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Coarctation of AORTA – A Case History

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Case History

We present a case of coarctation of aorta (CoA) in an adult female. The patient is 23 years old medical student and she is a female with no other significant complaints prior to her diagnosis. She is an athlete and sports person. She practiced Shuttle badminton with vigorous physical exercise to develop her stamina. She continually played the game with no clinical findings. She realized that she is gaining weight which did not respond to her strict exercise schedule. The patient went for clinical advice for her obesity and accidentally she was diagnosed to have very high Blood Pressure(180/110mm). Her endocrine profile and other parameters were analyzed which were found to be normal. She was diagnosed to have coarctation of Aorta. (Severe post-subclavian coarctation of Aorta)

Introduction

Obstruction to the flow of blood in the aorta may be called as Coarctation of aorta (CoA). CoA can be found in any region related to thoracic and abdominal aorta. Post subclavian artery is the most common site of CoA. In other words, CoA is reported to be present distal to the subclavian artery (the region where ductus arteriosus connects to the aorta) [1].

The first case of CoA was illustrated by Giovanni Morgagni, in the 18th century. Typically, there is presence of medial thickening with "shelf like" tissue protruding in the lumen of aorta from the posterior aortic wall. Dr, Crawford in 1944 performed the first surgical intervention which was followed by transcatheter balloon angioplasty in 1980. Recent technological intervention has prolonged life span of the patients [2,3].

About 25 to 30 percent of congenital anomalies is due to congenital heart disease (CHD). CoA is commonly found in 6-8 percent of CHD (approximately 4 per 10,000 live births).

CoA is also associated with other cardiac and other anomalies. CoA may be associated with bicuspid aortic valve defect, ventricular septal defect, patent ductus arteriosus and others reported in the literature [4].

Shone complex is a congenital (present at birth) heart disease. It affects blood flows from the left side of the heart. It may also be associated with CoA. CoA is associated with Turner syndrome and William syndrome. In William syndrome can be present anywhere in the aorta including abdominal aorta [5].

Aorta develops from pharyngeal arches and its arterial system. Aortic arch development starts in the third week of gestation. This then gets transformed into final fetal arterial arrangement during eighth week of gestation [6].

Aortic sac develops from ventral primitive aorta and the descending aorta from the dorsal primitive aorta. Pharyngeal arches help to connect these two portions. Of the six sets of pharyngeal arches, fourth pharyngeal arch is involved in the development of aortic arch and the development of branch of pulmonary arteries, ductus arteriosus and other aortic arch branches are contributed by other pharyngeal arches [6-7].

What may contribute to the development of CoA

1. Tissue from ductus arteriosus may be incorporated in the aortic wall connecting the descending aorta. When the ductus arteriosus constricts after birth, the tissue in the isthmus area may cause development of CoA.
2. The isthmus area between left subclavian artery and the ducutus arteriosus is narrow during developmental stage. Failure to grow in normal size can cause CoA.
3. An abnormal involution of a small segment of left dorsal aorta which may move cranially along with the subclavian artery to form CoA in isthmus region [8].

Variations in Coarctation of Aorta

There may be preductal, ductal and postductal coarctations. If the narrowing is proximal to the ductus arteriosus it is called as preductal coacrtation, if it is at the insertion of the ductus arteriosus it is considerated to be ductal coarctation and if it is distal to the insertion of ductus aarteriosus it is known as postductal coarctation.

Preeductal form results in a decrease in blood flow through the left side of the heart. In postductal coarctation, there is notching of the ribs, hypertension in the upper extremities and weak pusles in the lower extremities.

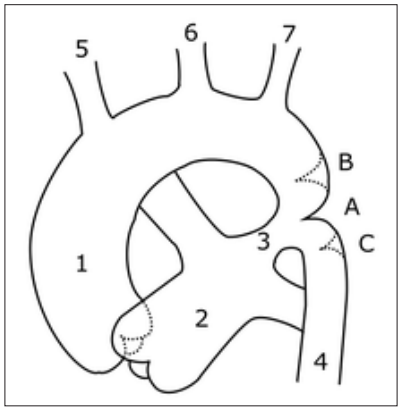

Figure: Line drawing of the possible locations of a coarctation of the aorta, relative to the ductus arteriosus. A: Ductal coarctation, B: Preductal coarctation, C: Postductal coarctation. 1: Aorta ascendens, 2: Arteria pulmonalis, 3: Ductus arteriosus, 4: Aorta descendens, 5: Truncus brachiocephalicus, 6: Arteria carotis communis sinistra, 7: Arteria subclavia sinistra

Clinical presentation

Based on patient's age and gender the clinical findings may vary and early clinical findings may be due to severe disease.

Younger Children

Patent ductus arteriosus in newborns and neonates perfuse lower body even if they have CoA and show no symptoms. Once ductus arteriosus closes after birth the babies may show delayed capillary refill, feeding problems, decreased responsiveness, metabolic acidosis, mesenteric ischemia, myocardial depression with absent or feeble femoral pulse.

Weak femoral pulse, upper extremity hypertension, a systolic murmur over sternal border with radiation to the back, and upper-lower extreme systolic blood pressure gradient may be present.9

Adolescent and Adults

Most of these cases with CoA are diagnosed when they go for measurement of blood pressure or heart murmur. Upper extremity hypertension, weak femoral pulse, arm-leg pressure difference (>20mmHg is significant), a systolic murmur following back flow through the coarctation segment or a continuous murmur from the collateral flow around the site of coarctation site are reported as clinical signs [10].

The present case history presented was diagnosed by accident and the demographics, diagnosis and therapeutic intervention results are given below: (The findings of Echo, the recording of the intervention will be given if required by the journal). Informed consent was obtained from the patient.

Discussion

The present case shows many a time a congenital anomaly may be missed and therefore, it is necessary to screen carefully for cardiac functions in children and adolescents even if there is a slight change in blood pressure measurement and other clinical signs. Apart from the known causes of congenital anomalies like artrial septal defect (ASD), Teratology of Fallot, Patent ductus arteriosus, one need to realize that coarctation of aorta (CoA) accounts for sizeable part of congenital heart diseases [11]. With the advances in technology, cardiac magnetic resonance imaging (MRI), echocardiography and computed tomography (CT) help in the early diagnosis of these conditions. The presence of such anomalies in children with very few cases in adults may give way to assumption that such anomalies including CoA may not be present in adolescents and adults [12].

Accidental finding of a very high blood pressure in the present case (180/110mm Hg) led to diagnosis of a severe subclavian CoA which needed immediate attention. A delay in the procedure carried out would have caused more irreversible damage including left ventricular hypertrophy (LVH). The pan systolic murmur identified in the patient had led the patient to do more screening of cardiac function and other related procedure to diagnose the condition. The balloon angioplasty and stenting in the patient had controlled her blood pressure. The patient is put on amilodipine (5mg) twice daily for an initial period of 6 months with exospirin as a prophylactic measure [13].

The patient is slowly recovering from the psychological pressure and regaining her confidence to build her capacity to exercise. The case also indicates the sudden weight gain which does not respond to severe exercise may point to any other associated conditions including CoA [13,14].

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