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Cardioembolic Stroke in Sinus of Valsalva Aneurysm, A Case Report

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Abstract:

A case of cardioembolic stroke in a patient with sinus of Valsalva aneurysm. The patient presented with chief complaints of syncope, with slurring of speech and a history of deviation of angle of mouth to the right side, which resolved over one day. Examination revealed no focal neurological deficit. CT Brain revealed a lacunar infarct over the left frontal cortex. Echocardiography showed sinus of Valsalva aneurysm of the non-coronary cusp. Hence a diagnosis of cardioembolic stroke secondary to thrombus present in the sinus of Valsalva of the aneurysm was considered. This is a rare presentation of Sinus of Valsalva as a cardioembolic stroke.

Key Words:

Acute aortic syndrome, cardio-embolic stroke, ruptured sinus of valsalva

Key Message:

Ruptured sinus of Valsalva presenting as a cerebrovascular accident is a rare differential for cardio-embolic stroke. Should be considered as a differential in the appropriate setting.

Rupture sinus of Valsalva (RSOV) is an uncommon life-threatening emergency which presents as acute onset chest pain and cardiac failure. Incidence of RSOV is 0.09% in the general population with no data on its presentation as stroke.^[1] It has rarely been described in literature as a source of emboli, while our review of literature found only four prior case reports in a similar manner. Here we report a case of RSOV which presented as a cardio-embolic stroke, with a comparison against previous reported cases.

Patient Information and Clinical Findings

A 73-year male had presented to casualty with a deviation of angle of mouth to the left with right upper limb weakness, dizziness and acute onset chest pain for the past 6 days. His chest pain was sudden in onset with moderate severity and decreased effort tolerance which progressed from New York Heart Association class I to IV over 4 days. Bilateral lower limb swelling was noticed since the event with swelling up to the knee at presentation and associated abdominal distension. There was no history of trauma, fever, cough. He was not diagnosed with diabetes or hypertension. He had no significant past medical

history. He consumes ethanol weekly and had a 40 pack-year history of consuming cigarettes. He did not give any history of iv drug abuse. He had presented to us on the 5th day of illness.

On examination, he was in distress and diaphoretic. He had a pulse of 128/min which was regular and bounding in character with a blood pressure of 100/60 mmHg. He had a respiratory rate of 35 per minute and required 60% of oxygen to maintain saturation. His JVP was elevated up to 10 cm with bilateral pedal edema extending up to the knees. He had no pallor, icterus, cyanosis, clubbing or lymphadenopathy. He had no peripheral signs of infective endocarditis or Marfan's syndrome. Neurological examination revealed a deviation of angle of mouth to the left with right upper limb monoparesis. Cardiovascular examination revealed a loud continuous murmur at the lower sternal border with diastolic accentuation.

Investigations

His baseline blood investigations revealed mild normocytic anemia with a left shift leukocytosis and thrombocytopenia. His renal function tests showed a pre-renal acute kidney injury with

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metabolic acidosis and medically correctable hyperkalemia. His liver function tests showed elevated liver enzymes along with coagulopathy and undetectable fibrinogen suggestive which along with low platelet count suggesting disseminated intravascular coagulation [Table 1]. His acute viral hepatitis markers were negative. His ECG revealed sinus tachycardia with non-specific T wave changes and chest radiograph revealed cardiomegaly with increased vascular markings. An MRI done in view of his deficits revealed acute infarct in the left frontal lobe on diffusion-weighted imaging. His Echocardiogram subsequently revealed a 5 mm rupture left coronary cusp sinus directly into the right atrium with no associated VSD or involvement of aortic valve [Figures 1-3].

Treatment

He was initially managed with oxygen supplementation and non-invasive ventilation along with appropriate anti-failure medication. He was monitored for features of overt bleed and hepatic encephalopathy. He was given supportive ventilatory care which was weaned off by day 4 of admission, and was clinically better with improvement in renal and liver function tests. His deficits had improved since admission. He underwent angiographic closure of the defect and is on regular follow up.

Discussion

Acute aortic syndromes are a spectrum of life-threatening aortic conditions which include aortic intramural hematoma, penetrating aortic ulcers and the most common and well known, aortic dissection.^[2,3] The prevalence of sinus of Valsalva aneurysms comprises 0.1-3.5% of all congenital cardiac anomalies with an incidence of 0.09% in the general population.^[1] The incidence of the embolic phenomenon of sinus of Valsalva is sporadic and is rare with most data confined to case reports.^[4] Congenital causes like Marfan, Ehler-Danlos

or other connective tissue diseases can predispose to these conditions. Acquired causes are due to aortic wall infection, trauma or inflammatory tissue diseases.^[5]

A large case series comprising 55 patients with ruptured sinus of Valsalva revealed that there was an 80% prevalence among the male gender. The presentation was also varied with extremes of asymptomatic to frank heart failure

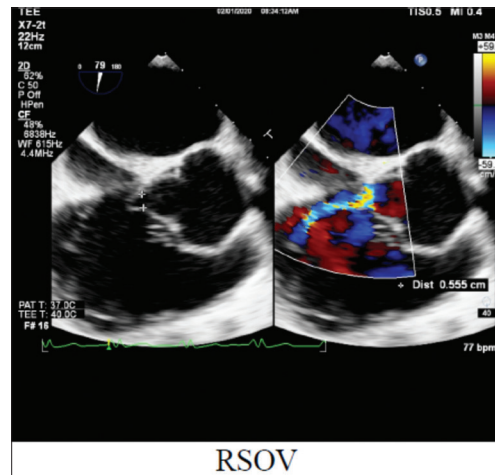


Figure 1: 2D Echocardiogram Revealing The Location of The Shunt

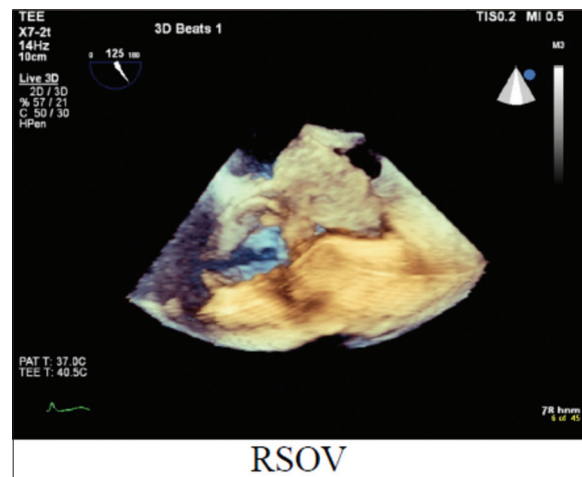


Figure 2 : 3D Echocardiogram Revealing Defect In The Left Coronary Cusp Sinus

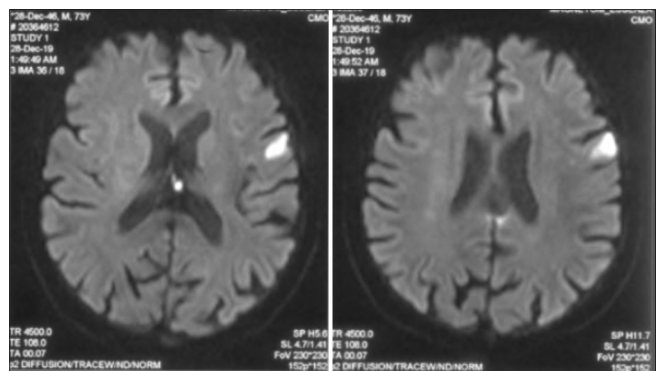


Figure 3: Magnetic Resonance Imaging - Diffuse Weighted Image Revealing A Left Cerebral Infarct In The Frontal Lobe

Table 1: Relevant laboratory investigations

ABG

pH: 7.37; pO₂: 76; pCO₂: 17;
Lactate: 7.0

CBC

Hb: 10.9 gm%;
TC/DC: 15,500 with 80% N;
Plt: 45,000/cumm

LFT

Total bilirubin: 6.19 (0.5-1.0)
Direct bilirubin: 5.22 (< 0.5)
Total protein: 6.2 (6.0-8.5)
Albumin: 3.3 (3.5-5.5)
SGOT: 1469 (8-40)
SGPT: 1601 (5-35)
ALP: 151 (40-125)

Coagulation profile

APTT: 42.7s (27.8-40.4)
INR: 4.84
Fibrinogen: undetectable (150-450)

RF

Urea: 228 mg/dl (15-40)
Creat: 2.08 mg/dl (0.5-1.4)

Table 2: Comparison of clinical outcomes with other case reports

	Our case	Ruzieh et al. 2016	Shahrabani et al. 1993	Stollberger et al. 1996	Nakajima et al. 2013
CEREBROVASCULAR ACCIDENT					
Anterior circulation	Present	Present	Present	Present	Present
Posterior circulation	Absent	Absent	Absent	Absent	Absent
Vessel involvement	Medium vessel	Medium vessel	Medium vessel	Medium vessel	Medium vessel
ECHOCARDIOGRAM					
Visualised Thrombus	Absent	Present	Absent	Present	Absent
MANAGEMENT	Medical management	Medical management	Medical management	Medical management	Medical management
30 DAY SURVIVAL	Yes	Yes	Yes	Yes	Yes

with more than 50% of the cohort however presenting in New York Heart Association (NYHA) class III or higher.^[6] Another case series of 53 cases showed that 60% of the cohort had presented with acute breathlessness with 25% having syncopal attacks. Although infective endocarditis is a known predisposing factor, less than 5% of patients in one cohort had the same.^[7]

An echocardiogram is the ideal first-line investigation as to its non-invasiveness & easy availability. In both the largest studies, the rupture of the right coronary cusp was the most common site followed by non-coronary cusp. The ventricular septal defect was the most common associated cardiac lesion followed by aortic insufficiency.^[6,7] The role of transesophageal echocardiogram is further characterizing the lesion size, location, associated endocarditis or cardiac lesion which may not be easily assessed on a trans-thoracic echo.^[8] The role of a computed tomographic angiogram is the modality of choice when assessing for any arch dissection or hematoma and should be considered in hemodynamically stable patients.^[9]

The common modalities of cerebral ischemia caused due to cardiac abnormalities are in the setting of arrhythmia like atrial fibrillation, ascending aortic atheromatous disease, patent foramen ovale or an atrial septal defect. Other etiologies like rheumatic heart disease, left ventricular thrombus and hypercoagulable states should also be considered.^[10-12] In the case of sinus of Valsalva, thrombosis into the sinus with subsequent artery-artery embolism during the event is the most likely mechanism of cerebral ischemia.^[13] Medical management for the stroke with antiplatelet is indicated with medical stabilization playing an essential role in patient management. Medical management of RSOV involves stabilization with heart failure medication with a perioperative assessment. A transcatheter repair is a feasible option in select cases however surgical repair, especially with the presence of intracardiac shunting, is indicated.^[14,15]

A comparison table of previously reported RSOV presenting as a cerebrovascular accident is presented in Table 2.^[4,13,16,17] All the previous cases were predominantly anterior circulation strokes with a majority presenting as monoplegia or hemiplegia with medium vessel occlusion. Only half of these patients had definitive echocardiographic evidence of a thrombus and all of them were medically managed with subsequent surgical repair of the anatomical defect. 30-day mortality was favorable in all cases.

Conclusion

Acute aortic syndromes are rarely considered in the differential for patients presenting as cardio-embolic stroke. Common causes such as LV thrombus, arrhythmias & RHD are often the most common. The management of this case was challenging as the clinical presentation was protracted and diagnosis was delayed. An RSOV would need prompt surgery, either by angiographic or surgical means based on imaging. The stabilization of these patients would require aggressive supportive care in an intensely monitored setting.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflict of interest

There are no conflicts of interest.

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