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CONGENITAL HEART DISEASE: TETRALOGY OF FALLOT

Luiz Carlos Gonçalves Filho
Centro Universitário Alfredo Nasser-Medicina

Fabricio Miranda Dutra de Amorim
Centro Universitário Alfredo Nasser-Medicina

Vanessa Teodoro Laureano Cândido
Centro Universitário Alfredo Nasser-Medicina

Laila Silva Teixeira Azeredo Bastos
Centro Universitário Alfredo Nasser-Medicina

Lailla Christye Faria Neves
Centro Universitário Alfredo Nasser-Medicina

Luana Cristina da Costa Mendes
Centro Universitário Alfredo Nasser-Medicina

Alex Jesus da Costa
Centro Universitário Alfredo Nasser-Medicina

Denes Silva Mendes
Centro Universitário Alfredo Nasser-Medicina

Fabio Andrade Mota
Centro Universitário Alfredo Nasser-Medicina

Raisa D´ Ricolli Rebouças Rocha
Centro Universitário Alfredo Nasser-Medicina

Carlos Eduardo Sobreiro Lourenço
Centro Universitário Alfredo Nasser-Medicina

Brigida de Cassia Ribeiro
Centro Universitário Alfredo Nasser-Medicina

Anádio Marcon Ribeiro
Centro Universitário Alfredo Nasser-Medicina

Pedro Henrique Luiz Salgado
Centro Universitário Alfredo Nasser-Medicina

Mattheus Duarte da Veiga Jardim
Centro Universitário Alfredo Nasser-Medicina

Abstract: OBJECTIVE: Tetralogy of Fallot (TOF) is the most common cyanotic congenital heart defect, occurring in approximately 1 in 3,500 births and accounting for 7% to 10% of all congenital heart malformations. Tetralogy of Fallot (ToF) occurs in approximately 1 in 3600 live births and accounts for 3.5% of babies born with congenital heart disease. The objective of this work is to carry out an epidemiological survey on Tetralogy of Fallot and its respective treatment. **METHODS:** This is a literature review, of the narrative type, which aims to describe the characteristics of Tetralogy of Fallot, from a theoretical point of view, through materials that have already been published on the subject in question, through analysis and interpretation of the literature. Inclusion criteria were: articles in Portuguese and English; published in the period from 2015 to 2023 and that addressed the themes proposed for this research, review-type studies available in full. After the selection criteria, 6 articles remained, which were subjected to thorough reading for data collection. The results were presented in a descriptive way, divided into thematic categories addressing: describing the subtitles or points that were mentioned in the discussion. **RESULT AND DISCUSSION:** Tetralogy of Fallot is the most common cyanotic heart disease in children who survive without treatment beyond neonatal age, requiring intervention in the first year of life. It accounts for 7% to 10% of birth defects, affecting men and women equally and occurring in 3 to 5 out of every 10,000 live births. The clinical presentation varies according to the severity of the obstruction of the right ventricular outflow tract, presenting more commonly in neonates with a certain degree of cyanosis. In some patients, cyanosis manifests itself months later in life when the rate of obstructions worsens. On auscultation, patients have a normal first heart sound with a single loud second heart sound. Currently,

early survival after a complete primary repair in large centers is reported to be between 98% and 100%. Despite this, complete primary repair in neonates and infants, in general, is still controversial. Since the 1970s, surgeons have recommended complete repair by 6 months of age and at the latest by 12 months for asymptomatic and non-ductal-dependent infants. **CONCLUSION:** The diagnosis and management of Tetralogy of Fallot are performed by an interprofessional team that includes a pediatrician, pediatric cardiologist, cardiac surgeon and radiologist. In general, all children with Tetralogy of Fallot require surgery; time may vary depending on symptoms.
Keywords*;* Heart disease; Tetralogy of Fallot; Treatment.

INTRODUCTION

Tetralogy of Fallot (TOF) is the most common cyanotic congenital heart defect, occurring in approximately 1 in 3,500 births and accounting for 7% to 10% of all congenital heart malformations. Tetralogy of Fallot (ToF) occurs in approximately 1 in 3600 live births and accounts for 3.5% of babies born with congenital heart disease.

Tetralogy of Fallot (TOF), historically and appropriately referred to as Steno-Fallot tetralogy, was first described by Danish physician/anatomist Dane Niels Stensen, also referred to as Nicoulas Steno in Latin, a pioneer in anatomy and geology. His work has made significant contributions to the field of cardiac anatomy and pathology. The discovery of the constellation of findings that characterize tetralogy was first described in a brief article entitled "Dissection of a Monstrous Fetus in Paris" in 1671, highlighting the unusual way in which the arteries emerge, the narrowing of the pulmonary artery, the absence of ductus arteriosus, a subaortic interventricular septal defect, an aortic duct common to both ventricles, and the physiology of the fetal cardiac circulation by describing how blood was redirected directly to the aorta instead of the pulmonary artery.

This article is not intended to be a comprehensive review, but rather to present areas of emerging science for clinicians and scientists to advance toward a better understanding of the long-term management of patients with Tetralogy of Fallot. Specifically, the following themes are presented: 1) Etiology 2) Pathophysiology 3) Evaluation; 4) Treatment.

METHOD

This is a literature review, of the narrative type, which aims to describe the characteristics of Tetralogy of Fallot, from a theoretical point of view, through materials that have already been published on the subject in question, through analysis and interpretation of the literature. Inclusion criteria were: articles in Portuguese and English; published in the period from 2015 to 2023 and that addressed the themes proposed for this research, review-type studies available in full. Exclusion criteria were: duplicate articles, available in summary form, which did not directly address the studied proposal and which did not meet the other inclusion criteria.

The review was carried out from July to August 2023, through searches in the Virtual Health Library (VHL), Latin American and Caribbean Literature in Health Sciences (LILACS) databases, National Institutes of Health's Library of *Medicine* (PubMed) and *Scientific Electronic Library Online* (SciELO). The following descriptors were used: "cardiovascular diseases", "epidemiology aspect", "diagnosis", ""treatment" and "preventions" in order to find articles relevant to the subject addressed.

After the selection criteria, 6 articles remained, which were subjected to thorough

reading for data collection. The results were presented in a descriptive way, divided into thematic categories addressing: describing the subtitles or points that were mentioned in the discussion.

DISCUSSION

The first case of Tetralogy of fallot was reported in America at the University of Pennsylvania by Thaxter in 1816, with subsequent cases reported by Peacock (1858 and 1869), Widman (1881) and finally Fallot (1888). Etienne-Louis Arthur Fallot described with style and elegant detail four key features that differentiate it from other cyanotic heart conditions, emphasizing that this was not a product of chance and that the cyanosis was not caused by a patent foramen ovale, as proposed by many others. He attributed this to an intrauterine pathological process and understood that this tetralogy was essentially just an anomaly involving the pulmonary artery and the subpulmonary infundibulum, causing pulmonary stenosis, interventricular communication, biventricular origin of the aorta and right ventricular hypertrophy, disqualifying the patent foramen ovale as fifth anatomical association. The names used by Fallot were "La maladie bleue" (the blue disease) or "cyanose cardiaque" (cardiac cyanosis). In 1924, Maude Elizabeth Seymour Abbott, a pioneer in pediatric cardiology from Montreal, Canada, titled it "tetralogy of Fallot".

Surgical repair was first introduced in the 1950s and there is now a large population of adults with repaired tetralogy. Many of the aspects of short-term management have been resolved, although it remains debatable whether symptomatic neonates must undergo primary repair or palliation first. In 2001, independently validated data, pooled from all 13 centers performing cardiac surgery in the UK, indicated a 97% survival of one year after the operation.1 Other reports indicate that of patients alive 30 days after the operation there is a survival of 98% in 20 years, and of those operated on as children, the 30-year survival is greater than 90%.2

This has focused attention on long-term follow-up, as many patients with repaired ToF are now middle-aged. Given that this population includes patients well beyond adolescence, it has also surpassed the term "adult congenital heart disease" (GUCH), which could be considered paternalistic. They have adult congenital heart disease and will need specialist care well into old age.

ETIOLOGY AND EPIDEMIOLOGY

The development of tetralogy of Fallot is multifactorial; has been associated with untreated maternal diabetes, maternal retinoic acid ingestion, phenylketonuria, chromosomal abnormalities (trisomies 21, 18, 13), chromosome 22q11.2 microdeletions, and Alagille syndrome with JAG1/NOTCH2 mutations (Khan SM. et al. 2019). Other genetic abnormalities predisposing to tetralogy of Fallot include mutations in the NKX2.5 transcription factor, methylenetetrahydrofolate reductase polymorphism, and mutations in TBX1 and ZFPM2 (Apostolopoulou SC et al 2019).

Tetralogy of Fallot is the most common cyanotic heart disease in children who survive without treatment beyond neonatal age, requiring intervention in the first year of life. It accounts for 7% to 10% of birth defects, affecting men and women equally and occurring in 3 to 5 out of every 10,000 live births (Wise-Faberowski L, Asija R, McElhinney DB. 2019).

PATHOPHYSIOLOGY

The development of the human heart begins around the 20th day of gestation, with the fusion of the external endocardial tubes into a single tubular structure, the heart tube. Posteriorly, the heart tube bends and loops, with the development of an atrium that is cranial and dorsal, and a primitive ventricle moves downward, ventrally, and to the right. The right ventricle is the dominant chamber in the embryo and fetus, receiving 65% of venous return, and is the major contributor to the lower body, placenta, and lungs. The right ventricle can be described by three components: the inlet, which consists of the chordae tendineae of the tricuspid valve and the papillary muscles; the trabeculated apical myocardium; and the infundibulum or cone. The exact embryological process that contributes to the development of tetralogy of Fallot is still unknown, but one observed association is an anterior and cephalad deviation of the infundibular septum that results in a misaligned ventricular septal defect, with a dominant aortic root causing a subsequent obstruction to the right ventricular outflow (Ho AB, Bharucha T, et al 2018).

Ventricular septal defects seen in patients with tetralogy of Fallot are usually perimembranous that may extend into the muscular septum. Different factors may contribute to right ventricular outflow obstruction, including the pulmonary valve, which is usually bicuspid and stenotic, the hypoplastic pulmonary valve annulus, infundibular septal deviation that causes subvalvular obstruction, and hypertrophy of the muscle bands in this region (McLeod G et al 2018). The degree of the dominant aorta usually varies and receives blood flow from both ventricles. The physiological process surrounding the hypercyanotic episodes or "Tet crises" in tetralogy of Fallot consists of a decrease in systemic vascular resistance or an increase in pulmonary resistance, contributing to a right-to-left shunt across the ventricular septal defect, causing desaturation marked (Senst B, Kumar A, Diaz RR, 2022).

The clinical presentation varies according to the severity of the obstruction of the right ventricular outflow tract, presenting more commonly in neonates with a certain degree of cyanosis. In some patients, cyanosis manifests itself months later in life when the rate of obstructions worsens. On auscultation, patients have a normal first heart sound with a single loud second heart sound. The greater the degree of obstruction, the more prominent the murmur, usually described as a crescendo-decrescendo with severe systolic ejection quality and best audible at the middle to upper left sternal border with posterior radiation. The murmur may sometimes have a regurgitant quality, and an early systolic click may be heard along the left sternal border. "Tet crises" or hypercyanotic episodes present during childhood and subside after 4 to 5 years of age. Dehydration or agitation often precipitates tet attacks, and if patients do not receive prompt and adequate treatment, they may develop severe cyanosis and hypoxia which subsequently can lead to syncope and even death.

EVALUATION OF THE PATIENT WITH TETRALOGY OF FALLOT

Up to 50% of patients are diagnosed prenatally by fetal echocardiography, anticipating the need for postnatal prostaglandin therapy if there is evidence of severe right ventricular outflow obstruction. Useful studies to aid in diagnosis and evaluation include chest X-ray, electrocardiogram, and echocardiogram. Chest X-rays usually show a normal-sized cardiac silhouette, with the apex facing upward and the main segment of the pulmonary artery concave, commonly known as "boot-shaped". On the electrocardiogram,

it is common to observe signs of right atrial enlargement and right ventricular hypertrophy showing axis deviation to the right, prominent R waves anteriorly and S waves posteriorly, vertical T wave in V1 (abnormal after 7 days of life up to 10 years of age). and a qR pattern in the right precordial leads. Among imaging tests, the echocardiogram is the gold standard, addressing the anatomy and severity of the right ventricular outflow obstruction, the location and number of ventricular septal defects, and evaluating anomalies or variants associated with the coronary arteries and the aortic arch. The main limitation of transesophageal echo in patients with tetralogy of Fallot (TOF) is the visualization of the distal pulmonary arteries. Cardiac magnetic resonance imaging can be used and is particularly useful in adults with repaired Tetralogy of Fallot. Cardiac catheterization is not commonly used but can help assess the level of obstruction, pulmonary stenosis or hypoplasia, coronary artery anatomy, and the presence of collaterals and accessory septal defects.

The most frequent causes of mortality in patients without surgical intervention include episodes of hypoxia (68%), cerebrovascular accidents (17%) and brain abscesses (13%) (Phillips S, Pirics M. 2017). In the first year of life, 25% of infants with severe right ventricular outflow tract obstruction die if untreated, 40% by three years of age, 70% by 10 years, and 95% by 40 years (Townsley MM et al. 2019).

TREATMENT

Newborns with severe right ventricular outlet obstruction presenting with profound hypoxemia and cyanosis may require prostaglandin therapy to maintain ductal patency and pulmonary flow prior to surgical repair (Sun HY, Proudfoot JA, McCandless RT 2018). Tet periods require a rapid and aggressive approach, including positioning (knee-chest) to increase systemic vascular resistance, oxygen therapy to cause pulmonary vasodilation and systemic vasoconstriction, intravenous fluid boluses to improve right ventricular filling and pulmonary flow; morphine, intravenous beta-blockers to help improve right ventricular outflow obstruction by relaxing the muscle, and intravenous phenylephrine to increase systemic afterload. If heart failure develops, digoxin and loop diuretics are good pharmacological therapeutic options (Mawad W, Mertens LL 2018; Refaat MM, Ballout J, Mansour M 2017).

Following the recommendation of the American Heart Association (2019) guidelines, all patients with unrepaired cyanotic congenital heart disease must receive prophylaxis for subacute bacterial endocarditis for dental procedures, respiratory procedures, or infected skin procedures. Other reasons for prophylaxis are heart valve prostheses, previous history of endocarditis, and congenital heart disease completely repaired with prosthetic material or device for 6 months after the procedure.

Understanding that tetralogy of Fallot involves only the subpulmonary infundibulum, "Stensen's monology", had important diagnostic and surgical repercussions. With the recognition of tetralogy of Fallot, the era of cardiac surgery to repair congenital heart defects arrived. The first surgical treatment was performed at John Hopkins with Dr. Helen Taussig. She noted that children with cyanotic heart problems had a better prognosis if the ductus arteriosus remained open. In 1939, after reading the first report of successful closure of a patent ductus arteriosus by Dr. Robert Gross, she thought of creating a duct for cyanotic children. She approached Dr. Alfred Blalock and Vivien Thomas, a surgical technician in Blalock's laboratory, and proposed a surgical solution

for Tetralogy of Fallot.

In 1944, Dr. Blalock, with Thomas at his side, successfully created an artificial canal through a left anterolateral thoracotomy, anastomosing the proximal end of the left subclavian artery with the left pulmonary artery in a 15-month-old woman who was critically ill with cyanosis. and a weight of 4 kg. The Blalock-Taussig or systemic pulmonary artery shunt, created by Blalock, Taussig and Thomas, marked the genesis of the era of congenital heart surgery, becoming an acceptable palliative therapeutic option for cyanotic defects with decreased pulmonary blood flow. Most congenital surgeons perform a modified BT shunt through a posterolateral thoracotomy or median sternotomy or a central shunt through a median sternotomy. The modified BT shunt uses a tubular prosthetic graft (made of polytetrafluoroethylene) that is interposed between a systemic artery and the pulmonary artery, which differs from what was initially described as a direct anastomosis with a higher probability of thrombosis.

From the 1950s to the 1970s there was an increase in understanding of tetralogy of Fallot, with standardization and advancement of surgical repair techniques leading to cardiopulmonary bypass and better postoperative management translating into a better survival rate (85% to 90%) and a decrease in perioperative mortality in the early 1960s, from 60% to 7% to 14%. Until the 1970s, few surgeons performed complete repairs on children younger than 3 to 5 years of age. Bonchek and Starr (1970) concluded that it was beneficial to perform a complete repair earlier in life, preventing worse obstruction by fibrosis and undergrowth of the right ventricular outflow tract. Barrat-Boyes, Kirklin, and Castaneda contributed support for early complete repair in neonates and infants with low mortality.

Currently, early survival after a complete primary repair in large centers is reported to be between 98% and 100%. Despite this, complete primary repair in neonates and infants, in general, is still controversial. Since the 1970s, surgeons have recommended complete repair by 6 months of age and at the latest by 12 months for asymptomatic and non-ductal-dependent infants. Controversy lies between symptomatic neonates undergoing a primary repair versus a two-stage procedure starting with shunt placement. Relative indications for a shunt include those patients with severely hypoplastic pulmonary arteries, anomalous left anterior descenders of a right coronary artery crossing the right ventricular outflow tract, or associated non-cardiac anomalies. Those who support complete repair believe that it promotes normal somatic growth and development, elimination of chronic hypoxemia, improvement of late ventricular function, decreased incidence of late dysrhythmias, and lower risk of hypercyanotic spells and their sequelae. Opponents believe there is a higher incidence of transannular patching and long-term consequences (lung failure), suggesting that a two-stage repair may increase pulmonary valve growth and branch pulmonary arteries, decreasing the chance of a transannular patch. Fraser et al., after trying an individualized surgical strategy, found no significant difference between the primary repair and the two-stage repair in terms of extubation time, length of stay in the intensive care unit, or length of stay. The transannular patch and ventriculotomy began as another therapeutic option for tetralogy of Fallot associated with pulmonary insufficiency and right ventricular dysfunction, presenting with exercise intolerance, ventricular dysrhythmias and a small incidence of sudden death. In 1963, the first transatrial transpulmonary repair was performed by Hudspeth and colleagues. This approach was revisited in the 1990s and is frequently used today across all

age groups, with a survival rate of over 99% and a low incidence of early intervention.

In 1978, a monocuspid valve was described for the first time by Zavanella et al. as an innovation to create competence in the right ventricular outflow tract and decrease or prevent pulmonary insufficiency in a patient requiring a transannular patch. Monocuspid valves made from autologous or bovine pericardium, allograft pulmonary valve cusp, or polytetrafluoroethylene membrane became popular in the 1990s because they reduce lung failure with long-term survival of 98%, no progression greater than moderate failure in 53 % and no reoperation within ten years in 88%. Valved conduits from the right ventricle to the pulmonary artery are used in patients with tetralogy of Fallot and pulmonary atresia or in those who require reoperation due to severe pulmonary insufficiency or recurrent stenosis. Pulmonary or aortic homografts are used in infants with a risk of reoperation of 50% in 5 years and when used in neonates in 3 years. Valved heterografts (bovine jugular vein graft) and autologous valved pericardial conduits are other alternatives.

SHORT- AND LONG-TERM COMPLICATIONS

Common complications in the immediate postoperative period are residual ventricular septal defects, as well as persistent right ventricular outflow obstruction. Arrhythmias may occur after tetralogy repair, with a risk of ventricular tachycardia, atrial fibrillation/flutter, and intra-atrial reentrant tachycardia. The ECG usually appears with a pattern of right bundle branch block or left bundle branch block associated with wide complex tachycardia. Sudden cardiac death can occur in post-repaired patients. Risk factors for tachyarrhythmias and sudden cardiac death include advanced age at repair, male gender, transient complete heart block beyond the third postoperative day, and QRS duration greater than 180 milliseconds.

The prevalence of adult patients with congenital heart disease is increasing by an estimated 5% per year, outpacing the pediatric population. Long-term consequences seen in these patients include right ventricular volume overload from pulmonary insufficiency, right ventricular aneurysm via outflow tract or ventriculotomy, distal pulmonary artery obstruction, ventricular hypertrophy, chamber enlargement, biventricular dysfunction, and dilation and insufficiency of the aortic root (Freitas RA 2019). The three main causes of mortality in patients with repaired tetralogy of Fallot are arrhythmia, heart failure and complications from reoperations. The risk of sudden death increases 30 years after the procedure to 6% to 9%; some of the factors associated with this risk are QRS duration greater than 180 milliseconds, advanced age at repair (greater than 3 years), significant pulmonary valve or tricuspid valve regurgitation, history of syncope, multifocal premature ventricular contractions, and ventricular tachycardia. The most common indication for reoperation is pulmonary insufficiency, and the criteria for pulmonary valve replacement have been based on severity measured by the regurgitant fraction on magnetic resonance imaging or computed tomography. The parameters observed with these studies are the end-systolic and end-diastolic volume indices of the right and left ventricles, ejection fractions and the presence of aneurysm causing obstructive flow. Patients may present with exercise intolerance, signs and symptoms of heart failure, syncope, and sustained ventricular tachycardia. Pulmonary valve replacement can also be achieved by a transcatheter pulmonary valve approach. (Viotto G et al. 2019).

CONCLUSION

Diagnosis and management of Tetralogy of Fallot are performed by an interprofessional team that includes a pediatrician, pediatric cardiologist, cardiac surgeon and radiologist. In general, all children with Tetralogy of Fallot require surgery; time may vary depending on symptoms (Rajpopat AD, Schmidt MR, Søndergaard L. 2019). In the US, most babies born with Tetralogy of Fallot undergo primary repair within the first 12 months of life. Outcomes for patients are good, but after two decades a significant number of them will require pulmonary valve replacement. Contrary to what happened in the past, today there is a percutaneous method of pulmonary valve implantation, but the long-term results are not known. After surgery, most children remain symptom-free. It is important to remember that surgery is not curative, but palliative; structural disease continues to progress at a variable rate.

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