CASE REPORT

Cor triatriatum and stroke

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SUMMARY

Cor triatriatum sinistrum (CTS) is a congenital anomaly where the left atrium is divided into two compartments by a fibromuscular membrane. This report aims to add to the literature on a rare cardiac condition that can cause neurological morbidity. We report a case of a 19-year-old female with an infarct in the right middle cerebral artery (MCA) territory initially maintained on aspirin. Eighteen months later, she had recurrence of weakness, for which repeat transthoracic echocardiography (TTE) and re-evaluation of the first TTE demonstrated a hyperechoic membrane spanning the width of the left atrium, clinching the diagnosis of CTS. Despite anticoagulation with apixaban, she was admitted for a third stroke where she succumbed to hospital-acquired pneumonia. Among cases of CTS associated with stroke, anticoagulation and surgery were the main modes of treatment. This case has the longest follow-up and the first to demonstrate failure of antiplatelet therapy and anticoagulation.

BACKGROUND

Cor triatriatum sinistrum (CTS) is a congenital anomaly where the left atrium is divided into two distinct compartments by a fibromuscular membrane. The superior chamber receives venous blood, whereas the inferior chamber remains in contact with the mitral valve and contains the appendage.^{1,2} Only about 0.1% of congenital heart diseases are attributable to CTS.^{1,3,4} CTS may result in other abnormalities such as mitral regurgitation and left atrial enlargement.⁵

The anomalous membrane may or may not have fenestrations connecting the two chambers and is considered the basis for classification as proposed by Loeffler in 1949.^{3,4} The three theories of malseptation, malincorporation and entrapment as aetiologies for CTS are briefly discussed later on.⁶ The disease commonly presents with symptoms of exertional dyspnoea, orthopnoea and haemoptysis, but may also be asymptomatic until adulthood. Apical murmur and pulmonary congestion are not uncommon findings in symptomatic patients.^{2,6}

Among patients with adult congenital heart disease, stroke incidence is considerably higher than the general population. This may be due to paradoxical embolisation, prior surgical intervention, infective endocarditis or concomitant acquired risk factors.⁶ Worldwide, there have been eight published reports of CTS associated with stroke; no such case has been reported in the Philippines. Explored treatments for the disease include surgery

and anticoagulation—both of which carry a significant possibility of morbidity. Due to lack of available published guidelines on the management of stroke in patients with CTS, a review of management and outcomes of reported cases is vital.

This report follows the case of a young adult who had cardioembolic stroke associated with cor triatriatum.

CASE PRESENTATION

A 19-year-old female of excellent functional capacity, able to do extended activities of daily living, came into the outpatient department for sudden onset left extremity weakness and facial asymmetry occurring 10 days prior to consult. She had no known comorbid conditions such as heart failure, hypertension, dyslipidaemia or congenital heart disease. Her family history was not significant for stroke, coronary artery disease, structural cardiac disease, congenital heart disease, hypertension, diabetes mellitus and dyslipidaemia. Her blood pressure on examination was 90/60 mm Hg on all four extremities with a normal heart rate of regular rhythm. Physical examination revealed no neck vein engorgement, clear breath sounds, no heaves, murmurs or thrills and an apex beat heard loudest in the fifth intercostal space at the midclavicular line. She was alert, following commands, with a circumductive gait. She had left-sided central facial palsy, grade 4/5 hemiparesis, decreased pain sensation and hyperreflexia.

The initial cranial CT scan done 2 weeks post-ictus (figure 1A) revealed a very subtle hypodensity on the right middle cerebral artery territory signifying an infarct of the corona radiata. A cranial MRI confirmed the finding of a subacute subcortical infarct in the aforementioned territory (figure 1B). MR venography was unremarkable; however, MR angiography showed decreased calibre of the right internal carotid, middle cerebral artery and vertebral arteries (figure 1C). Carotid Doppler studies could not appreciate flow in the right carotid artery. Four vessel catheter angiography confirmed MR angiography findings of non-visualised innominate artery, right common, internal and external carotid arteries, which were deemed to be congenital in origin at that time (figure 1E). A CT aortogram concurred with the findings of the angiography; furthermore, it demonstrated the lack of inflammatory changes related to aortitis (figure 2A). Takayasu arteritis was considered;

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Figure 1 (A) Cranial CT scan 2 weeks post-ictus showing CT fogging (white arrow). (B) Cranial MRI 3 months post-ictus confirming the presence of an infarct (white arrow). (C) Cranial MR angiography showing collaterals from the circle of Willis supplying the right side. (D) Cranial CT scan on readmission for new onset weakness about 18 months after the initial stroke. (E) Four vessel angiography confirming the findings of the cranial MR angiography.

Figure 2 (A) Transthoracic echocardiography done in the parasternal long axis view, showing cor triatriatum sinistrum (CTS), with an arrow pointing at the dividing membrane. (B) Transthoracic echocardiography done in the 4-Chamber (4C) view, showing the CTS. (C) Colour Doppler study done in the 4C view, demonstrating flow from the ULA into the LLA. ULA, upper left atrium; LLA, left lower atrium; RA, right atrium; RV, right ventricle; LV, left ventricle.

however, due to the lack of inflammatory findings on the CT aortogram, it was deemed less likely. Her prothrombin time (12.2 s), partial thromboplastin time (29.6 s), platelet count ($452 \times 10^9/L$) and haematocrit (0.37) were all normal. Assay for serum antinuclear antibodies was equivocal.

The baseline ECG was unremarkable except for the finding of poor R wave progression. A two-dimensional transthoracic echocardiography (TTE) revealed findings consistent with mitral valve sclerosis with mild mitral regurgitation, tricuspid valve sclerosis with mild tricuspid regurgitation (vena contracta 1.7 mm) and an ejection fraction of 76%. Twenty-four hour Holter monitoring done documented no episodes of arrhythmia. She was managed as a case of rheumatic heart disease and maintained on aspirin 80 mg per tablet once a day and benzathine penicillin injections. With rehabilitation over the next year, her deficits were reduced to a subtle drift on the left upper extremity and she was able to return to work as a sales clerk.

Eighteen months after her initial stroke, however, she presented at the emergency department with sudden onset left-sided weakness with a motor strength grade of 4/5. A repeat TTE demonstrated a previously undocumented hyperechoic membrane spanning the width of a normal-sized left atrium (LA), dividing the LA into two communicating chambers. The membrane was noted to be immobile during atrial systole and atrial diastole. The Doppler study demonstrated flow through this membrane. These findings were suggestive of CTS. No evidence of an intracardiac thrombus or rheologic stasis was found, and no significant regurgitant jets or pressure gradients were demonstrated across the membrane. Other findings in the study were concentric remodelling of the left ventricle, with adequate wall motion and contractility, and a mild mitral regurgitation. (figure 2A–C) Anti-coagulation was started. Because there was no consent for warfarin, she was sent home on apixaban (5 mg per tablet twice a day) instead. Unfortunately, she failed to go to her scheduled transoesophageal echo (TEE) and bubble contrast study.

About 19 months after the initial stroke, she had sudden onset right-sided weakness with the absence of verbal output. She was admitted at a different institution for 5 days where she developed hospital-acquired pneumonia. On transfer to our hospital, she was found highly febrile with crackles on her mid lung fields. She was awake without regard, globally aphasic, localising to pain with the left extremities and withdrawing to pain with the right extremities. A cranial CT scan showed a subacute infarct on the left middle cerebral artery territory in the cortical subcortical areas. (figure 1D) A repeat TTE showed the same hyperechoic membrane dividing the left atrium into two separate, communicating chambers (see online supplementary video). No evidence of thrombus or stasis was found, and no significant change in the previously noted left ventricular concentric remodelling and mitral regurgitation. During admission, she was started on warfarin; however, prior to achieving the therapeutic international normalised ratio she developed upper gastrointestinal bleeding. Despite maximal efforts, she succumbed to septic shock on the 12th hospital day. A scheduled TEE was no longer done.

Her postmortem imaging was reviewed by a neuroradiologist and a neurointervention specialist. The absence of the brachiocephalic artery, common carotid artery and internal carotid artery was deemed likely to be from embolic obstruction rather than agenesis due to the presence of a brachiocephalic artery stump (figure 3).

Figure 3 Oblique view of the CT aortogram showing no signs of inflammation; the proximal portions of the left common carotid artery, left subclavian artery and remaining stump of the brachiocephalic artery are also demonstrated.

OUTCOME AND FOLLOW-UP

Unfortunately, the patient expired after aspiration pneumonia, a complication of her last stroke, led to septic shock.

DISCUSSION

The cardiac primordium arises from splanchnic mesoderm in a cardiogenic region of the trilaminar embryo, where angioblastic cords develop and canalise to form bilateral endocardial heart tubes. Lateral folding of the embryo during development joins the bilateral heart tubes, producing a single primordial heart tube.⁷ This single heart tube elongates and undergoes looping, which allows for partitioning of the primitive heart into its four definitive cardiac chambers and vascular trunks.

The mature atria have two major origins. The atrial appendages (trabeculated portions) are derived from the atrial component of the primary heart tube, which balloon from either side of the outflow tract. The smooth-walled posterior portions of the atria are formed by the incorporation of the pulmonary veins on the left chamber and the sinus venosus on the right. Atrial septation begins with a membranous septum primum, dividing the common atrium into right and left chambers and is replaced by a muscular septum secundum around the fifth and sixth week of gestation.^{7,8}

Three schools of thought have been proposed regarding the anomalous presence of a fibromuscular band dividing the atrium into more than one chamber as seen in CTS. One theory suggests

an anomaly with atrial septation, whereas the two others suggest abnormalities with vascular incorporation into the cardiac chambers. The malseptation theory hypothesises that CTS is due to an anomalous growth of the septum primum. The theory of malincorporation suggests an incomplete incorporation of the common pulmonary vein into the left atrium, whereas the theory of entrapment postulates an anatomic entanglement of the pulmonary vein by the primitive horn of the sinus venosus, preventing its incorporation into the left atrium.^{3,9–11}

This is the ninth reported case of cor triatriatum associated with a cerebrovascular infarct. The previous cases are summarised in table 1. The mean age was 50 years, with a wide range of 17–70 years old. Interestingly, all but two of the patients were female. The strokes in those with available intracranial imaging spanned cortical and subcortical regions and included both anterior and posterior circulations. Three of the cases involved multiple infarcts. These morphological features are compatible with cardioembolic infarcts.¹² Similarly, our patient's neuroimaging exhibited these two features. Besides cor triatriatum, other risk factors for stroke were also identified in the cases: hypertension (2, 3, 6, 8), atrial fibrillation (2, 5, 6, 8), hyperlipidaemia (2, 8) and diabetes mellitus (8). Only three of the cases (1, 4, 7) did not have traditional risk factors associated with stroke.¹³

TEE is the imaging of choice for identifying cardiac embolic source in patients with stroke without pre-existing indication or contraindication to oral anticoagulation and is able to detect about 40% of cardiac embolic sources in patients with normal TTE, regardless of age.¹⁴ All the reported cases except for 1 and 7 had an echocardiogram that demonstrated either spontaneous echo contrast (SEC) or an intracardiac thrombus. SEC described as 'dynamic smoke-like echoes with distinct swirling motion within the left atrial cavity' was deemed to correlate well with the presence of left atrial thrombus in patients with rheumatic mitral valve disease.¹⁵ SEC representing red cell aggregation is related not only to stasis but is also associated with a hypercoagulable state, which is present in almost all cases of left atrial thrombus.¹⁶ The ability of the TEE to see posterior heart structures makes it the imaging of choice for detecting left atrial thrombi. TEE has a 100% sensitivity for left atrium thrombi, while TTE only has 35%–69% sensitivity.¹⁷ Unfortunately, our patient was not able to undergo TEE because she failed to show for her appointment. Despite our patient not having evidence of stasis or thrombus in the TTE done, a TEE may have been able to exhibit those with greater sensitivity. She would have been started on anticoagulation much earlier had SEC or thrombus been identified.

The patient's initial TTE was not able to detect CTS but instead found mitral regurgitation. There are reports on the co-occurrence of mitral regurgitation and CTS.^{18,19} CTS, however, is more often misdiagnosed as mitral stenosis (MS). The clinical presentation and haemodynamics of CTS may mimic MS. The continuous flow through CTS orifice and the dividing septum are some distinguishing characteristics of CTS.² Similar to MS, the size of the communicating orifice influences the clinical symptoms of patients with CTS. With the exception of case 6, none of the patients in the review and our own patient had any cardiopulmonary symptoms until they had their stroke. This emphasises that the clinical suspicion for CTS should be present even in those without cardiac symptoms.

There are no existing guidelines for the treatment of CTS. Three of the reviewed cases (2, 7, 8) underwent excision of the dividing septum. Of these, one was not maintained on any form of anticoagulation. In a series of 25 cases who underwent resection of membrane for CTS from the Mayo Clinic, survival rate

Table 1 Cor triatriatum and stroke: reported cases

Author, patient age and sex	Cranial imaging	Cardiac imaging	Treatment	Outcome	Additional information
Ridjab <i>et al</i> ²¹ 44/F	Infarct bilateral MCA and right PICA territory	TTE+TEE (-) Thrombus (-) Spontaneous echo contrast	Unspecified anticoagulation	No adequate follow-up; case report ended with diagnosis	(-) Comorbidities (-) Antithrombin III deficiency (-) Protein S deficiency (-) Protein C deficiency (-) Plasminogen deficiency (-) Activated protein C resistance (-) Prothrombin II GA 20210 mutation (-) Anticardiolipin antibodies (-) Atrial fibrillation by 24 hours Holter
Spengos <i>et al</i> ²² 55/M	Infarct left MCA territory cortical-subcortical	TEE+MRI (+) Thrombus (+) Dilated left atrium (+) Left ventricular hypertrophy	Unspecified anticoagulation and total membrane excision	Follow-up time frame not specified; improved over weeks	(+) Hypertension (+) Hyperlipidaemia (+) Atrial fibrillation No haematologic work-up
Patrascu <i>et al</i> ²³ 71/F	Infarct left MCA territory cortical-subcortical (parietal lobe, basal ganglia, internal capsule)	TTE+TEE (-) Thrombus (+) Atrial septal aneurysm (+) Spontaneous echo contrast	Warfarin anticoagulation	Follow-up time frame not specified; underwent rehabilitation and improved	(+) Hypertension (-) Activated protein C resistance (-) Prothrombin mutation (-) Lupus anticoagulant (-) Hyperhomocystinaemia (-) Atrial fibrillation: 72 hours Holter (-) Carotid Doppler studies
Park <i>et al</i> ²⁴ 55/F	Infarct right PCA territory cortical-subcortical (occipital lobe)	TTE + TEE (-) Thrombus (+) Spontaneous echo contrast	Unspecified anticoagulation	Follow-up time frame not specified; no recurrence of stroke	(-) Comorbidities (-) Cranial MR angiography (-) Carotid Doppler studies (-) Hyperhomocystinaemia (-) Antithrombin III deficiency (-) Protein S deficiency (-) Protein C deficiency (-) Antiphospholipid antibodies
Minocha <i>et al</i> ²⁵ 17/M	Infarct right MCA territory cortical-subcortical (parietal lobe)	TTE+TEE (+) Thrombus (+) Spontaneous echo contrast	Anticoagulation	Follow-up time frame not specified; no recurrence of stroke	(+) Atrial fibrillation (+) Dilated atrial shadow on chest X-ray (-) Antithrombin III deficiency (-) Protein S deficiency (-) Protein C deficiency (-) Antiphospholipid antibodies
Huang <i>et al</i> ²⁶ 65/F	Brain swelling	TTE+TEE (+) Mitral regurgitation (-) Thrombus (+) Spontaneous echo contrast (+) Dilated left atrium	Embolectomy of aortic thrombus; no anticoagulation	Expired from circulatory and central failure	(+) Exertional dyspnoea (+) Hypertension (+) Atrial fibrillation by routine 12-lead ECG (+) Total occlusion of the aorta at the second lumbar vertebra
Krasemann <i>et al</i> ²⁷ 39/F	Left-sided stroke	TTE+TEE (+) Atrial septal defect (-) Thrombus	Resection of membrane and patch on atrial septal defect	Follow-up time frame not specified; no recurrence of stroke	Other work-up not specified
Nishimoto <i>et al</i> ²⁸ <i>Translated from Japanese</i> 57/F	Infarct right PCA territory cortical-subcortical (thalamus and occipital lobe), left MCA territory subcortical (putamen)	TTE+TEE (+) Multiple thrombi in the left atrium	Medical therapy with anticoagulation and surgical therapy with clot resection of membrane	Follow-up time frame not specified; no recurrence of stroke	(+) Atrial fibrillation (+) Hypertension (+) Diabetes mellitus (+) Hyperlipidaemia (+) Metastatic brain tumour (+) Multiple occlusions on coronary angiography (+) Renal artery and popliteal artery occlusion

MCA, middle cerebral artery; PCA, posterior cerebral artery; PICA, posterior inferior cerebellar artery; TTE, transthoracic echocardiography; TEE, transesophageal echocardiograph.

was found to be 88% while 72% of patients were still New York Heart Association Functional Class 1 at the time of follow-up 12 years after the operation. Early mortality was not associated with the procedure. There was no mention of stroke in any of the patients.³

All of the cases reviewed except cases 6 and 7 were treated with anticoagulation. Even case 1 who had no SEC or thrombus was anticoagulated. The method of anticoagulation, however, was not specified except for case 3. None of the patients anticoagulated were reported to have recurrence of stroke. One mortality from circulatory and central failure, in one of the cases who underwent neither membrane excision nor anti-coagulation, was documented. Exact follow-up time was not specified.

Despite anticoagulation with apixaban, our patient still developed her third stroke. Earlier diagnosis and anticoagulation with warfarin may have led to a better outcome. In addition, a second opinion for the neurological imaging and echocardiography should have been sought earlier on. The initial reading of a congenital aetiology for the absence of the brachiocephalic trunk, common carotid artery, internal carotid artery and external carotid artery on the right has never been reported. A singular case report that followed the course of a 70-year-old man who developed aneurysms on top of the absence of the left internal carotid, common carotid and external carotid arteries was reported in 1987.²⁰ Had an embolus been suspected earlier on, anticoagulation and/or surgery would have been pursued much earlier.

This is the first case report on stroke associated with cor triatriatum with a follow-up time long enough to document failure of aspirin and anticoagulation in preventing recurrence of stroke. Removal of the brachiocephalic embolus, resection of the dividing membrane and possible intracardiac thrombus may have led to better outcomes as well.

Learning points

- This report documents a rare case of cor triatriatum in a young female presenting with left-sided weakness. The case suggests a direct association between stroke and cor triatriatum as a result of the heart condition's propensity to disrupt the normal flow of blood through the atria.
- The case highlights the importance of doing transoesophageal echocardiography in young patients with stroke to rule out cardiac embolic sources.
- The review of cases suggests stroke prevention and mortality benefit with anticoagulation and/or surgical resection of the dividing membrane. The present case, however, points out that even anticoagulation may not be enough to prevent stroke recurrence.
- This report adds to the literature on a rare cardiac condition that can cause significant neurological morbidity.

Contributors JDBD is the physician in charge of the care of the patient and he was the one who wrote the initial draft of the article. JJR and EG managed the cardiovascular concerns of the patient and gave important inputs to this paper. JLP is the supervising consultant. All the authors equally contributed to the writing of this paper and approved the final version.

Competing interests None declared.

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