diagnostics have reclassified SFTs as fibroblastic neoplasms with a characteristic NAB2–STAT6 gene fusion. While the pleura remains the most frequent site, SFTs can occur in diverse locations, including lungs, mediastinum, meninges, liver, bone, and soft tissues; thoracic SFTs account for over two-thirds of cases (Walid, 2012; Cardillo et al., 2012).

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Within the thorax, SFTs predominantly arise from the visceral pleura; parietal pleural and intrapulmonary tumors are rare, the latter representing ~3.5% of thoracic SFTs (Sakurai et al., 2008; Alghamdi et al., 2020). Intrapulmonary SFTs often mimic other pulmonary lesions, posing preoperative diagnostic challenges.

Most SFTs behave benignly; however, 10–20% exhibit malignant features, including aggressive local invasion, high mitotic activity, necrosis, or metastasis (Demicco et al., 2012; Khouzam & Khouzam, 2022). Tumors from parietal, fissural, or mediastinal pleura, particularly with parenchymal invasion, have a higher malignancy risk (Ajouz et al., 2023; Dörr et al., 2024). Histopathology alone may not predict behavior; benign tumors show a “patternless pattern” with collagenous stroma and branching vasculature, whereas malignant forms demonstrate hypercellularity, pleomorphism, mitoses, necrosis, and infiltrative borders (Demicco et al., 2012).

Immunohistochemistry aids diagnosis: SFTs are typically positive for CD34, Bcl-2, and vimentin, negative for cytokeratin. Nuclear STAT6, resulting from NAB2–STAT6 fusion, is the most sensitive and specific marker, distinguishing SFTs from mimics such as hemangiopericytoma or synovial sarcoma (Sagawa et al., 2007; Khouzam & Khouzam, 2022).

Radiologically, pleural SFTs present as well-circumscribed, lobulated masses, often incidentally discovered due to asymptomatic presentation in up to 50% of patients (Cardillo et al., 2012; Walid, 2012). Large tumors may cause chest pain, dyspnea, or paraneoplastic syndromes such as hypoglycemia (Doege–Potter syndrome) or hypertrophic osteoarthropathy. Intrapulmonary SFTs are usually asymptomatic unless large; their origin is debated but likely involves visceral pleura, subpleural mesenchyme,

interlobular septa, or pulmonary pericytes (Sakurai et al., 2008; Alghamdi et al., 2020).

CT imaging often shows well-defined, lobulated masses with heterogeneous enhancement. Malignant features include irregular borders, necrosis, pleural thickening, or adjacent invasion, yet imaging and needle biopsy cannot reliably distinguish malignancy, necessitating surgical excision for definitive diagnosis (Ajouz et al., 2023).

Complete surgical resection with negative margins is the cornerstone of treatment, crucial for long-term survival. Wedge resection is adequate for small peripheral lesions, whereas lobectomy or advanced techniques are required for larger, centrally located, or extensively invasive tumors. The hypervascular nature of some SFTs increases intraoperative bleeding risk, emphasizing careful surgical planning (Walid, 2012; Ajouz et al., 2023).

Long-term follow-up is recommended for all patients, as recurrences can occur decades later. Prognostic factors include malignancy, high mitotic index, tumor size, hypercellularity, necrosis, and incomplete resection. The Demicco risk model, incorporating age, tumor size, mitotic rate, and necrosis, aids metastatic risk prediction (Demicco et al., 2012).

Despite limited prospective studies, literature and case reports underscore key points: SFTs should be considered in the differential diagnosis of pleural or intrapulmonary masses; imaging and biopsy alone are insufficient; complete surgical resection provides excellent outcomes; and careful assessment for malignant features is essential. This chapter focuses on malignant SFTs, particularly intrapulmonary cases and parenchymal invasion, examining anatomical, pathological, radiological, and clinical

features, diagnostic strategies, and surgical management, illustrated by a representative case study demonstrating the challenges in diagnosis and importance of complete resection

2. Anatomy and Pathogenesis of Solitary Fibrous Tumors

Solitary fibrous tumors (SFTs) originate predominantly within the thoracic cavity, particularly from the pleura, making a detailed understanding of pleural anatomy essential for appreciating their growth patterns, clinical manifestations, and potential for malignant transformation. Although SFTs are classified as fibroblastic mesenchymal neoplasms capable of developing in nearly every anatomical region, their predilection for the pleura suggests a meaningful relationship between tumor biology and the structural, embryologic, and functional characteristics of this tissue. This section outlines the anatomy of the pleura and adjacent thoracic structures, explores the cellular and molecular origins of SFTs, and examines mechanisms believed to underlie tumorigenesis and malignant progression.

2.1 Anatomy of the Pleura and Adjacent Structures

The pleura consists of two thin serous membranes: the visceral pleura, which tightly adheres to the lung surface, and the parietal pleura, which lines the thoracic cavity, ribs, mediastinum, and diaphragm. These layers form the pleural space, a potential cavity containing a small amount of lubricating fluid that facilitates lung expansion during respiration. The visceral pleura is richly supplied with capillaries originating from the bronchial circulation, whereas the parietal pleura receives blood primarily from the intercostal, internal thoracic, and musculophrenic arteries. Lymphatic drainage is highly efficient and plays a crucial role in maintaining pleural fluid homeostasis.

Anatomically, the visceral pleura is composed of a single layer of mesothelial cells overlying connective tissue rich in elastin, collagen, fibroblasts, and sparse mesenchymal progenitor cells. These characteristics help explain the presumed origin of SFTs: a neoplastic proliferation arising from primitive mesenchymal or submesothelial fibroblastic cells in the pleural connective tissue (Walid, 2012; Cardillo et al., 2012). The parietal pleura, though structurally similar, contains a greater density of somatic innervation, making masses in this region more likely to produce symptoms such as chest pain.

The proximity of the pleura to the pulmonary parenchyma, interlobar fissures, and mediastinum helps elucidate how some SFTs extend into or originate within the lung. In particular, SFTs located along the major or minor fissures may appear intrapulmonary but actually represent pleural-based tumors growing along preformed anatomical planes. Conversely, truly intrapulmonary SFTs may arise independent of pleural attachment, likely from interstitial mesenchymal cells or perivascular fibroblasts located deep within the lung parenchyma (Sakurai et al., 2008; Alghamdi et al., 2020). These anatomical relationships are central to understanding tumor growth patterns and assessing surgical resectability.

2.2 Cellular Origin and Molecular Biology of SFTs

Early theories suggested that SFTs originated from mesothelial cells, largely because most early cases were localized to the pleura. However, immunohistochemical studies soon refuted this hypothesis, as SFTs consistently demonstrated negative cytokeratin staining and lacked the phenotypic features of mesothelial-derived tumors. Instead, they displayed strong positivity for CD34, vimentin, and Bcl-2—markers characteristic

of immature fibroblastic or dendritic mesenchymal cells. This profile supported a mesenchymal origin, eventually leading to the current classification of SFTs as fibroblastic neoplasms (Walid, 2012; Sagawa et al., 2007).

A major breakthrough in SFT biology came with the identification of the NAB2–STAT6 gene fusion, a pathognomonic chromosomal rearrangement resulting from intrachromosomal inversion on chromosome 12. This fusion protein acts as an aberrant transcriptional regulator, driving uncontrolled proliferation and altering cellular differentiation pathways. Its discovery firmly established SFT as a genetically defined entity across all anatomical locations. Immunohistochemical nuclear staining for STAT6 is now regarded as the most sensitive and specific method for confirming SFT diagnosis, even in cases with ambiguous morphology (Khouzam & Khouzam, 2022).

NAB2–STAT6 fusion variants exhibit subtle differences in transcriptional activation potential, and emerging studies suggest that certain fusion subtypes may correlate with tumor behavior, histologic pattern, or risk of malignancy. Although the clinical significance of these variants is still being clarified, they highlight the molecular complexity of SFTs and reinforce the notion that malignant transformation is driven by interactions between genetic drivers, microenvironmental influences, and host factors.

Beyond STAT6, other molecular markers have been studied for their role in tumorigenesis. For example, overexpression of growth factors such as platelet-derived growth factor and transforming growth factor-β have been described in SFTs. Angiogenesis-related pathways, including those involving vascular endothelial growth factor (VEGF), contribute to the prominent branching vascular patterns observed histologically. Some

malignant SFTs demonstrate increased expression of proliferation markers such as Ki-67, supporting the association between high proliferative index and aggressive clinical behavior (Demicco et al., 2012). Collectively, these molecular features support the view that SFTs encompass a spectrum of biological behavior, modulated by both intrinsic and extrinsic factors.

2.3 Mechanisms of Tumor Development and Malignant Transformation

Although the precise mechanisms underlying the development of SFTs remain under investigation, several theories offer insights into how these tumors arise and evolve. The presence of SFTs in numerous anatomical locations suggests that progenitor mesenchymal cells capable of giving rise to SFTs are widely distributed throughout the body. In the pleura, submesothelial fibroblasts or dendritic interstitial cells represent likely precursors. Cellular injury, chronic irritation, or dysregulation of signaling pathways may contribute to their neoplastic transformation, although definitive causative factors are not yet known.

One central question concerns the origin of intrapulmonary SFTs. Several mechanisms have been proposed:

1) Inward growth from visceral pleura**:** Some lesions thought to represent intrapulmonary SFTs may actually originate on the visceral pleural surface and grow into the lung parenchyma. Given the close adhesion of visceral pleura to the lung surface, a small pleural tumor could easily become engulfed by expanding lung tissue or extend inward along connective-tissue septa.

2) De novo origin from intrapulmonary mesenchyme**:** This theory proposes that SFTs arise from resident interstitial

fibroblasts or dendritic cells within the pulmonary parenchyma. Evidence supporting this includes cases with no demonstrable pleural attachment, as well as tumors located deep within the lung away from fissures or subpleural regions (Sakurai et al., 2008; Alghamdi et al., 2020).

3) Origin from perivascular cells**:** The rich capillary network of the lung contains pericytes and fibroblast-like cells that may serve as progenitors for SFTs. This theory aligns with the highly vascular nature of many SFTs and the presence of staghorn-like vasculature reminiscent of other perivascular neoplasms.

Malignant transformation in SFTs is multifactorial. Histologically malignant SFTs exhibit increased mitotic activity, significant nuclear atypia, necrosis, hypercellularity, and infiltrative growth patterns. Tumor size also correlates with malignant potential, with lesions larger than 10 cm demonstrating a significantly higher likelihood of adverse outcomes (Demicco et al., 2012). Additional predictors include older patient age and presence of tumor necrosis—each incorporated into the Demicco risk assessment model.

The microenvironment appears to play a role in malignant progression. Regions of hypoxia within larger tumors may promote necrosis and stimulate angiogenic pathways that facilitate rapid growth and potential metastasis. Tumors arising in the parietal pleura or mediastinal regions may develop more aggressive behavior due to differences in stromal composition, vascular supply, or local mechanical forces. For intrapulmonary tumors, the relationship between growth pattern and local microenvironment remains an area of ongoing study, but cases involving fissural invasion or extension along interstitial septa

suggest pathways that may predispose to more infiltrative behavior (Ajouz et al., 2023).

Despite these insights, the distinction between benign and malignant SFTs remains challenging. Some tumors classified as benign based on morphology eventually metastasize, while others with malignant histology remain indolent. This unpredictability underscores the importance of incorporating clinical, radiologic, histologic, and molecular findings into a comprehensive risk assessment rather than relying on any single parameter.

3. Classification and Histopathological Features of Solitary Fibrous Tumors

Solitary fibrous tumors (SFTs) are fibroblastic mesenchymal neoplasms with variable clinicopathologic behaviors. Historically regarded as benign pleural lesions, SFTs are now recognized as a heterogeneous group capable of arising throughout the body with variable malignant potential. Modern classification integrates histologic variants, cytologic features, immunohistochemistry, and criteria associated with aggressive behavior, unified by the NAB2–STAT6 gene fusion.

3.1 Historical Evolution of SFT Classification

Early descriptions emphasized spindle-cell morphology and benign pleural origin. CD34 positivity and lack of cytokeratin helped distinguish SFTs from mesothelial tumors (Walid, 2012; Sagawa et al., 2007).

Extrapleural occurrences—including meninges, soft tissues, retroperitoneum, and lung—expanded classification frameworks (Alghamdi et al., 2020). Hemangiopericytoma (HPC), previously considered distinct, shares morphology, immunophenotype, and

clinical behavior with SFTs. The NAB2–STAT6 fusion gene unified these entities under a single WHO-recognized diagnostic category (Khouzam & Khouzam, 2022).

3.2 Major Histologic Subtypes of SFTs

3.2.1 Conventional (Classic) SFT

Characterized by a “patternless pattern” of bland spindle-to-ovoid cells in collagenous stroma, with alternating hypercellular and hypocellular areas, branching “staghorn” vessels, low mitotic activity, and minimal atypia. These tumors are typically indolent, though size alone is not predictive of outcome (Sachan, 2024; Cardillo et al., 2012).

3.2.2 Cellular (Hypercellular) SFT

Displays increased cellular density, reduced collagen, and fascicular or storiform architecture. Features include moderate to marked hypercellularity, higher mitotic rate, more prominent nucleoli, and occasional necrosis. Cellular SFTs have higher malignant potential and may correlate with specific NAB2–STAT6 variants (Demicco et al., 2012).

3.2.3 Malignant SFT

Defined by ≥4 mitoses/10 HPF, nuclear pleomorphism, hyperchromasia, infiltrative margins, necrosis, and loss of the classic patternless architecture. These tumors carry increased risk of recurrence, metastasis, and mortality. Nevertheless, morphologically aggressive tumors may behave indolently, and some bland tumors may metastasize, highlighting the complex biological spectrum of SFTs (Demicco et al., 2012).

3.3 Cytologic Characteristics

SFTs consist of monomorphic spindle, ovoid, or fusiform cells with tapering cytoplasmic processes, fine chromatin, and indistinct cytoplasmic borders producing a syncytial appearance. Nucleoli are inconspicuous in conventional tumors but more prominent in cellular or malignant variants. Mitotic figures are rare in benign SFTs but more frequent in malignant forms.

Fine-needle aspiration (FNA) may show spindle cells with collagen fragments, mixed cellularity, and fragments of branching vasculature, but FNA alone is rarely diagnostic. Core biopsy is preferred for assessing architectural patterns critical for classification (Sakurai et al., 2008).

3.4 Architectural Patterns

SFTs display a “patternless pattern,” with cells arranged haphazardly. Additional features include:

- Collagenous stroma (sparse to hyalinized)
- Alternating hypercellular and hypocellular regions
- Hemangiopericytoma-like branching vessels
- Keloid-like collagen in conventional SFTs
- Occasional myxoid degeneration

These patterns help distinguish SFTs from other spindle-cell tumors such as synovial sarcoma or leiomyosarcoma.

3.5 Immunohistochemical Profile

Immunohistochemistry (IHC) is central to the diagnosis of SFTs.

Positive markers:

- **STAT6 (nuclear):** most specific, indicates NAB2–STAT6 fusion (Khouzam & Khouzam, 2022)
- **CD34:** positive in ~90%; may decrease in malignant areas (Walid, 2012)
- **Bcl-2, CD99, Vimentin:** commonly expressed

Negative markers:

- **Cytokeratins,** help exclude sarcomatoid mesothelioma
- **S-100,** typically negative, aiding differentiation from peripheral nerve sheath tumors
- **Desmin, SMA**— usually negative or only focally positive and help exclude mesothelioma or nerve sheath tumors

Loss of CD34, strong p53, or high Ki-67 may suggest malignancy (Demicco et al., 2012).

3.6 Risk Stratifiction and Malignant Potential

SFTs exhibit unpredictable behavior. The Demicco model incorporates age, tumor size, mitotic activity, and necrosis to stratify tumors into low-, intermediate-, and high-risk for metastasis (Demicco et al., 2012). Larger tumors (>10 cm), older age, and higher mitotic rates correlate with poor outcomes. No single histologic feature reliably predicts behavior; integrated assessment with clinical, histopathologic, and molecular data provides the most accurate prognostication.

4. Clinical Presentation and Radiologic Characteristics

Solitary fibrous tumors (SFTs) of the pleura or lung parenchyma exhibit a wide range of presentations, from asymptomatic incidental findings to large, vascular tumors causing respiratory

compromise or paraneoplastic syndromes. Clinical symptoms primarily depend on tumor size, location, mass effect, invasion, and biological behavior. Malignant SFTs may remain silent until large, delaying diagnosis (Cardillo et al., 2012; Walid, 2012). Accurate clinical and radiologic assessment guides diagnosis, surgical planning, and malignancy risk stratification.

4.1 General Clinical Features

Asymptomatic presentation: 30–50% of pleural SFTs are incidentally detected. Tumors may be small and slow-growing but can still possess malignant potential (Cardillo et al., 2012; Khouzam & Khouzam, 2022).

4.2 Symptomatic Manifestations

Respiratory symptoms: Dyspnea, chest pain, dry cough, recurrent infections, and rarely hemoptysis; severity correlates with tumor size, mass effect, and adjacent structure involvement (Alghamdi et al., 2020).

Systemic/paraneoplastic symptoms:

-Doege–Potter syndrome: Tumor-derived IGF-2 causes hypoglycemia, seen in ~4% of SFTs; resolves post-resection (Walid, 2012).

-Marie–Bamberger syndrome: Digital clubbing, periostitis, and joint pain; occurs in 10–20% of symptomatic cases, likely mediated by prostaglandins, PDGF, and VEGF (Sagawa et al., 2007).

4.3 Pleural vs Intrapulmonary SFTs

•Pleural SFT**:** Mostly visceral pleura (~70%), well-defined lobulated masses; symptoms from compression, not invasion.

•Primary intrapulmonary SFT**:** Rare; may cause cough, wheezing, or obstructive pneumonia; radiologically resembles bronchogenic carcinoma (Sagawa et al., 2007).

•Pleural SFT with parenchymal invasion: Tumors extend into lung tissue, causing mixed radiologic features and potential hemoptysis or wheezing; can mimic primary lung malignancies.

4.4 Radiologic Characteristics

Imaging evaluates origin, vascularity, and invasion but cannot definitively diagnose SFT.

Chest X-ray: Often first indication; shows well-circumscribed lobulated mass with possible displacement of adjacent structures; cannot reliably distinguish pleural vs pulmonary origin (Walid, 2012).

CT: Principal modality for assessing size, morphology, origin, vascularity, and invasion.

•Pleural SFT**:** Well-defined lobulated mass, broad-based or pedunculated attachment, heterogeneous enhancement, necrosis in malignant tumors (>10 cm) (Cardillo et al., 2012).

•Intrapulmonary/ parenchymal-invading SFT**:** Surrounded by lung, irregular margins, airway displacement, occasional satellite ground-glass changes; track-like extensions indicate invasion

MRI: Complementary for chest wall, diaphragm, mediastinal invasion, vascularity, hemorrhage, or myxoid components; T1 intermediate, T2 heterogeneous, post-contrast enhancement in hypervascular areas (Cardillo et al., 2012).

PET-CT: Limited; benign tumors show low FDG uptake, malignant may be moderate/high; mainly used for staging (Demicco et al., 2012).

4.5 Radiologic Indicators of Malignancy

Suspicious features include size >10 cm, heterogeneous architecture, necrosis, pleural effusion, infiltrative borders, rapid growth, and non-enhancing areas despite vascularity (Cardillo et al., 2012).

4.6 Challenges and Preoperative Planning

Benign tumors can grow large without invasion; malignant tumors may appear well-circumscribed; PET uptake varies; intrapulmonary extension can mimic lung cancer; vascularity is inconsistent. Imaging must be integrated with clinical, histopathologic, and molecular data.

Preoperative imaging guides surgical approach, anticipated resection extent, and resectability. CT and MRI together delineate tumor origin, pedicle, vascular relationships, and involvement of lung, diaphragm, mediastinum, or chest wall. Accurate imaging is crucial, as complete resection with negative margins is the strongest predictor of long-term survival (Cardillo et al., 2020).

5. Diagnostic Workup

Evaluation of pleural and pulmonary SFTs requires a systematic, multimodal approach, combining clinical assessment, imaging, tissue sampling, histopathology, immunohistochemistry, and molecular analysis. Radiologic features may suggest SFT, but definitive diagnosis depends on characteristic morphology and immunophenotype. Distinguishing benign from malignant variants

remains challenging, highlighting the need for a comprehensive pathway (Cardillo et al., 2012; Walid, 2012).

5.1 Initial Clinical Assessment

Workup begins with a detailed history and physical exam, focusing on:

- Duration and type of respiratory symptoms
- Paraneoplastic manifestations (e.g., hypoglycaemia, arthralgia)
- Risk factors for other etiologies (smoking, prior thoracic malignancy or surgery)

Marie–Bamberger syndrome, Doege–Potter syndrome, especially in tumors >10 cm, raises suspicion for SFT (Sagawa et al, 2007; Walid, 2012)

5.2 Radiologic Imaging

Imaging is central to initial evaluation, assessing tumor morphology, vascularity, and malignancy risk.

5.2.1 Determining Pleural vs Pulmonary Origin

CT differentiates tumor origin:

- **Pleural SFT:** obtuse angle with chest wall, pleural pedicle, extrapulmonary growth
- **Intrapulmonary SFT:** acute angles, full parenchymal encasement, no pleural attachment

Intrapulmonary lesions may mimic bronchogenic carcinoma, requiring pathological confirmation (Sagawa et al., 2007).

5.2.2 Assessment of Local Invasion

CT/MRI evaluate parenchymal invasion, vascular displacement, mediastinal or chest wall involvement, and diaphragmatic/pericardial contact. Malignant tumors may show infiltrative margins and heterogeneous enhancement (Cardillo et al., 2012).

5.2.3 CT-Guided Biopsy Considerations

Percutaneous biopsy has variable yield due to tumor heterogeneity, sampling error, and limited assessment of mitotic index or necrosis. Core needle biopsy may suggest spindle-cell neoplasm but often cannot definitively grade malignancy; intraoperative evaluation or direct resection is frequently necessary (Enon et al., 2012).

5.3 Histopathologic Evaluation

Classic SFT features: patternless spindle-cell architecture, alternating cellularity, dense ropy collagen, branching “staghorn” vessels, low mitotic activity in benign tumors.

Malignant features: hypercellularity, nuclear pleomorphism, necrosis, mitotic rate >4/10 HPF, infiltrative margins. Mitotic rate and necrosis are the strongest predictors of aggressive behavior (Demicco et al., 2012).

,5.4 Immunohistochemistry

Immunohistochemical (IHC) profiling is essential for confirming the diagnosis of SFT and excluding other spindle-cell tumors.

5.4.1 Key Diagnostic Markers

Marker	Role in Diagnosis
STAT6	Diagnostic hallmark; strong nuclear staining confirms NAB2–STAT6 fusion
CD34	Positive in 80–95% of cases; not specific but highly supportive
Bcl-2	Strong, diffuse positivity; helpful but nonspecific
Vimentin	Universally positive due to mesenchymal origin
CD99	Often positive but nonspecific

STAT6 nuclear expression is considered the most specific marker and is pivotal for distinguishing SFT from histologic mimics (Khouzam & Khouzam, 2022; Demicco et al., 2012). CD34 positivity, while common, may be reduced or absent in malignant or dedifferentiated variants.

5.4.2 Differential Diagnosis by IHC:

IHC is essential for ruling out: -sarcomatoid mesothelioma (WT-1+, calretinin+), -synovial sarcoma (TLE1+, SS18-SSX fusion), -spindle-cell thymoma (CK+, EMA+), -low-grade fibromyxoid sarcoma (MUC4+).

Absence of keratin and S100 staining also supports SFT diagnosis.

5.5 Molecular and Genetic Analysis

Advances in molecular pathology have significantly improved the diagnostic accuracy of solitary fibrous tumors (SFTs). The vast majority of SFTs harbor the characteristic **NAB2–STAT6 gene fusion**, resulting from an intrachromosomal inversion on chromosome 12. This fusion leads to constitutive nuclear expression of STAT6, providing a unifying molecular hallmark across all anatomical sites.

5.5.1 Diagnostic Utility

Molecular confirmation of the NAB2–STAT6 fusion is particularly valuable in diagnostically challenging cases. Its main utilities include:

- confirming SFT diagnosis in morphologically equivocal tumors,
- supporting STAT6 immunohistochemical findings,
- differentiating SFTs from histologic mimics.

Sagawa et al. (2007) highlighted the importance of combining immunohistochemistry with molecular testing, especially in rare intrapulmonary SFTs where morphology and imaging may be misleading.

5.5.2 Risk Stratification Models

The widely used model proposed by **Demicco et al. (2012)** integrates patient age, tumor size, mitotic activity, and necrosis to

stratify tumors into low-, intermediate-, and high-risk categories for metastasis and disease-specific mortality. Rather than serving as a diagnostic tool, this model primarily informs postoperative surveillance intensity and long-term follow-up strategies.

5.6 The Role of Surgical Exploration in Diagnosis

For many pleural-based SFTs, complete surgical resection serves both diagnostic and therapeutic purposes.

5.6.1 When Surgical Diagnosis Is Preferable

-large tumors with nondiagnostic biopsy;- high suspicion for malignancy; -pedunculated tumors where biopsy is technically challenging; -risk of bleeding due to hypervascularity; -tumors with pleural mobility suggesting benignity

Ajouz et al. (2023) emphasize that preoperative biopsy can be inconclusive and may not alter the need for surgery in resectable lesions.

5.7 Limitations of Current Diagnostic Tools

Despite advancements, several diagnostic challenges persist:

- Biopsy limitations in grading malignant potential
- Radiologic overlap with other thoracic tumors
- Inconsistent PET uptake
- Difficulty identifying true extent of invasion preoperatively
- Rare intrapulmonary variants complicate localization

Therefore, final diagnosis and risk stratification often depend on postoperative histologic evaluation and complete specimen analysis.

5.8 Integrated Diagnostic Approach

An optimal diagnostic workflow integrates: 1) Clinical assessment; **2)** High-quality thoracic imaging (CT ± MRI); 3) Histologic and immunohistochemical analysis (STAT6 as key marker); 4) Molecular testing for challenging cases, 5) Surgical resection when feasible and indicated

This multidisciplinary approach ensures accurate diagnosis, appropriate surgical planning, and effective postoperative management.

6. Surgical Principles and Treatment strategies

Solitary fibrous tumors (SFTs) of the pleura and lung parenchyma present a unique set of surgical considerations due to their unpredictable biological behavior, variable anatomical location, and diverse growth patterns. Although the majority of SFTs are benign, a significant minority demonstrate malignant features capable of local invasion, recurrence, and distant metastasis (Demicco et al., 2012). Consequently, surgery remains the primary and most effective modality of treatment. The surgical approach must be formulated through a comprehensive assessment of tumor location, extent, vascularity, relationship to adjacent structures, and patient comorbidities.

This section outlines the principles guiding surgical planning, operative techniques, risk mitigation strategies, and postoperative management in the resection of pleural and

intrapulmonary SFTs. Emphasis is placed on techniques relevant to tumors arising from visceral pleura, parietal pleura, pulmonary fissures, mediastinum, and rare cases of intrapulmonary origin. The literature, including contemporary reviews and clinical series, reinforces the critical role of complete en bloc surgical resection with negative margins, which remains the cornerstone of curative therapy (Ajouz et al., 2023; Cardillo et al., 2012; Abu Arab, 2012).

6.1 Preoperative Evaluation and Surgical Planning

Preoperative evaluation aims to determine the feasibility, extent, and optimal technique of resection. A robust assessment typically includes contrast-enhanced CT, MRI for selected cases, pulmonary function testing, and a careful review of angiogenic characteristics. Large SFTs often demonstrate marked hypervascularity due to a dense capillary network, which correlates with the well-known “staghorn pattern” seen histologically (Sagawa et al., 2007). This vascularity increases intraoperative bleeding risk, necessitating careful planning.

6.1.1 Role of Imaging in Planning

As outlined in Section 3, CT is indispensable for assessing tumor size, displacement of adjacent structures, pleural attachment, and potential fissural or parenchymal extension. MRI is selectively useful for determining diaphragmatic or chest wall invasion. Functional imaging does not reliably distinguish benign from malignant SFTs; hence, radiological findings must be integrated with clinical and histopathological predictors rather than interpreted in isolation (Abu Arab, 2012).

6.1.2 Assessment of Resectability

A tumor is considered surgically resectable if complete removal with acceptable functional preservation is anticipated. Criteria favoring resectability include:

- A pedunculated tumor arising from visceral pleura
- Absence of chest wall or diaphragmatic invasion
- Lack of adherence to major mediastinal structures
- Tumor confined to a single lobe or pleural sinus

Large tumors (>10 cm) with broad-based attachment or those originating from parietal pleura require meticulous evaluation, as such lesions more commonly display malignant potential and may necessitate extended resections (Walid Abu Arab, 2012; Enon et al., 2012).

6.2 Choice of Surgical Approach: Thoracotomy versus VATS

The selection between open thoracotomy and video-assisted thoracoscopic surgery (VATS) remains an area of evolving practice. VATS is feasible for small (<5–6 cm), pedunculated, well-circumscribed SFTs with minimal vascularity. For larger or hypervascular tumors, thoracotomy is preferred due to enhanced visualization and control over bleeding.

6.2.1 Video-Assisted Thoracoscopic Surgery (VATS)

VATS offers reduced postoperative pain, shorter hospitalization, and faster recovery. It is particularly suitable for: -Mobile, pedunculated SFTs;- Lesions confined to visceral pleura; -Tumors without fissural or mediastinal involvement

However, high vascularity increases the risk of uncontrolled bleeding during thoracoscopic resection. Conversion to thoracotomy should be promptly undertaken when vascular pedicles are broad or tumor manipulation is restricted (Ajouz et al., 2023).

6.2.2 Open Thoracotomy

Thoracotomy remains the gold standard for: -Large SFTs (>8–10 cm); -Broad-based pleural attachment;-Suspected malignancy; -Tumors requiring lung parenchymal resection (segmentectomy, lobectomy): -Chest wall or diaphragmatic involvement

Open access provides superior exposure and allows safe dissection of highly vascular tumors. Most series, including those by Enon et al. (2012) and Cardillo et al. (2012), report a primary reliance on thoracotomy due to the frequent size and vascularity of SFTs. Malignant SFTs, in particular, warrant an open approach to ensure en bloc removal with oncologically adequate margins.

6.3 Principles of Tumor Resection

Complete resection with negative surgical margins (R0 resection) is universally acknowledged as the strongest predictor of disease-free and overall survival (Demicco et al., 2012; Ajouz et al, 2023). Marginal (R1) or incomplete resections (R2) significantly increase the risk of recurrence, especially in malignant tumors.

6.3.1 En Bloc Removal

En bloc resection requires removal of the tumor along with any involved pleura, lung parenchyma, or adjacent structures. In

benign SFTs, simple excision with a rim of normal tissue is generally adequate. Malignant tumors, however, often demand more extensive resection, including: Wedge resection, Segmentectomy, Lobectomy, Chest wall or diaphragmatic resection

The decision depends on the tumor's depth, attachment, and invasion. Parietal pleural tumors have a higher tendency for local invasion and may necessitate full-thickness resection including intercostal musculature (Khouzam & Khouzam, 2022).

6.3.2 Vascular Control and Hemostasis

Given the dense vascular network that characterizes SFTs, early vascular pedicle control is critical. Preoperative embolization has been reported for extremely hypervascular tumors, but this is rarely required. Intraoperatively, meticulous dissection, ligation, and hemostasis reduce morbidity.

6.4 Management Based on Tumor Location

Tumor location significantly influences both the surgical approach and the extent of resection.

6.4.1 Visceral Pleural SFTs

These are typically pedunculated and possess the most favorable surgical profile. Simple excision is frequently curative. Lobectomy or segmentectomy is rarely required unless parenchymal invasion is present.

6.4.2 Parietal Pleural SFTs

These lesions are more frequently malignant and often demonstrate broad-based attachment. Surgical management includes wide pleurectomy or chest wall resection when muscle or rib invasion is suspected (Cardillo et al., 2012).

6.4.3 Intrapulmonary SFTs

Intrapulmonary SFTs are extraordinarily rare (3.5% of cases) and often mimic primary lung tumors radiologically (Sagawa et al., 2007; Sakurai et al., 2008; Alghamdi et al., 2020). Surgical resection generally requires wedge excision or lobectomy depending on depth. Because these tumors originate within or adjacent to lung parenchyma, obtaining negative margins typically necessitates anatomical resection rather than simple enucleation.

6.4.4 Mediastinal or Fissural SFTs

Tumors arising in fissures pose diagnostic challenges and may extend into adjacent lobes. Resection often requires anatomical lobectomy or combined wedge resections of adjacent lobes.

6.5 Intraoperative Challenges and Risk Mitigation

6.5.1 Bleeding Risk: Hypervascularity is the primary intraoperative challenge. Large feeding vessels may arise from intercostal or bronchial arteries. Strategies include: -Early identification and control of the vascular pedicle; -Avoidance of excessive tumor manipulation; -Use of energy sealing devices

6.5.2 Tumor Rupture: Rupture risks peritoneal or pleural dissemination of malignant cells. To prevent this:-Tumors should be handled gently; -En bloc removal without fragmentation is imperative; -Protective specimen retrieval bags should be used, particularly during VATS

6.5.3 Avoiding Parenchymal Injury: For fissural or parenchymal tumors, careful dissection is essential to preserve uninvolved lung tissue while maintaining clear margins.

6.6 Postoperative Management

6.6.1 Immediate Postoperative Care

Most patients recover uneventfully following complete resection. The chest tube is typically removed once air leak resolves and drainage is minimal. Early ambulation and respiratory physiotherapy are essential.

6.6.2 Complications

Reported postoperative complications include atelectasis, pneumonia, prolonged air leak, and, rarely, hemothorax. Large tumors and extensive resections increase complication risk (Ajouz et al., 2023).

6.7 Role of Adjuvant Therapy

There is **no established role for routine adjuvant chemotherapy or radiotherapy** following complete resection of benign or borderline SFTs. Evidence supporting adjuvant therapy remains limited, inconsistent, and largely based on case reports.

Radiotherapy may be considered in:-Positive surgical margins (R1/R2); -High-risk malignant tumors based on Demicco risk model¸-Recurrent disease not amenable to resection

Chemotherapy has limited efficacy given the tumor's mesenchymal origin. Targeted therapies remain investigational.

6.8 Follow-Up and Surveillance

Long-term surveillance is mandatory for **all** patients, regardless of tumor grade, due to the unpredictable risk of late recurrence—even 10 to 20 years postoperatively (Abu Arab, 2012).

Recommended follow-up includes: -CT scans every 6–12 months for the first 5 years; -Annual imaging thereafter; -Closer surveillance for malignant tumors (every 3–6 months initially)

Recurrence is more common in malignant, large (>10 cm), sessile tumors and in cases with incomplete resection. When recurrence occurs, repeat surgical resection remains the preferred treatment when feasible.

Summary of Surgical Principles

- Complete en bloc resection with negative margins is the definitive and most effective treatment.
- Surgical approach is dictated by tumor size, vascularity, attachment, and location.
- Thoracotomy is preferred for malignant, large, or hypervascular tumors; VATS is appropriate for small pedunculated lesions.
- Intrapulmonary SFTs often require anatomical lung resection.

- Adjuvant therapy is not routinely indicated but may be considered selectively.
- Lifelong surveillance is recommended due to the risk of late recurrence

7. Discussion

Solitary fibrous tumors (SFTs) of the pleura and lung parenchyma represent a diagnostically challenging and clinically heterogeneous group of mesenchymal neoplasms whose biological behavior ranges from indolent to overtly malignant. Solitary fibrous tumors (SFTs) of the pleura and lung parenchyma constitute a clinically heterogeneous group of mesenchymal neoplasms with variable malignant potential. Once considered benign and visceral pleura–originating, evidence now highlights broader anatomical distribution, unpredictable behavior, and occasional malignancy (Abu Arab, 2012; Cardillo et al., 2012). This discussion integrates radiologic, histopathologic, and clinical perspectives to contextualize management principles.

7.1 Clinical Heterogeneity and Diagnostic Challenges

SFTs exhibit substantial clinical heterogeneity; 54–70% of patients are asymptomatic, with tumors often detected incidentally (Enon et al., 2012; Abu Arab, 2012). Symptomatic tumors generally correlate with larger size or parietal pleural origin, presenting with dyspnea, chest pain, or systemic features. Rare paraneoplastic syndromes, such as Doege–Potter syndrome, indicate high tumor burden and resolve post-resection (Cardillo et al., 2012).

Preoperative diagnosis is limited: contrast-enhanced CT is standard but cannot reliably differentiate benign from malignant

tumors; MRI may aid soft tissue evaluation. Histopathology and immunohistochemistry, particularly STAT6 nuclear expression linked to NAB2–STAT6 fusion, remain definitive (Sagawa et al., 2007). Needle biopsy is controversial due to sampling limitations and potential complications.

7.2 Malignant Potential and Aggressive Predictors

Malignant potential is variable and difficult to predict. Classical histologic criteria—hypercellularity, nuclear atypia, mitotic activity (>4/10 HPF), necrosis, and infiltrative margins—correlate with recurrence risk (Khouzam & Khouzam, 2022). The Demicco model incorporates age, tumor size, and mitotic rate for metastasis risk stratification (Demicco et al, 2012). Even histologically benign lesions may recur or metastasize, necessitating long-term follow-up (Abu Arab, 2012).

Tumor origin influences malignancy: parietal pleura, fissure-based, or intrapulmonary tumors show higher aggressive potential than pedunculated visceral pleural lesions (Enon et al., 2012; Alghamdi et al., 2020).

7.3 Radiologic–Pathologic Correlation

Radiologic features such as size >8–10 cm, heterogeneous enhancement, central necrosis, and broad pleural attachment often suggest malignancy (Cardillo et al., 2012; Abu Arab, 2012). Correlating imaging with histopathology enhances surgical planning; for instance, “staghorn” vascular patterns on pathology often correspond to intense contrast enhancement, indicating hypervascularity and bleeding risk (Sagawa et al., 2007; Sakurai et al., 2008)

7.4 Surgical Resection

Complete en bloc resection with negative margins remains the main determinant of long-term survival. Even malignant SFTs have favorable outcomes with complete resection, whereas incomplete excision increases recurrence risk (Ajouz et al., 2023; Cardillo et al., 2012). Thoracotomy is preferred for large or hypervascular tumors; VATS is suitable for small, pedunculated lesions. Anatomical resections (lobectomy, segmentectomy) are often needed for intrapulmonary tumors. Extended resections are indicated for chest wall, diaphragmatic, or mediastinal involvement. Early detection via structured follow-up facilitates salvage surgery for recurrences (Abu Arab, 2012).

7.5 Adjuvant Therapies

Routine adjuvant radiotherapy or chemotherapy is not indicated after complete resection of benign or low-risk SFTs. Radiotherapy may be considered for positive margins or high-risk tumors, while chemotherapy has limited efficacy. Emerging molecular-targeted therapies, particularly anti-angiogenic agents, show potential but require further validation.

7.6 Long-Term Follow-Up

Lifelong follow-up is essential due to potential late recurrence *(>15–20 years post-treatment*) (Abu Arab, 2012).

Recommended surveillance includes: CT every 6–12 months for 5 years, Annual CT thereafter, Closer monitoring for high-risk or malignant tumors

Recurrences can be local or metastatic (lung, liver, bone), with repeat resection preferred when feasible.

7.7 Integration of Case Reports

Case reports highlight rare presentations such as intrapulmonary SFTs and unusual imaging features (Sakurai et al., 2008; Sagawa et al., 2007; Alghamdi et al., 2020).

Key clinical lessons:

- Intrapulmonary SFTs may mimic lung carcinoma
- Hypervascularity complicates biopsy and surgery
- Large tumors can still be resected successfully
- STAT6 immunohistochemistry is crucial for diagnosis

These observations are consistent across diverse settings, supporting their relevance in clinical practice.

7.8 Summary of Key Considerations

The collective literature paints a picture of solitary fibrous tumors as rare but clinically consequential thoracic neoplasms with the following defining characteristics:

- Marked clinical variability, from asymptomatic incidentalomas to large, symptomatic masses with systemic manifestations.
- Unpredictable malignant potential, requiring careful integration of histologic, radiologic, and molecular findings.
- Surgical resection as the definitive therapy, with complete removal being the single most important determinant of long-term survival.

- Limited role for adjuvant therapy, except in selected high-risk or incompletely resected cases.
    - Need for lifelong follow-up, due to the risk of late recurrence even after curative treatment.

These principles form the foundation for contemporary management strategies and guide clinical decision-making in the context of both common and rare presentations—including the malignant intrapulmonary case discussed later in this chapter.

The present case illustrates several diagnostic and therapeutic principles characteristic of malignant solitary fibrous tumors (SFTs) of the pleura with pulmonary parenchymal extension. Although SFTs are generally slow-growing neoplasms, malignant variants may exhibit more aggressive behavior, including direct invasion into the lung parenchyma, mediastinum, or diaphragm.

In this case, radiological evaluation revealed a pleural-based lobulated mass in the lower lobe measuring 10.8 × 5.2 cm, characterized by heterogeneous enhancement, minimal calcifications, and irregular margins, with no evidence of extrapleural or chest wall invasion; however, focal radiologic extension toward the fissure and the adjacent upper-lobe parenchyma was observed (Figures 1 and 2).

These features are consistent with previously reported imaging characteristics of malignant SFTs.

Histopathological examination confirmed a 13-cm, well-circumscribed but focally infiltrative spindle-cell neoplasm composed of hypercellular areas, abundant branching (“staghorn-like”) capillary vessels, and focal calcification. Additional malignant features included cytologic atypia, low-to-intermediate

mitotic activity, and focal necrosis. Immunohistochemistry demonstrated diffuse positivity for vimentin, Bcl-2, CD34, strong nuclear STAT6, and S100, EMA, CK7, calretinin negativity confirming the diagnosis of solitary fibrous tumor (Figures 3, 4). These findings align with the pathological criteria described by England et al. and subsequent classifications distinguishing benign from malignant SFTs

Figure 1 Preoperative and postoperative Chest X ray

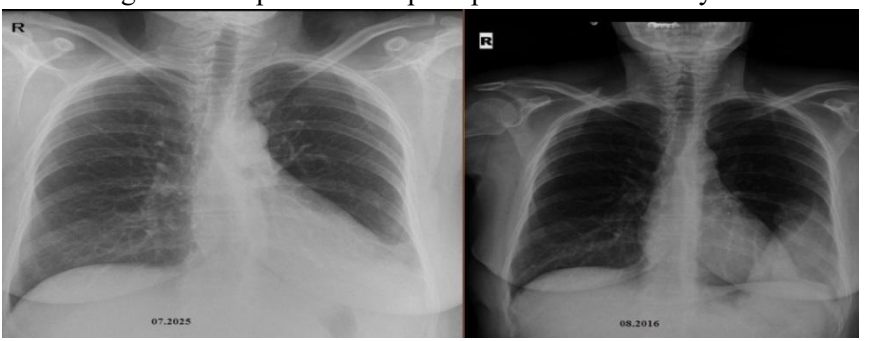


Figure 2:Preoperative and postoperative Thorax CT

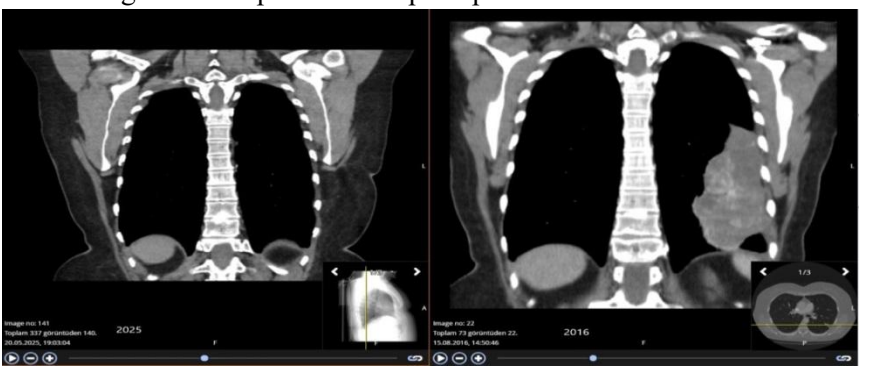
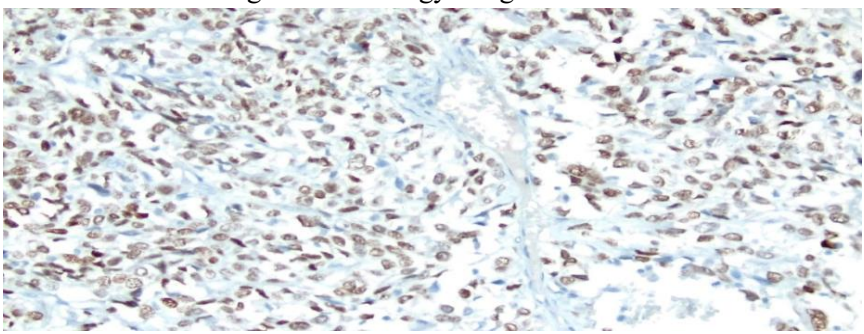
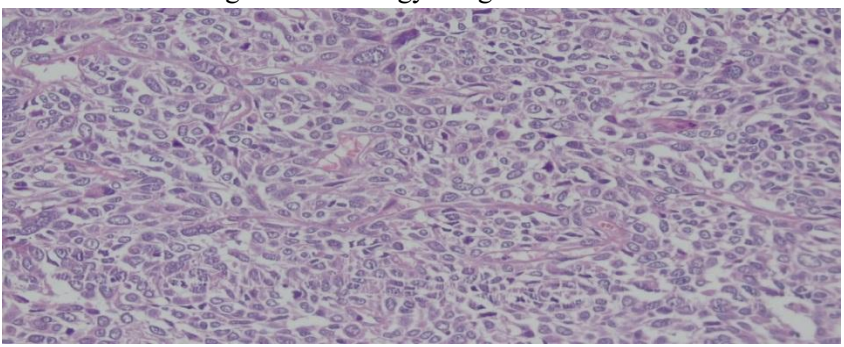


Figure 3:Pathology image 1-STAT6



Source: Widespread STAT6 staining is observed in spindle cells

Figure 4:Pathology image 2- HEx200



Source: randomly distributed, moderately pleomorphism, oval spinal cells,

Integration of Clinical Presentation

The 52 years old female patient presented with pruritus, exertional dyspnea and atypical chest discomfort rather than the classic paraneoplastic syndromes. This is consistent with epidemiological data indicating that only a minority of pleural SFTs manifest systemic symptoms. In this case, neither Deoge–Potter syndrome nor Marie–Bamberger syndrome was present, which underscores the heterogeneity of clinical manifestations and the fact that paraneoplastic syndromes correlate more strongly with very large or metabolically active tumors. The absence of such

findings does not exclude malignant potential, emphasizing the need for comprehensive radiological and pathological assessment.

Radiological–Pathological Discordance and Diagnostic Challenges

A notable aspect of the case was the partial discordance between preoperative imaging and the definitive pathological findings regarding tumor invasiveness. While contrast-enhanced CT demonstrated a lobulated pleural-based mass in the lower lobe measuring 10.8 × 5.2 cm with suspected parenchymal infiltration, intraoperative macroscopic assessment confirmed focal intraparenchymal invasion-firm adherence to the visceral pleura, without involvement of the parietal pleura. This mismatch underscores the well-recognized limitations of CT alone in distinguishing benign from malignant SFTs, particularly in large tumors that may exhibit expansive growth while maintaining deceptively smooth or well-circumscribed margins. Advanced imaging modalities such as MRI and PET-CT—both of which can provide additional information on soft-tissue planes and metabolic activity—were not performed in this patient. The absence of PET-CT is notable, as FDG uptake in SFTs is highly variable and may correlate with malignant histological features, although not reliably enough to guide management independently. Similarly, MRI might have offered superior delineation of pleural versus parenchymal invasion; however, given the tumor's size and CT characteristics, surgical resection remained the definitive diagnostic and therapeutic approach. The pathological confirmation of aggressive features in this case aligns with prior reports highlighting the inherent diagnostic challenges and the limited predictive value of imaging alone in assessing malignant potential

Surgical Decision-Making in the Case Context

Given the radiological suspicion of malignancy and the evidence of parenchymal invasion, the surgical team opted for an anatomic resection rather than simple enucleation or wedge excision. The operative plan, including anatomical lower lobectomy and wedge resection to upper lob, was consistent with contemporary guidelines emphasizing complete resection with clear microscopic margins.

Intraoperative findings confirmed the necessity of this approach: the mass demonstrated focal infiltration into adjacent lung tissue and a broad-based pleural attachment. These features are typical of malignant or high-risk SFTs and correlate with increased recurrence potential. Anatomic lung resection ensured oncologic clearance while preserving pulmonary function—a central objective in thoracic surgery for pleural tumors.

Pathology Correlation and Prognostic Considerations

The final pathological diagnosis—13x8x5 cm malignant solitary fibrous tumor with low mitotic index and high pulmonary invasion,—further validated the aggressive surgical approach. The presence of nuclear atypia, necrosis, and infiltrative growth pattern placed the tumor within the high-risk category of the WHO classification. Parenchymal invasion is recognized as an independent adverse prognostic factor and correlates strongly with recurrence in long-term follow-up studies.

In this context, prognosis will depend largely on:

- completeness of resection (R0 achieved in this case),
- tumor size (>13cm in this case), (large lesions carry increased risk),
- Ki-67 proliferative index (Ki-67 was 30% in this case)
- mitotic count,(Low-mitotic activity(2/10 HPF) in this case)
- presence of necrosis or dedifferentiation,
- and STAT6 nuclear positivity, which confirms diagnosis but does not directly predict aggressiveness.

Given these factors, this case—followed for nine years without recurrence—illustrates the necessity for long-term surveillance in patients with SFT. Ongoing follow-up should include CT imaging every 6–12 months for at least ten years, as recurrences in malignant SFTs may arise even after a decade, reflecting the unpredictable natural history of the disease.

Relevance of the Case to the Literature

This case contributes to the existing body of evidence in several ways:

- It demonstrates that malignant pleural SFTs may exhibit subtle or nonspecific symptoms, making early diagnosis difficult.
- It highlights the crucial role of multimodal imaging, particularly MRI, in evaluating pleural-based masses and detecting invasion.
- It supports the paradigm that anatomical lung resection should be performed when parenchymal invasion is identified or strongly suspected.

- It reinforces the need for lifelong follow-up due to the risk of late recurrence.
- It emphasizes that paraneoplastic syndromes are not universal, and their absence does not negate malignant potential.

Overall, the case aligns with published data emphasizing the variability of malignant SFTs and underscores the importance of a multidisciplinary approach that integrates radiology, surgery, pathology, and oncology.

8. Conclusion

Malignant solitary fibrous tumors of the pleura represent a rare but clinically significant subset of thoracic neoplasms characterized by unpredictable behavior, potential for local invasion, and risk of recurrence. Although many SFTs remain asymptomatic, malignant variants may manifest with rapid growth, mass effect, or parenchymal infiltration, as demonstrated in the presented case. Accurate diagnosis requires a combination of imaging, histopathology, and immunohistochemical confirmation—particularly STAT6 nuclear staining.

Complete surgical resection remains the cornerstone of effective management. In cases where parenchymal invasion is present or suspected, anatomic lung resection offers superior oncologic control compared to limited excision. Pathological features such as mitotic rate, cellular atypia, necrosis, and infiltrative margins provide essential prognostic information and guide postoperative surveillance strategies.

The presented case reinforces several key principles:

- malignant SFTs may lack paraneoplastic manifestations,
- imaging may underestimate the extent of invasion,
- complete resection with negative margins is vital,
- long-term follow-up is mandatory due to late recurrence potential.

In conclusion, the management of malignant pleural SFTs requires a highly individualized, evidence-based approach. The integration of clinical findings, advanced imaging, meticulous surgical technique, and thorough pathological evaluation is essential for optimizing outcomes in this rare but challenging disease.

Referances

Abu Arab, W. (2012). Solitary fibrous tumours of the pleura. European Journal of Cardio-Thoracic Surgery, 41, 587–597. doi:10.1093/ejcts/ezr009

Ajouz, H., Sohail, A. H., Hashmi, H., Martinez Aguilar, M., Daoui, S., Tembelis, M., Cerfolio, R. J. (2023). Surgical considerations in the resection of solitary fibrous tumors of the pleura. Journal of Cardiothoracic Surgery, 18(1), 79. https://doi.org/10.1186/s13019-023-02168-7

Alghamdi, Z. M., Othman, S. A., Al-Yousef, M. J., & AlFadel, B. Z. (2020). Intrapulmonary location of benign solitary fibrous tumor. Annals of Thoracic Medicine, 15, 98–101.

Cardillo, G., Lococo, F., Carleo, F., & Martelli, M. (2012). Solitary fibrous tumors of the pleura. Current Opinion in Pulmonary Medicine, 18(4), 339–346. doi:10.1097/MCP.0b013e328352f696

Dörr, N. M., Krüger, M., Möller, M., Zinne, N., Toennies, M., Schega, O., … Biancosino, C. (2024). Solitary fibrous tumours of the pleura: Do we need a different perspective on malignancy? European Journal of Cardio-Thoracic Surgery, 65, ezae096. https://doi.org/10.1093/ejcts/ezae096 OUP Academic

Enon, S., Kilic, D., Yuksel, C., Kayi Cangir, A., Percinel, S., Dizbay Sak, S., Gungor, A., Kavukcu, S., & Okten, I. (2012). Benign localized fibrous tumor of the pleura: Report of 25 new cases. The Thoracic and Cardiovascular Surgeon, 60(7), 468–473. doi:10.1055/s-0031-1295519

Sakurai, H., Tanaka, W., Kaji, M., Yamazaki, K., & Suemasu, K. (2008). Intrapulmonary localized fibrous tumor of the

lung: A very unusual presentation. Annals of Thoracic Surgery, 86(4), 1360–1362.doi:10.1016/j.athoracsur.2008.04.014 cir.nii.ac.jp

Sagawa, M., Ueda, Y., Matsubara, F., Sakuma, H., Yoshimitsu, Y., Aikawa, H., Usuda, K., Minato, H., & Sakuma, T. (2007). Intrapulmonary solitary fibrous tumor diagnosed by immunohistochemical and genetic approaches: Report of a case. Surgery Today, 37(5), 423–425. doi:10.1007/s00595-006-3422-3

Serkan Enon, D. K., Yuksel, C., Kayi Cangir, A., Percinel, S., Dizbay Sak, S., Gungor, A., Kavukcu, S., & Okten, I. (2012). Benign localized fibrous tumor of the pleura: Report of 25 new cases. Thoracic and Cardiovascular Surgeon, 60(7), 468–473. doi:10.1055/s-0031-1295519

England, D. M., Hochholzer, L., & McCarthy, M. J. (1989). Localised benign and malignant fibrous tumours of the pleura: A clinicopathologic review of 223 cases. American Journal of Surgical Pathology, 13(8), 640–658. https://doi.org/10.1097/00000478-198908000-00003,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