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Aortopulmonary window: An overview

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Abstract
Aortopulmonary (AP) window is exactly a rare natural anomaly that represents roughly 0.2 and 0.5 of all natural heart abnormalities. It consists of communication between the aorta and the pulmonary roadway or its branches. Aortopulmonary window occurs due to the abnormal development of the heart's major blood vessels during early fetal growth. In utmost cases, this heart disfigurement occurs by chance, with no clear reason. This condition can do on its own or with other heart blights similar as 1. Tetralogy of Fallot, 2. Pulmonary atresia, 3. Truncus arteriosus, 4. Atrial septal disfigurement, 5. Patent ductus arteriosus, 6. Intruded aortic bow. Treatment for aortopulmonary window involves surgery to close the hole between the aorta and the pulmonary roadway with a patch or device. Associated lesions are generally repaired during the same surgery. Associated lesions are generally repaired during the same surgery. More complex repairs and myocardial protection strategies are needed in cases with associated lesions, adding the morbidity and mortality associated with the operation.

Keywords: Aortopulmonary (AP) window, congenital heart disease, left-to-right shunt

Introduction
Aortopulmonary Window may be a rare congenital cardiac malformation which will cause congestive cardiac failure if left untreated. To avoid the high morbidity and mortality related to this condition, it must be promptly diagnosed and treated [1].

Definition
Aortopulmonary window could be a rare heart defect within which there's a hole connecting the key artery taking blood from the heart to the body (the aorta) and also the one taking blood from the heart to the lungs (pulmonary artery). The condition is congenital, which implies it's present at birth [2].

Etiology
An aortopulmonary window can occur in isolation or, in up to 50% of cases, in association with other cardiac defects, including interrupted aorta (most frequent), coarctation of the aorta, transposition of great vessels, and tetralogy of Fallot. Variation of pulmonary arteries, head and neck vessels and coronary are described in association with this defect. Although considered a conotruncal defect, DiGeorge syndrome isn't related to AP window [3].

Epidemiology
AP window was first described by J. Elliotson in 1830. This abnormality occurs during separation of the truncus arteriosus into the arterial blood vessel and aorta and is related to two separate semilunar valves. Aortopulmonary (AP) window is a rare congenital anomaly, representing 0.2 to 0.5 percent of all cases of congenital heart disease. The primary successful surgical closure of AP window was reported by Robert Gross in 1952 [4].

Pathophysiology
The pathophysiology of AP window is comparable to it of a ventricular congenital heart defect, PDA, or truncus arteriosus consisting of a left-to-right shunt. The quantity of left-to-right shunting is expounded to the dimensions of the defect and also the pulmonary vascular resistance. Patients with small defects will be completely asymptomatic. With large defects, and because the pulmonary vascular resistance decreases within the first weeks of life, symptoms of congestive coronary failure develop rapidly, and irreversible pulmonary vascular disease can occur as early as during the primary 12 months of life.

If untreated, 40 percent of patients will die of intractable failure during the primary year of life, and survivors will succumb to the sequelae of congestive coronary failure and severe irreversible pulmonary vascular disease during childhood [5].

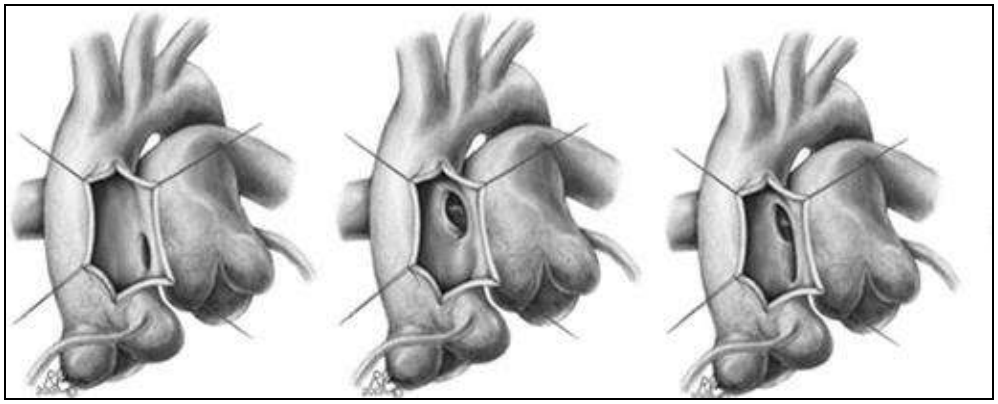
Types

AP window defects are separated into different subtypes supported location and arterial structures involved:

Type I: are proximal defects that are located in the proximal aorta above the sinus of Valsalva midway between the semilunar valves and pulmonary bifurcation.

Types II: are distal defects that are located in the upper portion of the ascending aorta before the aortic branches with aortic origin of right pulmonary artery.

Type III: are defects that are large and comprise both types I and II, such defect involves the majority of the ascending aorta, pulmonary trunk and the right pulmonary artery [6].



Type 1: Proximal Defect **Type 2:** Distal Defect **Type 3:** Total Defect

Fig 1: AP window defects are separated into different subtypes supported location and arterial structures involved

Symptoms

Babies with an aortopulmonary window generally don't feed well, have rapid breathing, are irritable and tire easily. Other symptoms include:

- delayed growth
- rapid heartbeat
- respiratory infections

Diagnosis

A chest X-ray will show cardiomegaly and increased pulmonary vascular markings. Electrocardiogram will demonstrate tachycardia and increased right and left-sided voltages. Echocardiography should completely evaluate the remaining cardiac structures, including evaluating other cardiac diseases like tetralogy of Fallot, interruption of the aorta, and care should be taken to identify the coronary arteries [7].

Treatment

Treatment for aortopulmonary window involves surgery to shut the opening between the aorta and also the pulmonary artery with a patch. This surgery is often done as soon as possible after the diagnosis is formed, usually when the kid could be a newborn. The definitive management within the AP window would be corrective cardiac surgery, which is performed typically within the time of life. Medical management of the congestive coronary failure is an option if the patient's conditions are conducive to perform corrective surgery. Earlier surgeries can prevent irreversible changes within the pulmonary vasculature. Surgical techniques are quite simple in isolated defects, which involves division and separation of the great vessels with patch closure of the defect [8].

Prognosis

In most cases, surgery is successful, though early diagnosis and treatment is key to success. Children who are treated early usually have no lasting health problems [8].

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