Solitary round pulmonary lesions in the pediatric population: a pictorial review

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Abstract
The purpose of this current pictorial review is to define the solitary round pulmonary lesion (SRPL), to familiarize with its prevalence in the pediatric population, and, moreover, to educate radiologists on its vast differential diagnosis and imaging manifestations. Furthermore, by highlighting valuable clues, it intends to assist radiologists efficiently partake in its diagnosis, work-up, and follow-up in order to narrow down the differential diagnosis by working alongside the clinician and combining clinical information, lab results, and radiological findings.

Keywords
Pediatric radiology, solitary round pulmonary lesions, computed tomography, chest imaging, chest X ray, review

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Introduction
A solitary round pulmonary lesion (SRPL) is a round or oval lesion in the lung parenchyma that can be seen on chest imaging. It is a rare finding in the pediatric population and, as a result, when stumbled upon, even an experienced radiologist might find it challenging to narrow down its extensive differential diagnosis. The numerous etiologies of a SRPL could optimally be categorized as: A = congenital; B = infectious; C = inflammatory; D = neoplastic; E = vascular; and F = others (Table 1).

In this pictorial review, we attempt to offer valuable and conclusive clues in order to assist, in the best and shortest way possible, in the approach of this vast entity.

Clinical entities

A. Congenital

Bronchopulmonary sequestration (BPS). Rare congenital malformation of the lower respiratory tract which consists of a non-functioning lung tissue mass (SRPL) (Fig. 1c) that lacks normal communication with the tracheobronchial tree and receives its arterial blood supply from the systemic circulation. They are classified anatomically as intralobar or extralobar (1,2). The clinical presentation of BPS is variable and depends upon the type, size, and location of the lesion. Extralobar BPSs are usually seen in newborns presenting with respiratory distress, cyanosis, or infection, while intralobar BPSs present in late childhood with recurrent pulmonary infections (1–4).

Computed tomography angiography (CTA) or magnetic resonance angiography (MRA) offer the ability to simultaneously visualize the arterial supply, possible venous drainage, and parenchymal involvement

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However, most cases are identified on routine prenatal ultrasound examination (Fig. 1a). The detection of a systemic artery from the aorta to the fetal lung lesion with color flow Doppler ultrasound is considered a pathognomonic feature (Fig. 1b) (4).

Their differential diagnosis includes lesions, such as congenital pulmonary airway malformations and bronchogenic cysts.

**Congenital pulmonary airway malformation (CPAM).** In contrast to BPS, CPAMs are connected to the tracheobronchial tree and are supplied from the pulmonary circulation (1,4). However, hybrid lesions with histologic characteristics of CPAM and a systemic arterial supply have been reported (1,4).

There are five types of CPAM according to the location of the developmental site of malformation: tracheobronchial (type 0) consists of small cysts; bronchial/bronchiolar (type 1) is the most common and consists of cysts measuring 3–10 cm; bronchiolar (type 2) is composed of smaller cysts (0.5–2 cm) and solid tissue; distal acinar (type 3) is composed of cysts that are so small the mass appears to be solid; and finally the bronchiolar/alveolar (type 4) contains cysts as large as 10 cm (4,5). They are usually detected in neonates presenting with respiratory distress and rarely in older children presenting with recurrent infection (3). Type 0 CPAM is considered lethal (1,4).

Radiographic features will vary depending on the type (Fig. 2). CTA is able to identify systemic arterial supply if present. Diagnosis is usually made on antenatal ultrasound. (4)

Type 4 CPAM has been linked to malignancy, specifically pleuropulmonary blastoma (PPB), which will be analyzed later on (4). Numerous studies have suggested that PPB and CPAM can be indistinguishable clinically and radiographically. Overlap on pathology between type 4 CPAM and type 1 PPB makes it even more challenging to tell them apart. Features predictive for PPB are family history of PPB or related tumors, symptoms such as pneumothorax, bilateral or multisegment involvement, the presence of a complex cyst, and a germline mutation in the DICER1 gene (found in two-thirds of PPB). Demonstration of pulmonary circulation or rarely systemic arterial supply will favor CPAM.

**Bronchogenic cyst.** Rare congenital malformation of the bronchial tree (with which it usually does not communicate) can occur in the mediastinum or be
intrapulmonary. Usually they are asymptomatic and found incidentally on chest imaging.

Typically, they appear as a well-circumscribed mass (SRPL) of variable attenuation (CT) or intensity (MR) depending on the amount of internal proteinaceous content (3,6). A fluid-fluid level can also be apparent, attributed to layering of variable fluid content. There is no solid contrast enhancement.

**Congenital bronchial atresia (CBA).** Results from anomalous development of a segmental or subsegmental bronchus with local obliteration or stenosis near its origin and subsequent failure to sustain communication with the central airway (3,7). Distal to the obstruction the bronchus is patent and dilated containing impacted mucus while peripherally the lung is over-aerated due to collateral air drift through the pores of Kohn (6,7). They are rarely symptomatic and commonly revealed on CXR performed for other indications (7).

Radiographically, an opaque round or club-like mucocele surrounded by over-inflated lung can be seen (3,6).

**Congenital lobar emphysema (CLE).** CLE is a condition characterized by hyperinflation of a lobe believed to be caused by bronchial obstruction due to primary cartilage deficiency or dysplasia (3,7). The majority of patients present with respiratory distress usually during the first six months of life (7).

Over-inflation of the affected lobe, herniation to the contralateral side, and compressive atelectasis of other lobes are the most common imaging findings (3,6). Postnatally, impaired clearance of retained lung fluid may increase the density of the affected lobe until it is resorbed (6,7).

**B. Infectious**

**Round pneumonia.** The commonest etiology of a SRPL is round pneumonia, which mostly occurs in patients aged < 8 years. Poor development of collateral airways (channels of Lambert, pores of Kohn) by this age limits air drifting between the parenchymal subsegments hence confining the infection in a round morphology. After this age, there should be increased suspicion of other underlying causes (1,8). The commonest causative microorganism is *Streptococcus pneumoniae*. The patient usually presents with cough and fever while lab results may show elevated inflammatory markers. Instead of chest symptoms, patients may rarely present with abdominal pain (8).

A soft-tissue density solitary lesion, which does not contain an air-fluid level can be seen on a chest X-ray, most often posteriorly and in lower lobes (Fig. 3).

CT is not indicated to evaluate for other potential causes (1). The patient should be treated with antibiotics and a repeat chest X-ray should be obtained in several weeks. In 95% of the patients, it will resolve on follow-up imaging rather than progressing to lobar pneumonia (8).

**Lung abscess.** Lung abscesses are uncommon in otherwise healthy children. They most frequently occur in children with underlying immunodeficiency or complex
medical disorders. Destruction of lung parenchyma from pulmonary infection may lead to suppurative necrosis, which then creates the lung abscess. They are most commonly caused by *S. pneumoniae*, *Staphylococcus aureus*, and oral bacteria, while in patients with pre-existing medical disorders the most common pathogen is *Pseudomonas aeruginosa* and fungi. Symptoms include fever, cough, and shortness of breath.

Chest imaging can show a round cavity that may contain an air-fluid level (Fig. 4), while wall enhancement can be demonstrated on contrast-enhanced CT (1,7).

**Granuloma – tuberculosis.** Most children with tuberculosis (TB) have primary disease, a clinical infection taking place after the first exposure to the organism, which is radiologically distinct from post-primary TB, the most common form occurring in adults. The diagnosis of primary TB in childhood is difficult to establish clinically due to the lack of physical symptoms. Up to 60% of children with pulmonary TB are asymptomatic and found solely through contact investigation. Because of the narrower diameter of their airways, younger children are more likely to have respiratory symptoms, which include cough and wheezing or rales over the involved region.

The radiologic hallmark is lymphadenopathy, typically involving the right hilar and paratracheal nodes (7). Primary disease presents as an area of consolidation or as a well-defined tuberculoma (SRPL), which accompanied with the previously mentioned lymphadenopathy, forms the Ranke complex (Fig. 5). On contrast-enhanced CT, involved lymph nodes usually demonstrate low-attenuation centers, representing areas of necrosis, and peripheral rim enhancement (7). Calcifications may develop over time at areas of previously formed tuberculomas.

Diagnosis in childhood is difficult and usually made on the basis of epidemiologic data. In the absence of a positive culture, the strongest evidence is recent exposure to an adult with active disease. Indirect diagnostic techniques such as the tuberculin skin test and chest X-ray provide supportive information.

**Granuloma – fungal.** Aspergillosis is uncommon in otherwise healthy children. It usually occurs in
immunocompromised children or children with underlying conditions such as asthma or cystic fibrosis (8). Clinical presentation is similar to other chest infections. Suspicion should be raised in immunocompromised and severely neutropenic patients as well as in patients with hematologic malignancies.

Imaging findings include a consolidation with a “halo” sign and an “air crescent” sign which is seen rarely when a mycetoma forms in a preexisting cavity as a superinfection (Fig. 6) (7).

**Hydatid disease.** Hydatid disease is a parasitic infection caused by *Echinococcus granulosus* or less often by *E. alveolaris* (multilocularis), which is usually transmitted to humans by contact with a definitive host (most often a dog, or other carnivores) or by consuming contaminated water or vegetables. The most common site of infection in children are the lungs but it can affect multiple other organs (such as the liver), sometimes synchronously. Although patients are usually diagnosed during adulthood, the parasite is typically acquired in childhood, but only 10–25% of patients are detected during this age, since most cysts are asymptomatic and found incidentally on chest X-ray. The clinical manifestations of cyst rupture include coughing, hemoptysis, and expectoration of cyst contents (i.e. scolices).

On chest X-ray, uncomplicated cysts are indistinguishable from other round lesions, such as abscesses or congenital cysts. However, the presence of imaging findings, such as the “water-lily sign” in ruptured cysts (produced by the collapsed hydatid membranes floating on top of the residual hydatid fluid), will aid in the diagnosis (Fig. 7) (3). In contrast to other organs, hydatid disease of the lung will rarely have

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**Fig. 5.** Ranke complex. Mildly calcified right hilar lymph node (arrowhead) and tuberculoma (arrow) manifesting as a solitary round pulmonary lesion in a child with TB.

**Fig. 6.** Aspergillus infection. (a) Chest X-ray showcases a SRPL in a patient with leukemia (arrow). (b) Further evaluation with CT demonstrates cavitation of the lesion and a ground glass halo sign (arrow). (c) Different immunocompromised patient with fungal infection showcasing the “air crescent sign” (arrowheads).

**Fig. 7.** Echinococcus cyst. Chest X-ray demonstrates a large round pulmonary lesion with an air-fluid level which also displays an irregular wavy nature, known as the “water-lily sign” (arrow).
calcifications (1). CT and MR imaging (MRI) can demonstrate the presence of daughter cysts and also show intact, ruptured, and secondarily infected cysts. The later might also display ring enhancement. Correlation of serologic and imaging findings will aid in the diagnosis.

C. Inflammatory

Pulmonary inflammatory pseudotumors. Inflammatory pseudotumor of the lung is a non-neoplastic pulmonary mass lesion of unknown etiology, which can mimic pulmonary malignancy. The majority of investigators regard this entity as a variant of an inflammatory repair process that is related to acute and chronic inflammation. Clinically, it tends to be silent. Symptoms may include coughing, fever, sputum, pain, hemoptysis, shortness of breath, and clubbing. Laboratory data are usually non-specific.

Usually it is detected on routine chest X-ray as a solitary, well-circumscribed round or oval mass (Fig. 8a). Other possible imaging characteristics (Figs. 8b and 9) include calcification, cavitation, heterogeneous attenuation with variable patterns of contrast enhancement, atelectasis, and pleuritic fluid (9, 10).

D. Neoplastic

Primary – benign

Hamartoma. Although rare in children, hamartoma is the second most frequent benign tumor. They are usually clinically silent and detected on routine chest X-ray.

On imaging, it can appear as a sharply demarcated round or lobulated structure, commonly <3 cm in diameter. Radiological diagnosis is typically based on the detection of “popcorn-like” calcifications and/or fat (Fig. 10) (10–12). MR can be helpful when neither calcification nor fat is demonstrated on CT.

Primary – malignant

The possibility of an incidental SRPL in a child representing a primary lung cancer is remarkably small; however, it should not automatically be assumed as benign (13).

Carcinoid. The most common primary malignant lung tumors are lesions such as bronchial carcinoids (BCs). BCs are rare, well-differentiated malignant neuroendocrine tumors arising from Kulchitsky (neuroendocrine) cell or APUD system cells (14). Diagnosis can be delayed owing to low clinical suspicion and the different ways in which BCs can present (15). Symptoms are aspecific, but their recurrence in a child with obstructive pneumonia localized in the same lobe should alert the physician for further evaluation (12, 14, 15).

On imaging, it appears as a well-defined ovoid lesion measuring 2–4 cm, often containing calcifications and usually located centrally involving the right middle lobe or lingula. Atelectasis and mucoid impaction related to endobronchial obstruction can also be seen. Contrast administration can further assist by detecting enhancement, mediastinal lymph node involvement, and/or metastases (16). Sensitivity of MR is not established in the literature (16). Uptake of I123 MIBG on nuclear scans is a very helpful finding (Fig. 11) since it can be seen in tumors originating from neural crest cells (neuroblastoma, neuroganglioma, carcinoid) (16, 17).
Neuroblastoma (NB). NBs are tumors of primordial neural crest cell origin that can be located anywhere along the sympathetic nervous system (17). Most cases are located in the adrenals (40%) while 19% are located in the chest and arise from the posterior mediastinum in which case extension into the lung parenchyma can mimic a SRPL (Fig. 12) (8,17). In young children aged <2 years, a posterior mediastinal mass is NB until proven otherwise. In addition, 50–60% of all NBs present with metastases (17) which can also present as a SRPL when located in the lung parenchyma. Clinical presentation can be unusual with neurologic and/or respiratory symptoms, depending on size and location. Rare (5%) but distinctive presentations include transverse myelopathy owing to cord compression (17).

When originating from the mediastinum, the classic “dumbbell” appearance (extension of the lesion into...
the neural foramen) is exquisitely demonstrated on MRI, which is the imaging modality of choice (as for all posterior mediastinal lesions).

The vast majority of patients with a NB show elevated levels of catecholamine in the serum and urine; thus, 24 h urine collection is an important diagnostic and follow-up test (17).

Pleuropulmonary blastoma (PPB). PPB is a very rare, highly aggressive, and malignant tumor, originating from

Fig. 11. Carcinoid tumor. A 13-year-old boy presenting with recurrent episodes of right lower lobe pneumonia. (a) Chest X-ray shows a solitary round pulmonary lesion close to the right hilum (arrowhead) with post-obstructive pneumonitis. (b, c) CT shows a homogeneously enhancing lesion of the right lower lobe which furthermore demonstrates endobronchial spread (arrow). (d) Nuclear scan shows a focus of increased uptake of I123 MIBG in the right lower lobe (arrow). Surgical excision revealed a carcinoid tumor.

Fig. 12. Neuroblastoma. After the detection of this SRPL on a chest X-ray in a febrile child (arrowheads), an ultrasound of the chest was performed which described the presence of a well-demarcated paravertebral mass with calcifications which had a clear transition to the healthy lung parenchyma. There was no co-existing pleural effusion.
either the lungs or pleura and occurring mainly in children aged < 6 years. They are classified into three types: type I PPB is completely cystic, henceforth is often mistaken with other non-neoplastic cystic lesions such as CPAMs; type II PPB is a mixed lesion with both cystic and solid components; and type III occurs as an entirely solid mass (5,11,12). The common presenting symptoms are dry cough, fever, dyspnea, tachypnea, chest pain, fatigue, pneumothorax, and respiratory infections that do not resolve with antibiotic therapy. They are difficult to diagnose preoperatively since the tumor has no characteristic imaging findings. May appear as a cystic lung lesion or cause a partial or complete opacification of the hemithorax with mediastinal deviation to the contralateral side (5,11).

Surgical resection is recommended when such malignancy is suspected. Adjuvant chemotherapy also has an important role (5). Suspicion for PPB must be high in any child presenting with a cystic lung lesion and specifically in one with poor weight gain.

Secondary – malignant

Solitary lung metastasis. Metastatic disease to the lung is far more common than a primary lung tumor, with Wilms tumor being the most common primary lesion in infants and children. Other common primaries include rhabdomyosarcoma, osteosarcoma, and Ewing sarcoma (14). Clinical suspicion should be raised in patients with a known primary malignancy but may also be stumbled upon during routine imaging.

Atypical radiographic features include consolidation, cavitation, calcification, hemorrhage, and secondary pneumothorax.

E. Vascular

Vascular lesions can arise from the aorta, systemic veins, pulmonary artery or vein, or a combination of the two (i.e. pulmonary arteriovenous malformation [PAVM]).

Pulmonary arteriovenous malformation. A PAVM is a rare vascular anomaly of the lung, in which abnormally dilated vessels provide a right-to-left shunt between the pulmonary artery and vein that may appear as a SRPL (Fig. 13a) (18,19). Despite most patients being asymptomatic, affected children may present with cyanosis, dyspnea, hemoptysis, clubbing as well as polycythemia (14,19).

CTA, MRA, and pulmonary angiography will demonstrate enhancement of the feeding artery, the aneurysmal part and the draining vein (Fig. 13b) (19). Pulmonary angiography is the method of choice when percutaneous treatment is also planned.
Fig. 15. Extraparenchymal lesion (neurofibroma). (a) Chest X-ray of a 16-year-old girl with a history of neurofibromatosis shows an ovoid, soft-tissue density in the left hemithorax, which appears to be extraparenchymal and originating from the chest wall due to its clear, well-demarcated boundaries and obtuse angles. (b) Contrast-enhanced CT demonstrates inhomogeneous enhancement of the lesion, which appears to have a broad base to the chest wall. (c) On T2-weighted MRI, the lesion displays inhomogeneous high signal intensity, more evident at its periphery. In addition, it appears to elevate the adjacent pleura with associated pleural fluid accumulation in the upper lobe (arrowhead).

| Table 1. Conclusive table showcasing the differential diagnosis of a SRPL in the pediatric population, as well as helpful clues for diagnosis. |
|---|
| **A. Congenital** |
| Bronchopulmonary sequestration | Round pneumonia |
| Detection of the non-functioning lung tissue mass, systemic arterial supply, and possibly venous drainage on CTA/MRA |
| Congenital pulmonary airway malformation | Abscess |
| Connected to the tracheobronchial tree and supplied from pulmonary circulation CTA is able to identify systemic arterial supply if present |
| Bronchogenic cyst | Primary benign Hamartoma |
| Typically appears as a well circumscribed mass of variable attenuation or intensity depending on the amount of internal proteinaceous content Fluid-fluid level may be apparent attributed to layering of variable fluid content No solid contrast enhancement |
| **B. Infectious** |
| Round pneumonia | Lobar pneumonia |
| Aged 5–8 years Cough, fever, ↑WBC/CRP Round pulmonary density |
| Abscess | Immunocompromised patients |
| Cough, fever Round cavity that may contain an air-fluid level and showcase wall enhancement on contrast-enhanced CT |
| **C. Neoplastic** |
| Primary benign Hamartoma | Lymphangioleiomyomatosis |
| Clinically silent, healthy patients, unremarkable laboratory findings Detection of “popcorn-like” calcifications and/or fat |
| Primary malignant Carcinoid | Lymphangiomyoma |
| Aspecific symptoms Recurrent episodes of obstructive pneumonia localized in the same lobe Uptake of 1113 MIBG on nuclear scans |
| Neuroblastoma | Intracranial |
| In children aged < 2 years, a posterior mediastinal mass is neuroblastoma until proven otherwise Unusual presentation with neurological and/or respiratory symptoms “Dumbbell” appearance (extension of the lesion into the neural foramen) on MRI ↑ levels catecholamine May manifest as a SRPL if metastatic to the lung from primary NB elsewhere in the body or from direct extension from mediastinum |
| **D. Neoplastic** |
| Arteriovenous malformation | Hemangioma |
| Pulmonary angiography/CTA/MRA will demonstrate enhancement of the feeding artery, the aneurysmal part, and the draining vein |
| Neuroblastoma | Metastatic neuroblastoma |
| In children aged < 2 years, a posterior mediastinal mass is neuroblastoma until proven otherwise Unusual presentation with neurological and/or respiratory symptoms “Dumbbell” appearance (extension of the lesion into the neural foramen) on MRI ↑ levels catecholamine May manifest as a SRPL if metastatic to the lung from primary NB elsewhere in the body or from direct extension from mediastinum |
| **E. Vascular** |
| Overlying artifact | Very round or other geometric shapes suggesting unnatural cause Removing possible artifact and repeating the CXR, will help in diagnosis Chest wall origin | Rib destruction/erosion, broad base to the chest wall and clear borders with obtuse angles from the surrounding parenchyma are helpful in detecting chest wall origin of lesion Abdominal origin | | Congenital diaphragmatic hernia | Diagnosed on prenatal US and postnatal CXR and US CT will demonstrate diaphragmatic defect and herniated abdominal contents, differentiating it from other lesions |

(continued)
**Pulmonary hematoma.** It is the result of vascular injury. History of trauma along with associated injuries (fractures, pneumothorax, etc.) are strong diagnosis indicators. Clinical presentation depends on trauma mechanism and associated injuries.

A pulmonary laceration may manifest as a round cavity, which, if filled with blood, will form a pulmonary hematoma and appear as a dense round opacity. It might also be filled with air (pneumatocele) or both air and blood (hematopneumatocele) (20). Follow-up will show gradual resolution.

**F. Others**

Extrapulmonary lesions can appear as lung parenchyma lesions on a chest X-ray mimicking SRPLs. Such lesions are listed below.

**Overlying artifacts.** Overlying foreign bodies should always be considered as potential cause of any round lung radiodensity (8). Often, they will be very round or have other geometric shapes suggesting unnatural cause. Buttons (Fig. 14), hair braids, clothing decorations, monitor leads, or other medical devices are the most common causes. When in doubt, removing the possible artifact and repeating the chest X-ray will help in diagnosis.

**Extrapulmonary lesions/pseudolesions**

**Chest wall origin.** Chest wall lesions, (i.e. neurofibromas) (Fig. 15) can appear as round lung lesions. Look for rib destruction or erosion, broad base, clear boundaries, and obtuse angles to show chest wall origin of lesions (8).

**Abdominal origin.** Congenital diaphragmatic hernias are also pulmonary lesion mimickers. They are the commonest abnormalities of the diaphragm in children and can be symptomatic depending on location, size, and contents. They are usually diagnosed on prenatal ultrasound and postnatal plain radiographs and ultrasound. CT can show the diaphragmatic defect and the hernia contents (7).

**Conclusion**

In conclusion, detecting an incidental pulmonary nodule in a child merits a different management from finding one in an adult, which means that applying the adult guidelines (Fleischner Society) is not suitable (13).

A chest X-ray is usually adequate in assessing the work-up of a SRPL. Ultrasound could be a valuable tool when targeted use is being applied. CT, MRI, PET-CT, and nuclear scan should be retained unless the appropriate clinical set-up is in place.
The radiologist should be familiar with the imaging manifestations of the SRPL. Imaging findings along with the patient’s general health status, vaccinations, previous medical history, laboratory results, and clinical examination help in elaborating and narrowing down the differential diagnosis aiding in the diagnostic approach. When diagnosis is uncertain, follow-up may offer valuable insight on the nature of the lesion.

Helpful conclusive clues for diagnosis and take-home messages are offered in Table 1.

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