

Case Report

Bifid sternum

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ABSTRACT

A bifid sternum is a rare congenital anomaly generally diagnosed as asymptomatic at birth. It is sometimes associated with other congenital anomalies. The sternal defects are best corrected surgically during the neonatal period within the first month of life. Sometimes the defect may be diagnosed only in adult life. We here report a case of bifid sternum which was diagnosed incidentally in a 23-year-old male.

KEY WORDS: Bifid sternum, congenital anomaly, humans

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INTRODUCTION

Sternal cleft is the separation of the sternum with orthotopic normal heart and normal skin coverage. The anomaly appears to represent an isolated developmental defect of multifactorial etiology. The defect is extremely rare and the exact incidence of this anomaly is difficult to reach but it occurred in 0.15% of all chest deformities seen in an institution during a 25-year period.^[1] Bifid sternum is generally observed at birth and is asymptomatic. However, patients with this defect may present late in childhood or even in adult life. We herein report a case of bifid sternum because of its rarity and presentation in adulthood.

CASE REPORT

A 23-year-old male presented with a bony defect in the central upper part of his chest wall which had been present since birth. On interrogation it was revealed that he was a full-term baby at birth, with a birth weight of 2500 g and was delivered by a spontaneous uneventful vaginal delivery at 39 weeks of gestation. On examination there was a wide gap in the upper part of the sternum, more evident during

inspiration [Figure 1]. Pulsation of great vessels was easily seen through the defect which was covered by a thin layer of skin. No obvious abnormality was noted in any other system. Laboratory investigations were within normal limits. Chest X-ray postero-anterior view revealed a wide gap between the medial ends of the clavicle, and absence of manubrium and upper part of the sternum. Computed tomography scan of the thorax confirmed the defect showing the incomplete superior cleft of the sternum in a U shape [Figure 2]. Echocardiography showed normal pericardium and normal cardiac anatomy. Ultrasonography of the abdomen was normal. Cardiothoracic surgeon was consulted, who opined for surgical repair of the defect. However, the patient refused to undergo surgery and was lost to follow-up.

DISCUSSION

Sternal bands form in the sixth week of intrauterine life from the lateral plate mesoderm, and the fusion of these separate bands on either side of the anterior chest wall occurs in the ninth week in a craniocaudal direction.^[2] The manubrium arises from the mesenchymal promordia between the ventral ends of the developing clavicles. The manubrial ossification centers on each side of the mid-sternum are fused at birth and fusion of ossification centers in the sternal body is completed by the sixth year. Failure of fusion of the sternal body results in congenital sternal clefts. Sternal clefts are generally observed at birth without symptoms, and are more common in females.^[1,3] The clefts can be divided into complete and incomplete varieties, the latter being more common.^[4] Incomplete clefts can be further classified into superior and inferior.

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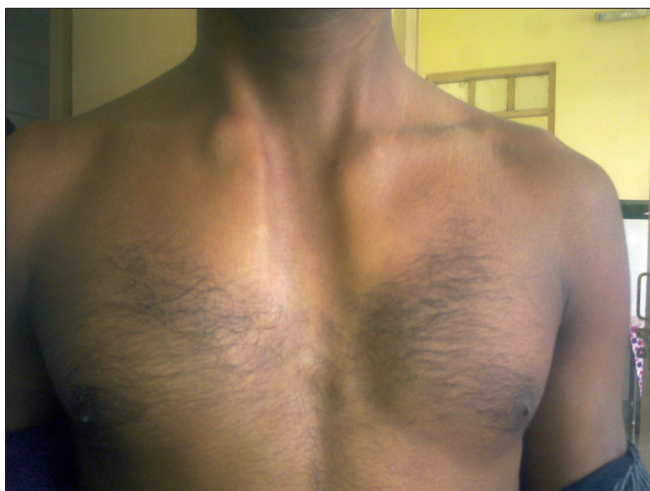


Figure 1: Wide gap in the upper part of the sternum covered with skin

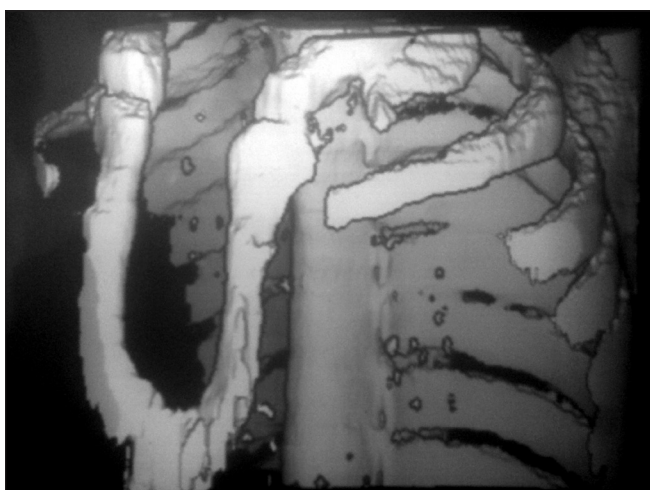


Figure 2: 3D reconstruction of CT thorax showing U-shaped gap of the sternum

The superior sternal cleft may be V-shaped when the cleft reaches the xiphoid process, or U-shaped, with a bony bridge joining the two edges ending at the third or fourth costal cartilages,^[5] as seen in our case. Inferior defects are extremely rare, and often occur as a part of Cantrell's pentalogy which consists of bifid sternum, omphalocele, deficiency of anterior diaphragm, defects in diaphragmatic pericardium and congenital heart disease like ventricular septal defect or left ventricular diverticulum.^[6] As fusion of the sternum occurs craniocaudally, it is expected that the failure of fusion of the upper part would result in cleft of the whole sternum; the cause of the more common occurrence of the superior defect with normal fusion of the lower part is obscure.^[7]

Although often occurring as an isolated defect, sternal cleft may be associated with different congenital anomalies like midline abdominal raphe, ventral hernias, partial or total ectopia cordis, patent ductus arteriosus, absent pericardium, cervicofacial hemangioma, diastasis recti, etc.^[2,6-12]

The diagnosis of sternal cleft is easily done by inspection and palpation. Diagnostic investigations are directed to exclude associated anomalies. Prenatal diagnosis is difficult but has been reported with the help of ultrasonography.^[13]

Repair of the defect is necessary to restore bony protection to the mediastinal structures, to provide normal intrathoracic pressure relationships and to eliminate paradoxical movement of the thoracic viscera, and the large visible contour deformity.^[9] Age of the patient is an important factor in determining the technique of surgical correction.^[9,10] The optimal choice of treatment is primary direct closure in the neonatal period when flexibility of the chest wall is maximal and compression of the underlying structure is minimal.^[1-3,5,8] If the patient presents later in life, reconstruction surgery is required because a simple approximation is impossible because of increasing rigidity of the chest wall and difficulty in accommodating the heart and lungs within the circumference of chest.^[10] Various techniques have been used for the reconstruction of the chest wall. One of the earliest successful techniques was sliding costal cartilage technique by Sabiston.^[11]

Subsequently, either prosthetic material like Marlex mesh, Teflon mesh, silicon elastomer, and acrylic plate, or autogenous tissues like parietal skull, costal cartilage, pectoralis major muscles have been used for the repair of the cleft.^[5,6,14-16] However, it is always advocated to prefer the autogenous tissue over the prosthetic materials, considering the risk of infection and inability of the prosthetic inert materials to grow with the growth of the patient.^[16,17]

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