Intrapericardial foregut cyst associated with intrauterine death

E E Mooney, T D Wax, K A Reimer

Abstract
Intrapericardial developmental foregut cysts are rare and are most frequently incidental findings at necropsy in adults. A 29 year old Asian woman delivered a 24 week stillborn fetus seven days after diagnosis of intrauterine death caused by rupture of a foregut cyst. Multiple cysts occupied the wall of the right atrium and its rupture caused haemopericardium and cardiac tamponade.

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Keywords: fetal death; haemopericardium; foregut cyst

A 29 year old Asian woman (gravida 3, para 3) delivered a 24 week stillborn fetus seven days after diagnosis of intrauterine death. The pregnancy was complicated by gestational diabetes mellitus and hypothyroidism. The fetus was normally developed, weighed 513 g, with a crown-rump length of 23 cm. There was moderate maceration, but no external abnormalities were identified.

At necropsy, the pericardium was distended with 3 ml of clotted blood, with a rupture site visible in the wall of the right atrium. There were no anomalies of the gross anatomy of the heart (3.1 g) or great vessels.

Microscopically, the atrial wall was of variable thickness, with recent and organised haematomata at the rupture site (fig 1). Multiple cysts up to 3 mm in diameter were identified in the atrial wall. These were lined by a columnar epithelium that was ciliated (fig 2) and showed focal pseudostatification. No inflammatory infiltrate was present. The cysts contained cell debris that was partly calcified. Concentrically arranged smooth muscle, but no cartilage or seromucinous glands, was present beneath the epithelial lining. No neuroectodermal structures such as skin, skin appendages or neuronal tissue were identified. The atrial septum and ventricles were normal. The remainder of the necropsy was unremarkable and the placenta (166 g) showed changes consistent with intrauterine fetal death. Cytogenetic analysis performed on amniotic fluid following diagnosis of intrauterine fetal death showed a normal female karyotype (46, XX).

Discussion
Developmental foregut cysts within the pericardium are rare, with approximately 35 cases reported. Although most frequently an incidental necropsy finding in adults, some cases have presented with symptoms of pericarditis. They are frequently described as intrapericardial bronchogenic cysts (IPBC) among other terms. One third of patients with such cysts are infants, who are almost always asymptomatic with respiratory distress and cyanosis, the size and location of the cyst causing vascular compromise. IPBC are usually not associated with any cardiac anomalies. Microscopically, they should be distinguished from the more common teratomas that contain tissues of neuroectodermal derivation such as skin, teeth or glial tissue. As little distinction was made between IPBC and teratomas in the earlier literature on the subject, some of the deaths ascribed to cardiac teratoma may have been due to IPBC. For example, Jellen and Fisher reported a case of neonatal death secondary to what they called an intrapericardial teratoma, although the histology suggests that this may have been a bronchogenic cyst.

Cystic tumour of the atrioventricular node has a characteristic location, and as an endodermal derivative may also show ciliated epithelium. Ciliated epithelial cysts have been reported on the tricuspid valve and in the left ventricle. Despite the absence of other bronchial tissues such as cartilage, their histogenesis is similar. Before the obliteration of the dorsal mesocardium between the 7th and 16th somatic stages, the foregut is in close proximity to the developing heart. Sequestration of foregut tissue may occur before fusion of the paired coelomic cavities that become the pericardium, and result in intrapericardial or intramyocardial cystic rests. Those in the myocardium are
Gastric outflow obstruction caused by gall stones and leading to death by complex metabolic derangement

C O Wight, M Seed, W W Yeo, T A McCulloch

Abstract
A 67 year old woman was admitted with a three week history of vomiting, having become increasingly confused for three days. Investigations revealed deranged serum biochemistry consistent with a combination of a diabetie non-ketotic hyperosmolar state and a metabolic alkalosis consistent with gastric outflow obstruction. She was treated with intravenous saline, intravenous insulin, and subcutaneous heparin, but did not improve clinically and had an asystolic cardiac arrest the following day; she was transferred to the intensive care unit and despite treatment with inotropes she died 40 hours after admission. Necropsy revealed that the stomach was massively diluted with gas and stomach contents, and contained many small black faceted gall stones. In addition a large non-faceted brown-yellow gall stone was wedged in the pyloric antrum causing total obstruction. The patient had died from a complex metabolic derangement including...