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CASE REPORT

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Undiagnosed patent foramen ovale: a rare cause for acute pulmonary embolism and cryptogenic stroke

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Abstract

Patent foramen ovale (PFO) is a congenital cardiac anomaly found in 25-30% of the population. Though often asymptomatic, it may lead to paradoxical embolism and cryptogenic stroke. We present a case of a young man with simultaneous acute pulmonary embolism (PE), ischaemic stroke, and lower limb deep vein thrombosis (DVT), culminating in cardiopulmonary arrest. Transoesophageal echocardiography revealed an undiagnosed PFO, supporting the theory of a paradoxical shunt causing stroke. This case highlights the critical role of optimising anticoagulation in managing unprovoked DVT. PFO is a silent culprit and may result in adverse outcomes in patients with pulmonary embolism.

Keywords: acute pulmonary embolism, cryptogenic stroke, paradoxical embolism, patent foramen ovale

Introduction

Stroke remains the third leading cause of death and disability when considered together.(1) The overall incidence of stroke has significantly risen over the past decade to 85-94 per 100,000 people in a year, but it is recorded to be as high as 1151-1216 per 100,000 in a year, in people who are above 75-years.(2) The reported crude stroke prevalence rate for Sri Lanka is 10.4 per 1000 adults.(3)

Among stroke patients, 85% contribute to ischaemic strokes whereas the remainder accounts for haemorrhagic strokes. Important causes of ischaemic stroke could be categorised into atherosclerotic, cardio-embolic and lacunar infarctions. About 25-39% of ischaemic strokes do not have an identifiable cause on thorough evaluation, and are labeled cryptogenic strokes.(4)

Most cryptogenic strokes are predominantly seen in younger adults, in particular those who are less than 55-years of age.(5) It is considered to be embolic in origin when arising from proximal arterial sources, venous sources, or from the heart with right to left shunts, such as patent foramen ovale (PFO).(6) PFO is a connection between left and right atria that could allow blood or blood clots to pass paradoxically from right to left atria. PFO could be found in 20-25% of the general population. Mostly they are asymptomatic.(7) However, rarely it could lead to cryptogenic strokes by paradoxical embolisation. This accounts for 10-77% of cryptogenic strokes.(8)

Here we present a young man with concurrent acute pulmonary embolism (PE) and acute ischaemic stroke (AIS), leading to cardiac arrest.

Case presentation

A 32-year-old man was found to have severe dyspnoea and reduced level of consciousness and was rushed to the Emergency Department (ED) of

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Teaching Hospital Jaffna. Upon arrival at the ED, he was in asystole and was given cardiopulmonary resuscitation (CPR) for 15 minutes, after which he achieved haemodynamic stability.

He had a history of unprovoked deep vein thrombosis (DVT) in lower extremities in 2018. He had defaulted follow-up and treatment three months after the initiation of anticoagulation. He did not have a family history of vascular anomalies or coagulopathies. He is a non-smoker and claimed not to consume alcohol or illicit drugs.

His blood pressure was recorded as 100/60 mmHg, pulse rate was 130 bpm, oxygen saturation 92% on room air, and the Glasgow coma scale (GCS) was 14/15. The respiratory and abdominal examinations were unremarkable. On neurological examination, he was disoriented and confused. He had dysarthria with grade 4 motor weakness on the right upper and lower limbs.There were no sensory deficits, and reflexes were intact with absent Babinski sign. He was transferred to the intensive care unit for post resuscitation care.

An urgent 12 lead ECG revealed S1, Q3, T3 pattern (figure 1) and CT pulmonary angiogram demonstrated a thrombus involving bilateral main and descending pulmonary arteries (figure2). Non-contrast CT brain revealed an acute infarction involving posterior cerebral artery (PCA) territory on the left side (figure 3). As he had right-side lower limb swelling, a venous doppler study was requested, which confirmed DVT involving external iliac vein. A bedside 2D-echocardiogram showed right atrial and right ventricular dilation, a Tricuspid Pressure Gradient (TRPG) of 52 mmHg with a mild tricuspid regurgitation.

Basic biochemical work including renal and liver profiles were unremarkable. The coagulation profile is listed in table 1.His homocysteine levels were normal and bilateral carotid duplex scan did not show a significant stenosis. Due to the strong suspicion of a cardiac structural anomaly with right to left shunt, a transoesophageal echocardiography with agitated saline bubble study was arranged, which revealed the presence of a small PFO (<3 mm) with a right to left shunt (figure 4).

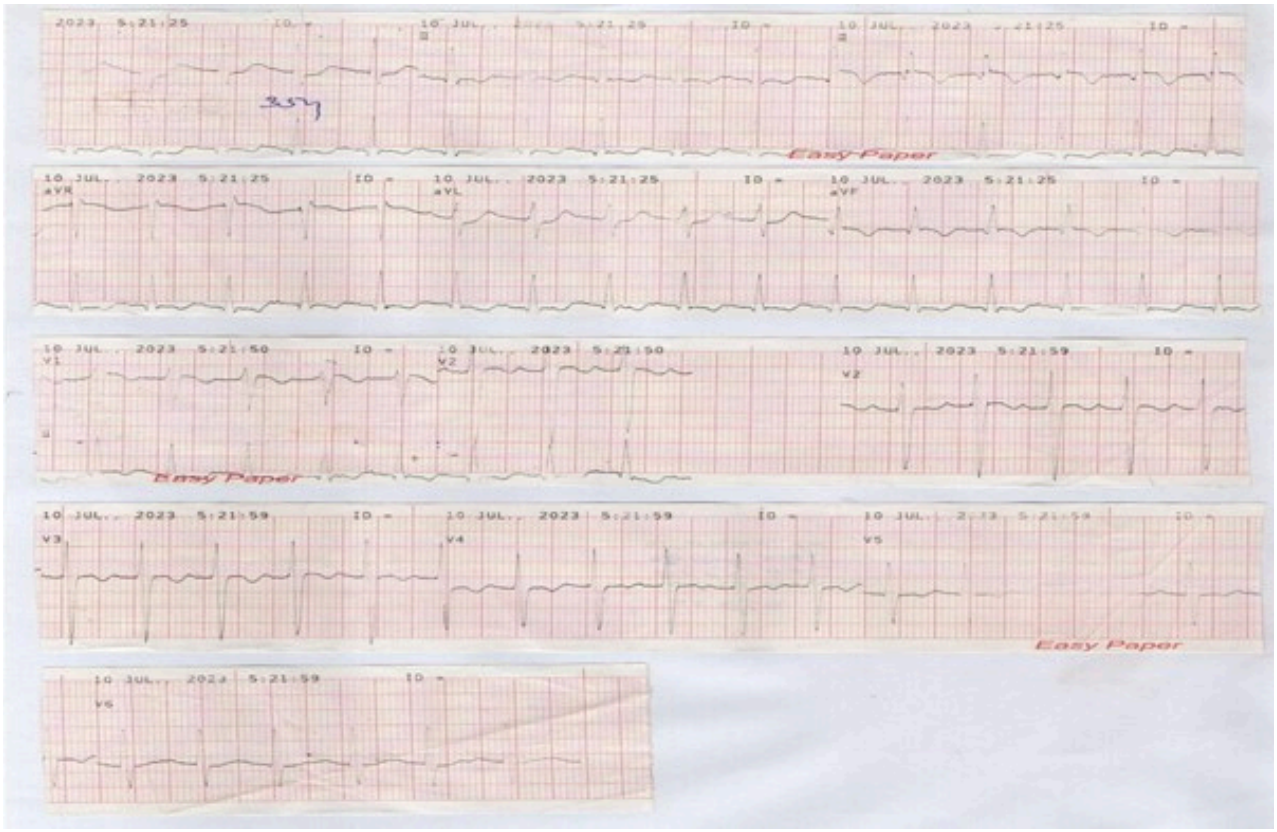

Figure 1 - Electrocardiogram showing sinus tachycardia and S1Q3T3 pattern

Since the patient recovered haemodynamically after prolonged CPR it was decided not to administer intravenous recombinant tissue plasminogen activator (IV rt-PA). Antiplatelet therapy was started with aspirin. Due to the presence of concurrent acute PE, anticoagulation was started with low molecular weight heparin 1 mg/kg subcutaneously together with warfarin 9 mg daily. Physiotherapy and logopedic rehabilitation were initiated and carried out to improve the strength deficit and dysarthria.

As the patient was clinically well and had achieved the therapeutic INR target with warfarin, he was discharged after arranging cardiology and medical clinic follow-up.

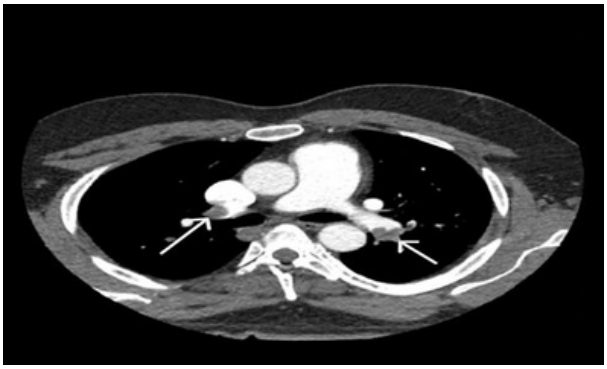

Figure 2 - CTPA showing bi-lateral pulmonary embolism

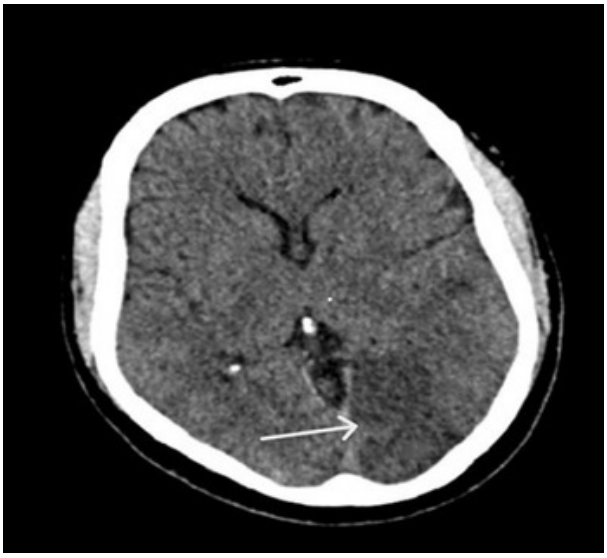

Figure 3 - NCCT brain showing left side PCA territory infarction.

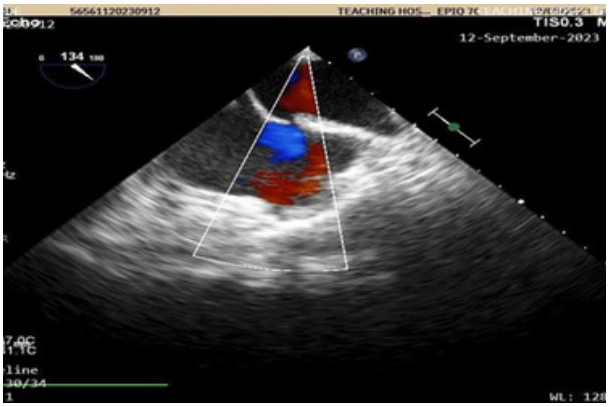

Figure 4 - Transesophageal echocardiogram showing patent foramen ovale

Discussion

Both PE and acute ischaemic stroke (AIS) carry very high morbidity and mortality. In a rare clinical scenario, they could co-exist in the presence of a lower limb DVT in a patient with PFO. In a retrospective study carried out using confirmed cases of stroke in 2021, out of 439 total confirmed cases only 2 cases had simultaneous AIS and PE.(9)

The risk of development of an AIS following a PE is approximately 1-10%. However, PE is the main cause of mortality in the first 2 to 4 weeks following an AIS. (10)

The treatment of these conditions simultaneously is complex due to the concurrent need of rapid administration of IV rt-PA for medical thrombolysis in AIS when they present within 4.5 hours and mechanical thrombectomy up to 24 hours of symptom onset.(11) On the other hand PE would require systemic anticoagulation in order to prevent clot progression and new clot formation which are well-known contraindications following medical thrombolysis owing to high bleeding risk. There are no specific management protocols formulated to guide the management whenever AIS and PE co-exist. Therefore, multidisciplinary approach should be sought to decide upon further management.(12,13)

Surgical closure of PFO is believed to be beneficial in secondary prevention of further strokes. However, presence of a PFO could serve to counteract the increased RA pressure and maintain cardiac output at the expense of low systemic saturation as described in this particular scenario.(14)

In our case a 32-year-old man, with a past history of venous thrombosis presented with sudden onset shortness of breath, hypotension and circulatory collapse leading to cardiac arrest suggestive of a massive PE. Later it was confirmed by CTPA. While evaluating for an acute confusion and right-side hemiparesis, NCCT-brain revealed an acute PCA territory infarction. The venous doppler confirmed the presence of DVT in the external iliac vein.

The triad of concomitant DVT, acute PE and arterial thrombosis with left side PCA territory infarction made us consider an intracardiac shunt leading to a paradoxical embolism. Massive PE results in severe pulmonary hypertension and elevated RA pressure, which in turn could open up an undiagnosed intracardiac right to left shunt causing a paradoxical embolus, which could have been the culprit of the stroke.

To confirm the sequence of clinical events, we requested a 2D-echocardiogram which revealed right atrial and right ventricular dilatation and moderate pulmonary hypertension. However, an intracardiac shunt was not demonstrable. Therefore, a transesophageal echocardiogram was requested, which revealed a small PFO with a right to left shunt.

Conclusion

The triad of DVT, PE, and AIS suggests a possible intracardiac defect with a right-to-left shunt, leading to paradoxical embolism. Cardiac shunts like PFO are common but often underdiagnosed. Physicians should remain vigilant to identify these rare associations for comprehensive care.

Declarations

Conflicts of interest
The authors declare that they have no conflicts of interest

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None

Consent for publication
Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

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Table 1 - Coagulation profile

Investigation	Result
International normalize ratio (INR)	0.8 (0.8-1.1)
Prothrombin time	13 s (11-13 s)
International normalize ratio (INR)	0.8 (0.8-1.1)
Activated partial thromboplastin time (APTT)	32s (25-35 s)
Prothrombin gene mutation	negative
Factor V laden mutation	negative
Methylenetetrahydrofolate reductase (MTHFR) mutation	negative
Protein C deficiency	negative
Protein S deficiency	negative
Beta 2 glycoprotein antibody	negative
Lupus anticoagulant	negative
Anti cardiolipin antibody	negative

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