Pitfalls in the sonographic diagnosis of juxtadiaphragmatic pulmonary sequestrations

Abstract Background. Sonographic evaluation of juxtadiaphragmatic lesions is frequently the initial imaging modality of choice in the pediatric population. The increasing sophistication of sonographic and Doppler technologies has led to the suggestion in the literature that lesions suspected of being juxtadiaphragmatic pulmonary sequestrations can be confidently imaged by ultrasound examination alone.

Objective. To present four cases which raise concern over the accuracy of sonographic examination of these lesions.

Materials and methods. We present four such lesions for which an initial erroneous diagnosis of pulmonary sequestration was suggested on an initial ultrasound examination.

Conclusion. The potential of a missed diagnosis of neuroblastoma has led us to propose that any lesion not demonstrating unequivocal sonographic findings of pulmonary sequestration should undergo further investigation and, if necessary, tissue sampling or excision.

Introduction

Sonographic evaluation of juxtadiaphragmatic thoracic lesions has matured into a well-accepted, sophisticated and non-invasive investigational tool. The addition of complementary Doppler technology for evaluation of vascular abnormalities permits this modality to be an excellent potential tool to investigate lesions suspected of being pulmonary sequestrations. These lesions demonstrate the sonographic and Doppler findings of the presence of an abnormal systemic arterial feeding vessel in association with a juxtadiaphragmatic mass in the first few years of life. Current literature would suggest that this combination of findings is, therefore, strongly suggestive of the diagnosis of a pulmonary sequestration [1–3, 5]. Unfortunately, our current literature is deficient in establishing the sensitivities and specificities of many of the individual sonographic findings seen in this disorder. We present a series of four cases for which the tentative erroneous diagnosis of pulmonary sequestration was made based on sonographic and Doppler findings alone. We suggest that this diagnosis can be difficult when based solely on sonographic and Doppler criteria and that cases that do not demonstrate unequivocal sonographic findings described in the literature should undergo further investigation.

Case reports

Case A

A 25-week, 725-g premature infant initially demonstrated typical clinical and radiographic features of hyaline membrane disease, which only partially responded to surfactant therapy and conventional ventilation. Supplemental intravenous alimentation was begun and, in the 3rd week of life, the clinical course and chest radiograph deteriorated, with clinical signs of sepsis and progressive right lower lobe opacity (Fig. 1A, B). A bedside ultrasound revealed an echogenic area of solid tissue in the region of the right lower lobe with a prominent central artery seen on gray scale, as well as Doppler interrogation, emanating from the region of the descending aorta (Fig. 1C, D). Pulmonary sequestration was suggested as a likely etiology. The child deteriorated and at subsequent autopsy, an infected, infarcted right lower lobe was discovered. The isolated etiologic organism was a fungus, Mala-
Fig. 1  Frontal and lateral radiographs (A, B) demonstrate uniform opacity at the right base and blunting of the costophrenic angle. Sonographic images (C, D) demonstrate an echogenic mass with a large arterial vessel originating from the descending thoracic aorta. At autopsy, this proved to be a consolidated lower lobe that was infected and infarcted by an angioinvasive fungus. A prominent systemic collateral vessel was noted.

**szzia furfur**, an angioinvasive organism that had infarcted the lung by invasion into the pulmonary artery. A large systemic bronchial collateral vessel was also seen.

Case B

Prenatal and postnatal sonographic examination of a fetus at 37 weeks and at 5 days of life demonstrated a uniformly hypoechoic mass in a left suprarenal location (Fig. 2A, B). No internal cysts or calcification was seen. Doppler interrogation demonstrated a large arterial branching vessel extending from the left superolateral margin of the mass into its center (see Fig. 2C, D). At surgery, a well-defined mass was removed from the lateral limb of the left adrenal gland. Pathologic examination demonstrated findings diagnostic of an adrenal adenoma.

Case C

A chest radiograph at a local hospital of a 9-month-old child who presented with a 3-day history of cough and fever revealed a uniform density mass in the left lower posterior mediastinum that persisted on serial radiographs despite treatment with broad-spectrum intravenous antibiotics. The child was referred to a pediatric center for evaluation. In retrospect, the widening of the rib spaces present on the outside chest radiographs was not initially appreciated at the outside institute, and the films were unavailable at the time of referral. Sonographic examination (performed at the pediatric center before the outside examinations were received) revealed an inhomogeneous mass in the left posterior mediastinum, with bright areas suggesting calcification (Fig. 3). However, Doppler study revealed a large, low-resistance arterial vessel going from the abdominal aorta into the mass. Subsequent CT demonstrated findings of internal calcification and rib erosion consistent with a neuroblastoma.

Case D

Chest radiographs performed on a normal, full-term infant boy with prolonged grunting and O₂ requirement demonstrated findings suggestive of diffuse pneumonia; however, a persistent right basal uniform opacity was seen. Ultrasound examination at 10 days of age demonstrated a uniformly echogenic mass in the right posterior thorax with an associated large systemic vessel extending into the mass (Fig. 4A). Extralobar sequestration was suspected and serial ultrasound examinations were performed at 1 month and 6 months of age, demonstrating no significant change in size or appearances. The child presented with lower-limb weakness and constipation at 10 months of age, which proved to be secondary to intraspinal invasion of the child’s thoracic neuroblastoma (Fig. 4B).