

## TETRALOGY OF FALLOT: ORIGIN, DIAGNOSIS, AND PATHOLOGICAL CHANGES

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**Introduction:** Tetralogy of Fallot (see Tetralogy of Fallot image) accounts for 7-10% of all congenital heart defects. Associated anomalies include right aortic arch defects (25%), altered coronary artery anatomy (5-10%), pulmonary artery stenosis, presence of aortopulmonary collateral vessels, patent ductus arteriosus, complete atrioventricular septal defect, atrial septal defect, and accessory septal valve defect. Tetralogy of Fallot has 4 features: a large ventricular septal defect, obstruction of the right ventricular outflow tract and pulmonary valve stenosis, right ventricular hypertrophy, and dextroposition of the aorta. Symptoms include cyanosis, dyspnea on feeding, poor growth rate, and severe hypercyanotic symptoms (sudden, potentially fatal episodes of severe cyanosis). A coarse systolic murmur is often heard at the upper left sternal border, with a single second heart sound (S2). The diagnosis is made by echocardiography. The definitive treatment is surgical repair of the defect.

The ventricular septal defect in tetralogy of Fallot is often described as a type of displacement, as the conus septum is displaced anteriorly. This displaced septum projects into the pulmonary outflow tract, often resulting in obstruction and hypoplasia of key structures, including the pulmonary valve, pulmonary trunk, and branches of the pulmonary arteries. The ventricular septal defect is usually large; therefore, the systolic pressures in the right and left ventricles (and aorta) are the same. The pathophysiology depends on the degree of right ventricular outflow obstruction. Moderate obstruction may result in a pure left-to-right shunt across the ventricular septal defect; severe obstruction causes a right-to-left shunt, resulting in low systemic arterial saturation (cyanosis) that is unresponsive to supplemental oxygen.

**Research methods and materials:** These factors lead to the formation of a vicious circle, which is caused by an initial decrease in arterial blood PO<sub>2</sub>, which stimulates the respiratory center and leads to hyperpnea and increased adrenergic tone. The increase in circulating catecholamines increases contractility, which increases the obstruction of the outflow tract.

**Signs and symptoms of tetralogy of Fallot**

Newborns with severe right ventricular outflow tract obstruction (or atresia) will have significant cyanosis and dyspnea during feeding, leading to poor weight gain. Newborns with mild obstruction may not have cyanosis at rest.

Hypercyanotic spells may be triggered by activity and are characterized by hyperpnea (rapid, deep breathing), irritability, prolonged crying, increasing cyanosis, and decreased or absent heart sounds. Attacks occur most often in young children; the peak incidence occurs between 2 and 4 months of age. Severe attacks can cause lethargy, seizures, and sometimes death. Some infants may occasionally drool during play, a condition that increases systemic vascular resistance and aortic pressure, which reduces right-to-left ventricular shunting and therefore increases arterial oxygen saturation.

Auscultation reveals a coarse systolic ejection murmur of grade 3-5/6 at the left mid-upper border of the chest (see Heart Murmur Intensity Table). The murmur in tetralogy is associated with pulmonary stenosis. The ventricular septal defect is usually asymptomatic because it is large and lacks a pressure gradient. Therefore, the murmur is softer as the pulmonary outflow obstruction becomes more pronounced. The second heart sound (S2) is usually single because the pulmonary component is significantly reduced. Right ventricular beats and systolic chest vibrations may be present.

**Results:** The diagnosis of tetralogy of Fallot is supported by history and clinical examination, chest radiography and ECG, and is established by two-dimensional echocardiography with color flow and Doppler studies.

Chest radiography reveals a wooden shoe heart with a concave segment of the pulmonary artery and reduced pulmonary vascular markings. A right aortic arch is present in 25%.

The ECG reveals right ventricular hypertrophy, right axis deviation, and sometimes right atrial enlargement.

Cardiac catheterization is rarely required unless there is a suspected coronary artery anomaly that may interfere with surgical management (e.g., anterior descending coronary artery disease) and is not detectable by echocardiography. MRI or CT may also be used to demonstrate coronary artery anatomy.

**Conclusion:** The diagnosis of generalized anomalous pulmonary venous drainage is based on chest radiography and echocardiography. Cardiac catheterization is rarely required; Sometimes cardiac MRI or CT angiography may be necessary to better define the anatomy of the pulmonary venous return. Radiography reveals severe diffuse pulmonary edema in the presence of small heart

and pulmonary venous obstruction; otherwise - cardiomegaly with increased pulmonary vascular pattern. In the supracardiac type of generalized anomalous pulmonary venous drainage (TAPVD), the entire heart shadow begins to resemble the shape of a snowman, the upper part of its circumference is formed by a rounded shadow given by the entry of the pulmonary veins into the drainage vein.

The ECG shows right axis deviation, right ventricular hypertrophy, and sometimes right atrial enlargement.

Preoperative treatment of heart failure with medications (e.g., diuretics, digoxin, angiotensin-converting enzyme inhibitors)

In neonates with complete anomalous pulmonary venous return obstruction, immediate surgical reconstruction is required. Later in life, heart failure should be treated, followed by surgery once the infant's condition has stabilized.

Surgical treatment consists of creating a wide anastomosis between the pulmonary venous connection and the posterior wall of the left atrium. It is important to ligate the vessel to decompress the site of the connection to the systemic venous circulation to prevent postoperative left-to-right shunting. Correction of the defect to return to the coronary sinus is different: in this case, the coronary sinus is transferred to the left atrium and its opening to the right atrium is closed.

Endocarditis prophylaxis is recommended before surgery, but is required for the first 6 months after defect repair unless there is a residual defect near the surgical site or prosthetic material.

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