Original Article

The Prevalence of Associated Congenital Cardiac Anomalies (Shunt-Dependent Lesions) in Children with Transposition of Great Artery

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Abstract

Background: Transposition of the great arteries (TGA) is a rare cyanotic heart disease associated with congenital cardiac anomalies; often called shunt-dependent lesions. Transposition of the great arteries is also linked to syndromic correlates. **Aim:** This study was to document the prevalence of TGA and various forms of shunt-dependent lesions in children with TGA. **Methods:** This was a descriptive study where one thousand and five echocardiography were carried out in two health institutions over 9 years. **Results:** Data were analyzed with IBM statistical software version 20. The prevalence of children with TGA was 1.7%. The majority of the infants, 73.3%, had ASD as shunt-dependent lesion. All the infants, 100.0%, presented with cyanosis, breathlessness, and poor weight gain. Less than one-tenth of the infants, 6.7%, had an intact septum. The highest proportion of the infants, 80.0%, has more than one congenital cardiac anomaly (shunt-dependent lesions). Infants with TGA have lower left ventricular mass (LVM) of 119.1 ± 116.5 compared with control (18.8 ± 12.6). This was statistically significant, Mann–Whitney U test (11.024), $P < 0.001$. The under-filled left ventricular mass was found in 73.3% of the infants. Infants with TGA co-existing with Teratology of Fallot (TOF) was (1/15) 6.7%; large Ostium secundum atrial septal defect was (OS ASD) (1/15) 6.7% and cases of ventricular septal defect (VSD) with associated ASD were (1/15) 6.7%. All infants had left aortic arch, which is a normal variant. None (0/15) had any surgical intervention as most were lost to follow-up. **Conclusion:** The prevalence of TGA was low in this setting. There were several shunt-dependent lesions associated with TGA. Atrial septal defect is the commonest. Poor management outcome was due to late presentation.

Keywords: *Children, congenital, congenital heart defect, cyanotic, neonates, TGA*

Introduction

Transposition of the great arteries (TGA), often called complete transposition, is a congenital cardiac disease that occurs when there is atrioventricular concordance and ventriculo-arterial (VA) discordance.[1] The cardiac defect is estimated at 1 in 3,500–5,000 live births, with a male preponderance.[1,2] TGA affects children of all races.[3,4]

TGA is associated with noncardiac malformations and other cardiac malformations (often referred to as shunt/ductal dependent lesions). These lesions include atrial septal defect (ASD), ventricular septal defect (VSD), and left ventricular outflow tract obstruction.[5] The onset and severity of the disease depend on functional and structural variants of these dependent lesions that influence the degree of saturation between the two

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circulations.[6] If no obstructive lesions are seen and there is a large VSD, cyanosis may not be noticed and may be seen clinically when the child cries. In this scenario, signs of congestive heart failure ensue.[6,7] The diagnosis is confirmed by echocardiography, which also provides the morphological details required for future surgical management.[8] Prenatal diagnosis by fetal echocardiography is desirable, as it may improve early neonatal management and reduce morbidity and mortality.[8] Palliative treatment with prostaglandin E1 and balloon atrial septostomy is usually required soon after birth; however, surgical correction is performed later.[9] Usually, the Jatene arterial switch operation is the procedure of choice.[9-13]

There are very few studies on the burden of TGA in sub-Saharan Africa and Nigeria in particular.[14] The descriptive study aimed to document the prevalence of TGA and various forms of shunt-dependent lesions in children with TGA. This work will be a reference point in the future. In our clime, this article is of great importance as it helps to highlight various shunt and ductal-dependent lesions seen in children with TGA. These dependent lesions vary in etiology, clinical approach, and management. The study was done to show the rarity of TGA on the one hand and to harness various clinical and management approaches on the other hand. Besides, this is the first time this study was in this vicinity.

Methods

Study area

This study was carried out in two hospitals in Enugu metropolis, namely the Blessed Children Specialist Hospital, and Triple Care Specialist Hospital. The hospital of study provides general pediatric services and pediatric cardiology services.

Study population

A total of one thousand and five echocardiography was done between 2016 and 2024 among children aged 1 month to 18 years. Children with TGA with or without other related heart defects were included in the study.

Study design

This was a retrospective study that involved two health centers that also served as referral centers for children with cardiac diseases. The infants who had suspected cardiac lesions had echocardiography over a 9-year period. Within this period, infants who had TGA on echocardiography were consecutively recruited. A detailed history and an elaborate clinical examination was done. Those with other cardiac lesions were also seen and managed appropriately.

Echocardiography of TGA

Echocardiography of the infants with TGA was studied and every associated cardiac anomaly was ascertained. Transthoracic echocardiography of the TGA with all the views such as subxiphoid, parasternal, apical, suprasternal, and the modified 4 chamber views, 2-dimensional, color Doppler, pulse wave, continuous wave Doppler, and M-Mode were ascertained. This was in line with the American Society of Echocardiography guidelines.[14] The position of the great arteries was assessed using the sagittal, subxiphoid frontal, and parasternal long-axis view.[14] Multiple views were used to determine the presence and size of the atrial septal defect, ventricular septal defect, and other shunt-dependent lesions. The presence and morphology of left ventricular outflow tract obstruction were determined using the subxiphoid, parasternal long-axis view, and apical five-chamber views. The aortic arch was viewed via a suprasternal sagittal view. The LVM was determined using M-mode.

Ethical consideration

Ethical clearance for the study on congenital heart diseases was obtained from the Research and Ethical Committee of the University of Nigeria Teaching Hospital Ituku-Ozalla.

Results

Table 1 shows the sociodemographic characteristics of the infants. The median age of the infants was 1.5 months. One-third of the infants, 33.3%, were less than 1 month old. A higher proportion of the infants, 66.7%, were male. The prevalence of TGA was 15/1005 (1.5%).

Table 2 shows children with TGA with various shunt and ductal-dependent lesions. The prevalence of children with TGA was 1.7%. The majority of the infants, 73.3% had ASD as shunt-dependent lesion. All the infants, 100.0% presented with cyanosis, breathlessness, and poor weight gain. Less than one-tenth of the infants,

Table 1: Characteristics of the infants

Variable	Frequency (*n*=15)	Percent (%)
Age of infants in months		
Minimum	0.2	
Maximum	11.0	
Mean±SD	1.9±2.7	
Median	1.5	
Age of infants in groups		
<1 month	5	33.3
≥1 month	10	66.7
Gender		
Male	10	66.7
Female	5	33.3

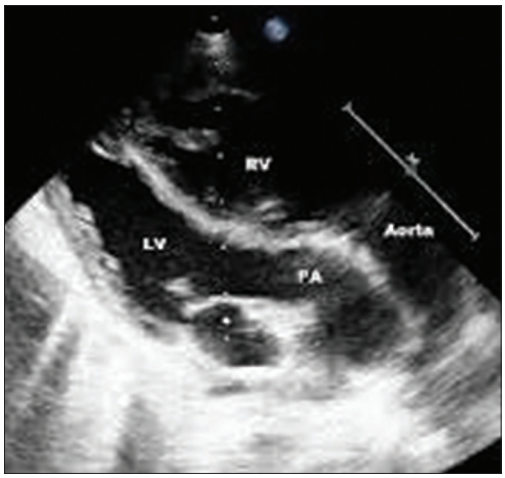

Figure 1: Parasternal long axis view. The left ventricular outflow tract is seen bifurcating into branch pulmonary arteries and has lost its usual tapering

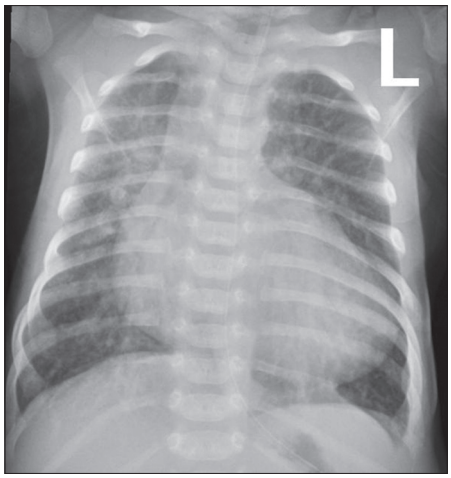

Figure 2: Chest X-ray showing egg-on-stick appearance and a narrow mediastinum

Table 2: Presentation of the infants

Variable	Frequency (*n*=15)	Percent (%)
ASD	11	73.3
PDA	2	13.3
VSD	8	53.3
Tricuspid atresia	1	6.7
Pulmonary stenosis	4	26.7
Pattern of presentation of associated cardiac anomalies		
One associated cardiac defect	3	20.0
Two associated cardiac defect	7	46.7
Three associated cardiac defect	4	26.7
Four associated cardiac defect	1	6.7

6.7%, had an intact septum. The highest proportion of the infants, 80.0%, has more than one congenital cardiac anomaly (shunt-dependent lesions). Infants with TGA have lower left ventricular mass (LVM) of 119.1 ± 116.5 compared with control (18.8 ± 12.6). This was statistically significant, Mann–Whitney U test (11.024), $P < 0.001$. The under-filled left ventricular mass was found in 73.3% of the infants. Infants with TGA co-existing with Teratology of Fallot (TOF) was (1/15) 6.7%; large Ostium secundum atrial septal defect was (OS ASD) (1/15) 6.7% and cases of ventricular septal defect (VSD) with associated ASD was (1/15) 6.7%. All infants had left aortic arch, which is a normal variant. None (0/15) had any surgical intervention as most were lost to follow-up.

Figure 1 shows a parasternal long axis view where the left ventricular outflow tract was seen bifurcating into branch pulmonary arteries and has lost its usual tapering, while Figure 2 showed a chest X-ray with an egg-on-stick appearance and a narrow mediastinum.

Discussion

This study aimed to document the prevalence of TGA and various forms of shunt-dependent lesions in children with TGA.

Among the total number of echocardiography done, TGA was seen in 15 children who were referred for suspected cardiac diseases. The prevalence of TGA in this study was 1.5%. This was lower than the reportage of Ossa Galvis *et al*.[15] who noted a prevalence of 5–7% of all children with congenital heart disease with an overall annual incidence of 20–30 per 100,000 live births.[15,16] Some studies have documented a prevalence rate of 0.2 per 1000 live births.[14,17-19] The low prevalence obtained from this study could be explained by the late presentation of the infants. Besides, the caregivers could not pay for the investigations, medical management, and surgical intervention. Duru *et al*.[20] documented a high burden of out-of-pocket payment among caregivers of children with a congenital heart defect and noted that well-planned health insurance could help avert these malady.[20]

The majority of the children with TGA in this study were mainly seen in the neonatal period and were born at term. Infants with TGA are usually born at term, with cyanosis apparent within a few hours of delivery.[1] We noted no syndromic correlates in our series. This finding is also supported by Unolt *et al*.[21] who noted that though inheritance is multi factorial, it is rarely associated with syndromes or extracardiac malformations.[21]

Among the shunt-dependent lesions, the study showed ASD as the commonest associated lesion seen among children with TGA. This is contrary to the study of Hornung *et al*.[22] who noted VSD as the commonest associated shunt-dependent lesion occurring in almost

50% of cases.[22] In addition, majority of children (80%) with TGA have at least, more than one associated shunt-dependent lesion as seen in this study. These shunt-dependent lesions are very necessary to sustain life. These they do by augmenting oxygenation and tissue perfusion.[22]

Transposition of the great arteries commonly occurs in the male population as seen in this study. Gender differences in congenital heart defects are well known.[14,17-19] A study has noted 60–70% male predominance. A significant bias in sex ratio is known in congenital heart disease with several lesions occurring more frequently in males.[23] This difference may be linked to differences in the hormonal constitution as fetal sex is partially determined by hormone levels of both parents.[24] The literature has shown that children with transposition of the great arteries present with male preponderance, but the reason for this is conjectural. Nevertheless, the male dominance seen in heart lesions with contruncal origin may explain this gender bias.[25]

The mean age of the children with TGA was 1.5 months. This was not similar to the study of Animasahun *et al*.[26] and Adegboye *et al*.[27] in South-western Nigeria who noted a mean age of 6.8 ± 2.4 months. The difference in mean age could be explained by the sample size used in both studies. Late presentation and delayed cardiac surgery timing could also explain the findings above.[28]

All the infants with TGA in this study presented with cyanosis and breathlessness. The cyanosis observed in infants with TGA is due to the parallel circulation seen in the disease; the desaturated blood that returns from the systemic veins flows out of the aorta and the saturated blood that returns from the pulmonary veins flows out of the pulmonary artery.[29] Aggarwal *et al*.[29] in their series noted that the majority of children with TGA presented with cyanosis immediately after birth, especially those with an intact ventricular septum.

Furthermore, progressive cyanosis within the first day of life is the most common finding in infants with TGA if no significant mixing at the atrial level is evident. The commonest clinical signs among infants with TGA seen in this study are the presence of a single heart sound, loud P2, and occasional splitting of the second heart sounds.[30] However, those with large VSD may show signs of pan-systolic murmurs. The loud P2 occurs from the anterior placed aorta.[30]

Most infants with TGA had right aortic arch anatomy, as seen in this study. The combination of TGA with both the right and left aortic arch (double aortic arch) as seen in some studies is conjectural and poses an embryologic surprise.[31,32] The literature has shown that only one patient was described with a double aortic arch associated with transposition of the great arteries.[31] This was in keeping with Edwards's hypothesis of aortic arch development.[32] It is also reported that approximately 10% of cases with TGA have an associated aortic arch abnormality. This is usually coarctation, interruption, or right aortic arch.[33,34]

Coronary anatomy could not be ascertained among children with TGA in the current study. A study has noted that in about 33% of patients with TGA, the coronary artery anatomy was found to be abnormal. The commonest coronary artery abnormality is the left circumflex coronary arising from the right coronary artery in one third of cases.[35]

The under-filled left ventricle is an important finding noted in children with TGA in the current study. This study showed under-filled left ventricle in 6.7% of infants with TGA coexisting with TOF, 6.7% of infants with large OS ASD, and 6.7%, cases of VSD with associated ASD. This could be explained by loading conditions which alter both the left and the right ventricle geometry. Left ventricular regression is characterized by a crescent/banana-shaped left ventricle, bulging of interventricular septum toward the left ventricle with paradoxical septal motion of the right ventricle.

It is important to note from this study that infants with TGA have lower left ventricular mass when compared with those with no cardiac lesions/control. Those with large shunt defects were found to have preserved LV mass compared with those with intact septum. The actual time when the LV under-fills and loses sustenance to systemic function is not known.[29] However, LV mass remains preserved in certain conditions. These include the presence of large VSD, obstruction of LV outflow, large PDA, unregressed pulmonary vascular resistance PVR, or major aorto-pulmonary collateral.[29] On the contrary, in a large isolated ASD, LV mass is poorly preserved especially when the PVR falls after the 8th to 12th week of life.[29] This could be explained by the paucity of inflow to the left ventricle.[29]

About two patients died while others were all lost to follow up and may have died at home. The reason for the loss of follow-up was because the caregivers could not afford definitive surgical intervention. The mortality rate of children presenting with TGA is very high. For instance, it is documented that more than 90% of children with TGA died in infancy, while a few will die at about 14 months of age.[36] However, after surgical intervention with no other underlying risk factors, the life expectancy after surgery is high. Animasahun *et al*.[26] also noted a mortality rate of 90% in their reportage.

The mortality rate from TGA is very high especially if they present with intact septum. Survival is based on rapid diagnosis and timely intervention. Literature has documented a very high mortality rate of 97% if left untreated.[30] However, surgery in the first week of life reduces morbidity and mortality, improving the 20-year survival to more than 90%.[37-41] Prenatal vigilant surveillance is prime for children with known risk factors to reduce the burden of the disease.

The current study did not show any link between TGA and consanguinity. However, Villafañe *et al*.[41] noted that consanguinity increases TGA's odds ratio by nearly 3-fold and that the prevalence of consanguinity in families with TGA was 46% compared with 20% seen in the control group. This study also was in keeping with that of Nabulsi *et al*.[42] whose study found no significant association between consanguinity and TGA. This zero prevalence of consanguinity seen in this study is possibly due to the rarity of consanguineous marriages in the region of study.

The main stay of diagnosis of children with TGA is echocardiography. Parasternal long axis view shows loss of tapering of the left ventricular outflow tract while the short axis view reveals the aorta being placed right and anterior to the pulmonary artery with the tricuspid valve maintaining its cephalad position to the mitral valve, a very important difference from congenital corrected transposition of the great artery (CCTGA), where there is total loss of the cephalad nature of the tricuspid valve. Chest radiograph shows the typical egg-on-string appearance and an apparent narrowing of the superior mediastinum. This is as a result of the main pulmonary artery being posterior to the aorta.

Our series received an antifailure regimen which includes angiotensin-converting enzyme inhibitors and high ceiling diuretics doses. None of our infants had surgical intervention.

Limitation

The main limitation of this study is that it was a retrospective study. A prospective cohort where children with TGA are followed over time with respect to clinical progression and management outcome will be worthwhile.

Conclusion

The prevalence of TGA is low in this setting. There are several shunt-dependent lesions associated with TGA, atrial septal defect being the commonest. Poor outcome was due to late presentation and loss to follow-up.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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