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A Case Of Congenitally Corrected Transposition Of Great Arteries Of Elderly Male: A Rare Event

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ABSTRACT

Background:
Congenitally corrected transposition of the great arteries (CCTGA) is a rare congenital cardiac anomaly, accounting for less than 1% of all congenital heart defects. It features both atrioventricular and ventriculoarterial discordance, resulting in physiologically corrected circulation. Despite this functional correction, the morphologic right ventricle supports systemic circulation, predisposing patients to progressive ventricular dysfunction, tricuspid valve regurgitation, and conduction system abnormalities. While often diagnosed in childhood when associated with other cardiac anomalies, isolated CCTGA may remain clinically silent and undiagnosed until adulthood or even late life.

Case Presentation:
We report the case of a 53-year-old male who presented with a 7-month history of breathlessness. He was nonhypertensive, nondiabetic, and afebrile with stable vital signs. Clinical examination revealed parasternal heave, a loud second heart sound, and a diastolic murmur at the tricuspid area. Transthoracic echocardiography revealed classic features of CCTGA, including atrioventricular and ventriculoarterial discordance, global left ventricular hypokinesia, moderate systolic dysfunction with an ejection fraction of 35–40%, and severe tricuspid regurgitation. Electrocardiogram findings showed evidence of atrioventricular conduction abnormalities. The patient was managed conservatively with diuretics, ACE inhibitors, beta-blockers, nebulization, and supportive therapy. He responded well to medical management and was discharged in a stable condition with outpatient follow-up.

Conclusion:
This case emphasizes the importance of considering congenital heart diseases like CCTGA in adult patients presenting with nonspecific cardiac symptoms. Elderly patients with asymptomatic or minimally symptomatic CCTGA may remain undiagnosed for years. Individualized medical management and routine follow-up are essential for optimizing outcomes and monitoring for progressive ventricular dysfunction or arrhythmias.

INTRODUCTION

Congenitally corrected transposition of the great arteries (CCTGA) is an uncommon congenital cardiac anomaly, accounting for less than 1% of all congenital heart diseases. This condition is marked by a distinctive anatomic arrangement wherein both the atrioventricular and ventriculoarterial connections are reversed. In simpler terms, the right atrium connects to a morphologic left ventricle, which then directs blood into the pulmonary artery, while the left atrium connects to a morphologic right ventricle that pumps blood into the aorta. Despite these unusual connections, the circulatory pattern remains functionally normal, hence the term "congenitally corrected." However, the systemic circulation is maintained by a morphologic right ventricle, a structure not ideally designed to withstand high-pressure systemic workload for a lifetime, raising the risk of gradual deterioration over time [1, 2].

Many individuals with CCTGA may remain undiagnosed for years, especially if no associated anomalies are present. The absence of early cyanosis or heart murmurs can contribute to the delayed identification of the condition. In numerous cases, diagnosis only occurs when symptoms such as breathlessness, fatigue, palpitations, or chest discomfort begin to emerge, typically in adulthood. In some exceptional instances, the condition remains completely asymptomatic until it is incidentally discovered during imaging for unrelated complaints. While pediatric presentations of CCTGA are more frequently reported due to associated cardiac defects like ventricular septal defects or pulmonary stenosis, isolated or uncomplicated CCTGA might only come to light in middle or late adulthood. With advancing age, the morphologic right ventricle, which has long been performing the role of the systemic ventricle may begin to exhibit signs of failure. Moreover, the systemic atrioventricular valve, namely the tricuspid valve, may undergo regurgitation due to prolonged exposure to high pressures, leading to symptoms of right-sided heart failure [3, 4].

In addition to mechanical strain, the abnormal spatial positioning of the conduction tissue in CCTGA makes affected individuals particularly susceptible to rhythm disturbances. A considerable proportion of adults with this condition experience conduction system abnormalities, including atrioventricular block and complete heart block, often necessitating pacemaker insertion. The location of the conduction pathways in CCTGA is often displaced, rendering them vulnerable to fibrosis and degeneration with age. As a result, elderly patients may present with syncope, bradycardia, or even sudden cardiac events if such conduction abnormalities go unnoticed. Furthermore, the structural disarray associated with this condition can predispose patients to supraventricular arrhythmias such as atrial fibrillation or flutter, especially in the setting of right atrial enlargement or ventricular dysfunction [5, 6].

Despite these potential complications, some patients with isolated CCTGA exhibit remarkable compensatory adaptation, remaining largely symptom-free for decades. In such individuals, early diagnosis is typically missed, as clinical findings may be subtle or absent. Our case involves an elderly male who had never been diagnosed with a cardiac anomaly previously but presented with nonspecific symptoms like breathlessness. His clinical evaluation, including echocardiographic imaging, revealed the presence of CCTGA along with significant global hypokinesia of the left ventricle, moderate systolic dysfunction, and severe tricuspid regurgitation. The ejection fraction was found to be in the range of 35–40%, indicating compromised ventricular contractility. The absence of prior cardiac complaints in this patient highlights the capacity of some CCTGA cases to remain latent for many years, particularly when associated defects are minimal or absent [7, 8].

The delayed diagnosis in this elderly male also emphasizes the importance of thorough cardiovascular evaluation in patients with unexplained dyspnea or fatigue, even in the absence of overt signs of structural heart disease. In such cases, transthoracic echocardiography often plays a pivotal role in identifying anatomical discordances and assessing ventricular function. Advanced imaging modalities like cardiac MRI may further delineate the ventricular morphology, detect myocardial fibrosis, and evaluate the condition of valves and great arteries. In our patient, conservative medical management was employed, including diuretics to manage fluid overload, ACE inhibitors and beta-blockers to support ventricular function, and nebulization to relieve respiratory symptoms. Given his age and the absence of significant cyanosis or other congenital anomalies, surgical correction or anatomical repair was not deemed necessary at this stage [9].

Management strategies in elderly individuals with CCTGA need to be individualized, taking into account the patient's age, comorbidities, symptom burden, and overall cardiac function. While surgical correction, such as double-switch procedures is commonly considered in younger patients with associated defects or early signs of systemic ventricular failure, such interventions carry higher risks in elderly patients and are often avoided unless clearly indicated. Instead, treatment is aimed at optimizing cardiac performance, controlling arrhythmias, and alleviating symptoms. The focus is also on regular follow-up to monitor progression of tricuspid regurgitation, systemic ventricular function, and potential emergence of conduction abnormalities. Periodic ECG and echocardiographic evaluations are thus essential in the long-term care of such patients [9, 10].

The occurrence of asymptomatic or minimally symptomatic congenitally corrected transposition of the great arteries (CCTGA) in elderly individuals is rare and highlights the need for heightened clinical awareness. This case, diagnosed in late adulthood, reflects the wide variability in CCTGA presentation and demonstrates that patients with favorable anatomical features may remain stable for decades. However, age-related cardiac decline can eventually lead to decompensation. The case underscores the importance of including congenital anomalies in the differential diagnosis of unexplained cardiac symptoms in older adults and illustrates how advanced imaging and thorough clinical evaluation are crucial for timely diagnosis and optimal management [11].

CASE DESCRIPTION

A 53-year-old conscious male presented to the Department of General Medicine with a complaint of shortness of breath for 7 months. He was nonhypertensive, nondiabetic and afebrile. His biochemical, haematological and serological examinations were normal. His vitals including blood pressure were 120/70 mm of Hg with pulse of 50/min and respiratory rate of 18/min. Epigastric pulsation was visible. Apex pulse seen in -6th ICS with parasternal heave grade III+. There was S2 loud and diastolic murmur heard in tricuspid area. R/S air entry B/L equal and B/L Wheeze+ was reported. Rest systemic examination was found to be within normal limits.

2D echocardiogram findings were suggestive of CCTGA with global LV hypokinesia, severe tricuspid regurgitation (TR), and

moderate LV systolic dysfunction with an ejection fraction between 35–40%. As shown in the echocardiographic images (**Figures 1a, 1b, and 1c**), the aorta arises from the right ventricle and the pulmonary artery from the left ventricle, demonstrating ventriculoarterial discordance. These findings are consistent with the diagnosis of CCTGA. (Table 1).

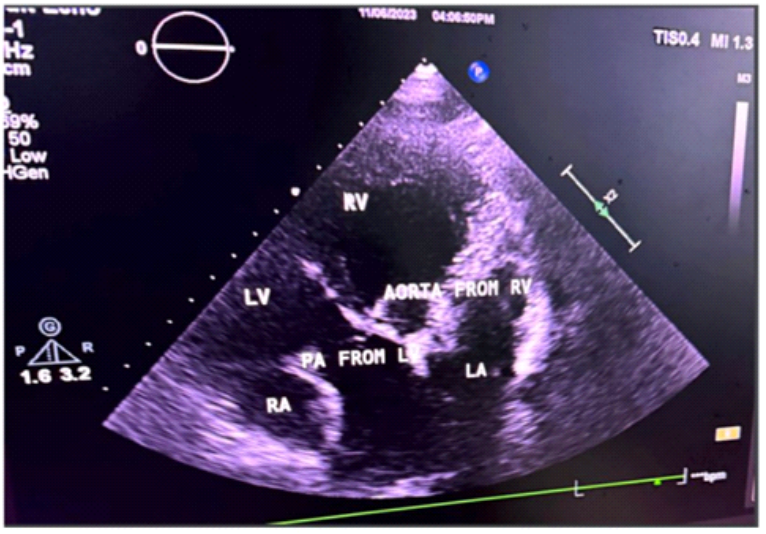

Figure 1a: 2D echocardiographic image showing the aorta arising from the right ventricle (RV) and pulmonary artery from the left ventricle (LV).

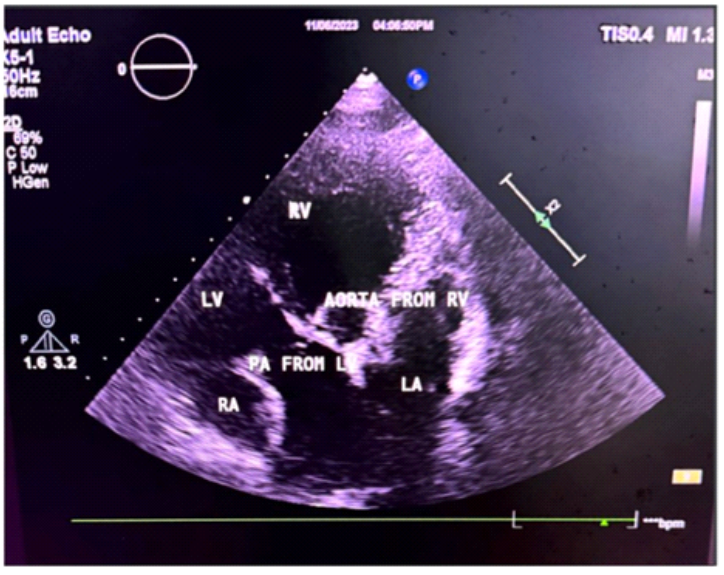

Figure 1b: Echocardiographic cross-section highlighting atrioventricular and ventriculoarterial discordance.

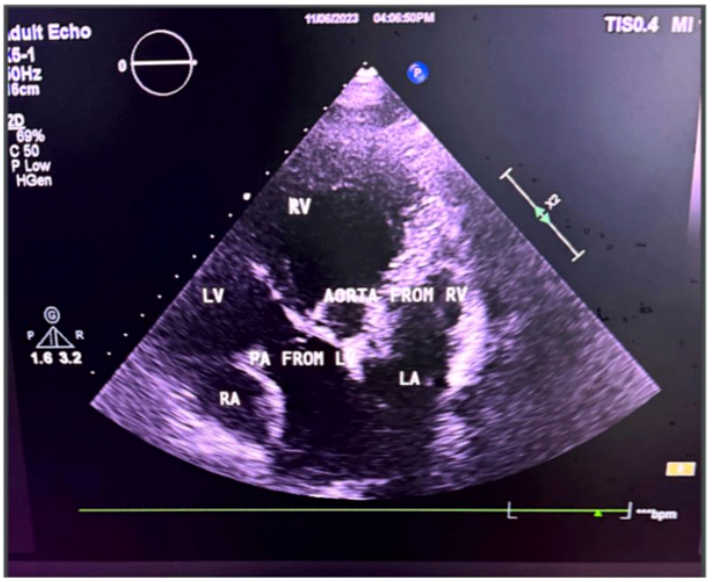

Figure 1c: Clear labeling of cardiac chambers and great vessel malposition in CCTGA.

Table 1: 2D Echo Findings of the Case Patient

DIMENSIONS	VALUE	NORMAL RANGE
AO (ed)	2.40 cm	(2.1-3.7)
LA (ES)	4.50 cm	(2.1-3.7)
RVID (Ed)	4.83 cm	(1.1-2.5)
LVID (ED)	4.88 cm	(3.5-5.2)
LVID (es)	2.88 cm	(2.3-3.2)
IVS (ed)	1.08 cm	(0.6-1.2)
LVPW (ed)	1.11 cm	(0.6-1.2)
LVEF	35%	(55%-85%)
FS	17%	(28%-42%)

Electrocardiogram findings were also correlated with the echo findings suggestive of LV systolic dysfunction with severe TR (Figure 2).

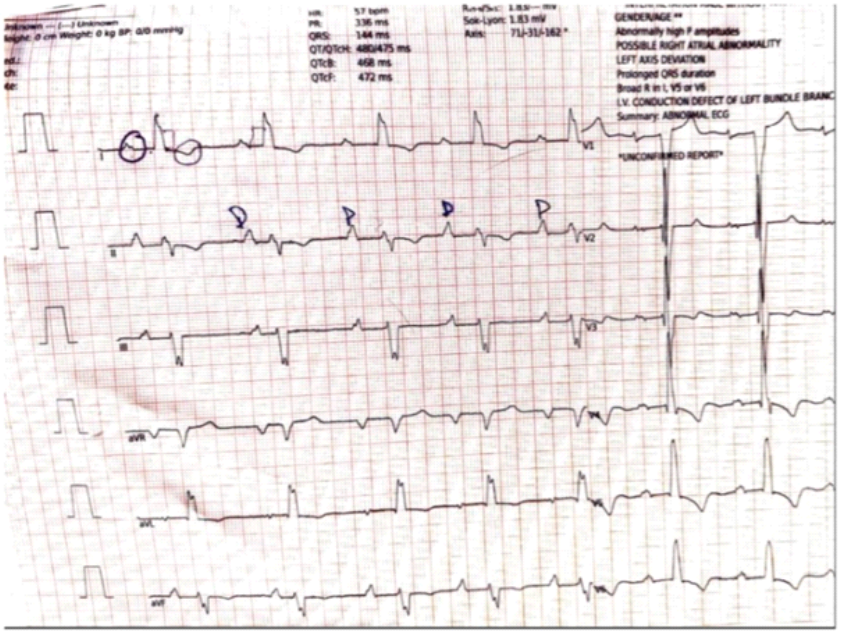

Figure 2: Electrocardiogram interpreting AV block and low heart rate.

The patient was medically managed with diuretics, ace inhibitors, beta-blockers, nebulization and other supportive treatments. At discharge, the patient was asymptomatic without evidence of any breathing complications. Oral medication was suggested to the patient with the advice of follow-up after day 7th in OPD.

DISCUSSION

Rare conditions of Congenitally corrected transposition of the great arteries (CCTGA) ensue lacking any known anomalies however sometimes accompanied by associated complications. In our case, the elderly male patient was asymptomatic and reported only breathing issues and was later unpremeditatedly diagnosed with the presence of Congenitally corrected transposition of the great arteries (CCTGA) when he attended the Department of General Medicine in a conscious state. Furthermore, haematological, biochemical and vitals were found to be within normal limits. 2D echo cardiogram findings later revealed global LV hypokinesia with severe TR, and moderate LV systolic dysfunction along EF between 35-40%. Symptoms like easy fatiguabillity , dyspnea and palpitations are associated with the TR in CCTGA as also reported by another previously published study [12].

In our case, the patient was investigated and diagnosed with Congenitally corrected transposition of the great arteries (CCTGA) during the routine cardiac investigation. Similar findings were observed by authors of the previously published study in which many patients' attending hospital with CCTGA are asymptomatic and diagnosed during routine cardiac evaluation on chest X-ray or ECG [13]. In our case we observed severe TR, however, the same observation was reported previously in CCTGA patients '[14].

Asymptomatic LV systolic dysfunction (ALVSD) was reported in our case and our observation was supported by a previously published study [15]. It was depicted that if CCTGA was not diagnosed antenatally without any known adverse cardiac events, there is a high possibility that the patient remains asymptomatic until their fourth or fifth decade [1]. It was proven that in post fifth decade, the patient is at risk of developing LV dysfunction and congestive heart failure despite a lack of associated abnormalities [16]. The progression of LV dysfunction and tricuspid insufficiency were known to be associated with significantly increased mortality in CCTGA patients. The precise mechanism of eventual LV dysfunction still needs to be investigated in detail concerning physiologic and anatomic factors that are known to be responsible for its pathogenesis. Some of the known mechanisms include coronary artery blood flow, arrhythmias, conduction irregularities and left ventricle dysfunction [17]. It is unclear, how these pathobiological contributors initiate the progression of LV dysfunction development and allow CCTGA patients to be asymptomatic until their eighth or ninth decades.

It is estimated that among CCTGA patients 20-50% showed moderate to severe TR contributing to substantial morbidity and mortality. This can be seen as a major cause of ventricular dysfunction as occurred in our case. Progressing age can contribute to the development of atrial fibrillation and flutter as shown by previously published studies, but in contrast, we found lower heart rate in our case [12].

In a recent finding one of the previously published observed acute MI in CCTGA patients with systolic dysfunction and EF of 30% . The same observation with EF between 35-40% was observed in our case and supported by findings of previously published study [18]. However, in our case, the patient did not present with any adverse cardiac event.

CONCLUSION

Asymptomatic congenitally corrected transposition of the great arteries (CCTGA) is an exceptionally rare finding, particularly in a 53-year-old male. The patient presented with breathing difficulty and was conscious, leading to echocardiographic evaluation that revealed CCTGA with global left ventricular hypokinesia, severe tricuspid regurgitation, and moderate systolic dysfunction with an ejection fraction of 35–40%. He was effectively managed with medications such as diuretics, ACE inhibitors, beta-blockers, nebulization, and supportive care. This case highlights the importance of further clinical correlation to assess non-surgical management options for asymptomatic patients and to explore long-term outcomes without anatomical or physiological correction.

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