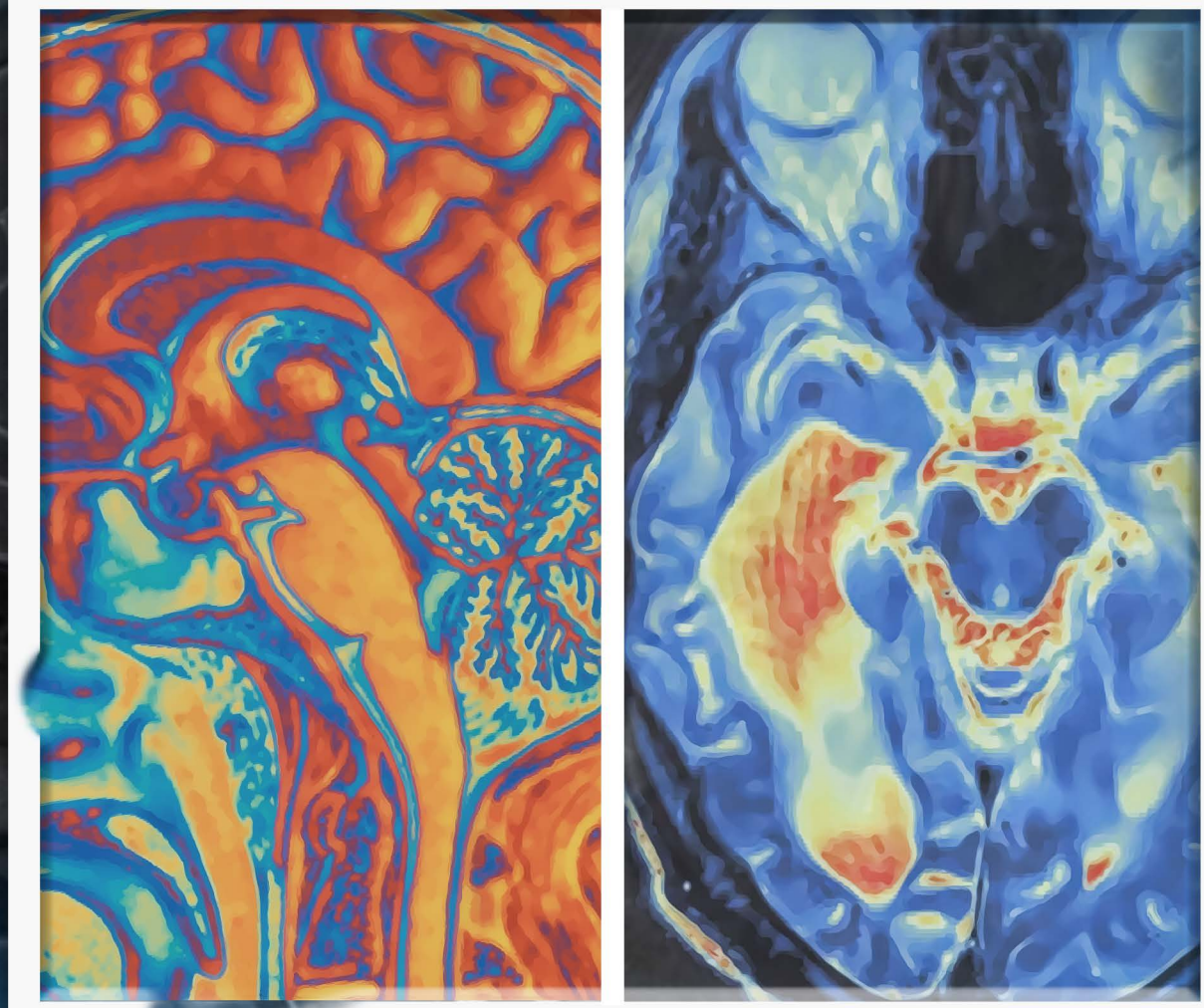
CVNS JOURNAL
Journal of Cardiovascular, Neurovascular & Stroke
Vol3 No4
Dec 2021
eISSN : 2600 -7800
https://doi.org/10.32896/cvns.v3n4



Fatal disseminated paradoxical embolism in inferior sinus venosus atrial septal defect

Fetal posterior communicating artery as a conduit for concurrent anterior and posterior circulation infarct: A case report

Putra acute Stroke protocol

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eISSN 2600-7800

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JOURNAL OF CARDIOVASCULAR, NEUROVASCULAR & STROKE

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FATAL DISSEMINATED PARADOXICAL EMBOLISM IN INFERIOR SINUS VENOSUS ATRIAL SEPTAL DEFECT

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DOI: https://doi.org/10.32896/cvns.v3n4.1-6
Published: 31.12.2021
Article History: Received June 19, 2021; Revised September 30, 2021; Accepted October 28, 2021

ABSTRACT
Inferior sinus venosus atrial septal defect (ASD) is a rare congenital cardiac anomaly. Similar with other types of ASD and patent foramen ovale, this defect results in arteriovenous shunting with the risk of developing paradoxical embolism (PDE) to the systemic circulation from venous emboli. We report a case of a 67-year-old lady who presented to emergency department with massive pulmonary embolism (PE) and recurrent acute limb ischaemia. Computed tomography pulmonary angiography also showed an incidental finding of inferior sinus venosus ASD which led to the diagnosis of PDE. Intravenous thrombolysis was administered followed by open mechanical thrombectomy. The patient developed massive lower gastrointestinal bleed post thrombolysis and passed away despite embolization treatment. This case report describes the catastrophic effect of PDE leading to disseminated multisystem thromboembolism. It also emphasizes the importance of early detection of a right-to-left shunt in patients who present with PE and recurrent acute limb ischaemia of indeterminate aetiology. Transthoracic echocardiography has lower sensitivity than transoesophageal approach in detecting inferior sinus venosus ASD.

Keywords: arteriovenous shunting, pulmonary embolism, limbs ischaemia

INTRODUCTION
Atrial septal defect (ASD) refers to a group of congenital cardiac anomalies that allows communication between the right and left atrium. It includes defects in the interatrial septum and at the cardiac terminations of the systemic and pulmonary veins i.e. sinus venosus defects.[1] ASD accounts for 10 to 15% of congenital heart disease, with a reported birth prevalence of 1 to 2 per 1000 live births.[2] Presence of ASD leads to an increased risk of paradoxical embolism (PDE) with reported incidence of up to 14%.[3] PDE refers to the passage of venous thrombi into the systemic circulation via an arteriovenous shunt. It is a rare phenomenon and accounts for less than 2% of all arterial emboli.[4] The risk of PDE in patients with ASD increases with age due to chronic left ventricular hypertrophy which has decreased compliance and results in progressive enlargement of the ASD.[5] Accurate diagnosis of this condition is challenging due to the variation in clinical manifestation and proven to be fatal without prompt treatment. We present a rare case of extensive thromboembolism due to PDE with an incidental finding of inferior sinus venosus ASD in an elderly.

CASE PRESENTATION
A 67-year-old female presented with a history of left upper limb numbness and coldness for five days associated with left upper limb weakness.

She denied any history of trauma, recent surgery, fever or seizures. No chest pain or shortness of breath. Physical examination of the left upper limb showed no skin discoloration but absence of left brachial, radial and ulnar arteries pulsation. No signal gained on arterial Doppler examination, which thus supported the diagnosis of acute left upper limb ischaemia. Emergency embolectomy was performed followed by heparin infusion. Upon discharge, she was well with good left upper limb perfusion. She was prescribed oral Cardiprin 100 mg daily for a month.

She presented again 25 days later with sudden onset bilateral upper limbs and right lower limb pain, worse on the right lower limb associated with weakness. Clinically, she had tachypnoea with tachycardia. Her right lower limb appeared dusky and cold on palpation. The distal arteries of her right lower limb were non-palpable. She was diagnosed as having recurrent acute limb ischaemia. Due to persistent tachycardia and a Wells score that indicated high probability of pulmonary embolism (PE), an urgent computed tomography pulmonary angiography (CTPA) and limb CT angiography (CTA) were performed. Massive PE was detected with saddle embolus in the pulmonary trunk bifurcation. Pulmonary artery was dilated but no evidence of pruning. However, there was an incidental finding of a defect at the inferior part of interatrial septum consistent with inferior sinus venosus ASD. The right heart chamber size appeared mildly prominent. The interventricular septum was intact but flattened and appeared to be in systolic phase suggestive of increased right heart pressure. There was no significant evidence of right ventricular wall hypertrophy (Figure 1 and 2). The pulmonary veins demonstrated normal drainage into the left atrium. Other cardiovascular structures were also unremarkable.

Limbs CTA showed extensive thrombus within bilateral upper limbs and right lower limb arteries. In the right upper limb, complete occlusion of the subclavian, axillary, radial and ulnar arteries was seen with partial occlusion of the right brachial artery. Long segment thrombus was also seen in the left subclavian, axillary, and brachial arteries (Figure 3). Complete occlusion of the right lower limb arteries was noted involving the entire length of the right common and superficial femoral arteries (Figure 4).

Transthoracic echocardiography (TTE) examination revealed normal atrial and ventricular size. There was interventricular septal hypertrophy and the interatrial septum 'appeared' intact. No obvious ASD was identified.

Disseminated intravascular coagulopathy screening showed normal fibrinogen level at 2.4g/L (normal values: 2.32 – 4.44b/L), which made coagulopathy a less likely cause of the extensive thromboembolic disease.

She received alteplase infusion and underwent open mechanical thrombectomy of right lower limb. Unfortunately, she became markedly anaemic with melena during thrombolytic treatment. Mesenteric CTA confirmed the presence of active lower gastrointestinal bleed. The bleeding artery was successfully embolized through transcatheter approach. However, her condition deteriorated further during hospitalization due to multiorgan failure and worsening septicaemia. She succumbed to the condition after ten days of hospitalization.

DISCUSSION

PDE is a rare phenomenon especially in combination with inferior sinus venosus ASD. To the best of our knowledge, this is the first case of an inferior sinus venosus ASD causing extensive PDE affecting the extremity arteries and pulmonary artery. Inferior sinus venosus ASD is a defect below the atrial septum which leads to an overriding inferior vena cava (IVC) and interatrial connection. Previous case reports have described the association of PDE with patent foramen ovale (PFO). One of the cases was found in an adult patient with bilateral deep vein thrombosis associated with recent air travel that later developed massive PE and acute left upper and lower limbs ischaemia. The subsequent investigation with contrast-enhanced transoesophageal echocardiography (TOE) revealed right-to-left shunt through channel-like interatrial communication consistent with PFO.[6] A case series described two patients with arteriovenous thromboembolism due to PDE. In the series, one patient had concurrent episodes of acute cerebral infarction and PE in the presence of

an ASD, complicated with atrial septal aneurysm and a left-to-right shunt. The other patient had PFO associated with upper extremity artery embolism, venous thromboembolism and PE.[7]

Concomitant limb ischaemia and PE with indeterminate aetiology should alert the treating physician the possibility of arteriovenous shunt and PDE as depicted in our case. Early diagnosis is crucial due to its high mortality risk if left undetected, particularly among older generations. Nonetheless, inferior sinus venosus ASD is extremely rare. Its posterior location also makes the diagnosis through routine TTE a great challenge, hence missed as proven in our case. In view of the proximity between the sinus venosus defects and the transducer, guidelines from the American Heart Association suggested TOE to diagnose this anomaly.[8] However, due to the lack of early clinical suspicion of intracardiac shunt, presence of technical issues with the apparatus and deteriorating clinical condition, TOE was not performed in our patient.

The inferior sinus venosus defect was incidentally discovered in our CTPA examination, prompting the scrutiny of other possible associated cardiovascular anomalies particularly the anomalous right inferior pulmonary venous return to the right atrium. Despite the limited functional information in CTPA, careful evaluation of the cardiovascular structures still able to provide useful indirect information particularly related to complication of longstanding uncorrected shunt as we have described above. Although this type of defect can cause volume overload and is less likely to result in Eisenmenger syndrome due to its low pressure, our patient had some features which suggested the presence of pulmonary arterial hypertension, likely related to the extensive PE. For this reason, TTE is able to compare the chamber size and function; measure the ventricular wall thickness; measure the flow velocity across the tricuspid and pulmonary valves; assess the morphological and dynamic changes of interventricular septum and IVC. The more invasive TOE technique allows better morphological and flow assessment of sinus venosus defect. Having the advantage of unrestricted field of view and non-invasive approach, magnetic resonance imaging is able to accurately demonstrate the location and size of the defect. Using the velocity encoding gradient, it also can accurately quantify and compare the flow volume of the right and left heart chambers, hence grade the severity of the shunt.

CONCLUSION

PDE secondary to intracardiac shunt should be considered in differential diagnosis for patients with concomitant limb ischaemia and PE of unknown aetiology since it can potentially lead to extensive thromboembolism. The high mortality rate can be secondary to disease severity or complications of treatment. CTA is the alternative imaging modality to detect sinus venosus ASD provided there is sufficient knowledge of congenital cardiovascular disease and its complication.

DISCLOSURES

Source of Support and Funding: The authors received no specific funding for this work.
Conflict of Interest statement: The authors declare no conflict of interest.
Author Contributions Statement:
ZKAI contributed to writing the manuscript.
KAS contributed to case selection, proofreading the manuscript and providing expert opinion.
MSA supervised the overall manuscript preparation.
Consent to Participate:
Verbal consent was taken from the next of kin of the patient described in the case report since the patient died due to the illness. Ethical board approval for submission of this case report was obtained from Research Ethical Committee of Hospital Universiti Sains Malaysia

FIGURE LEGENDS

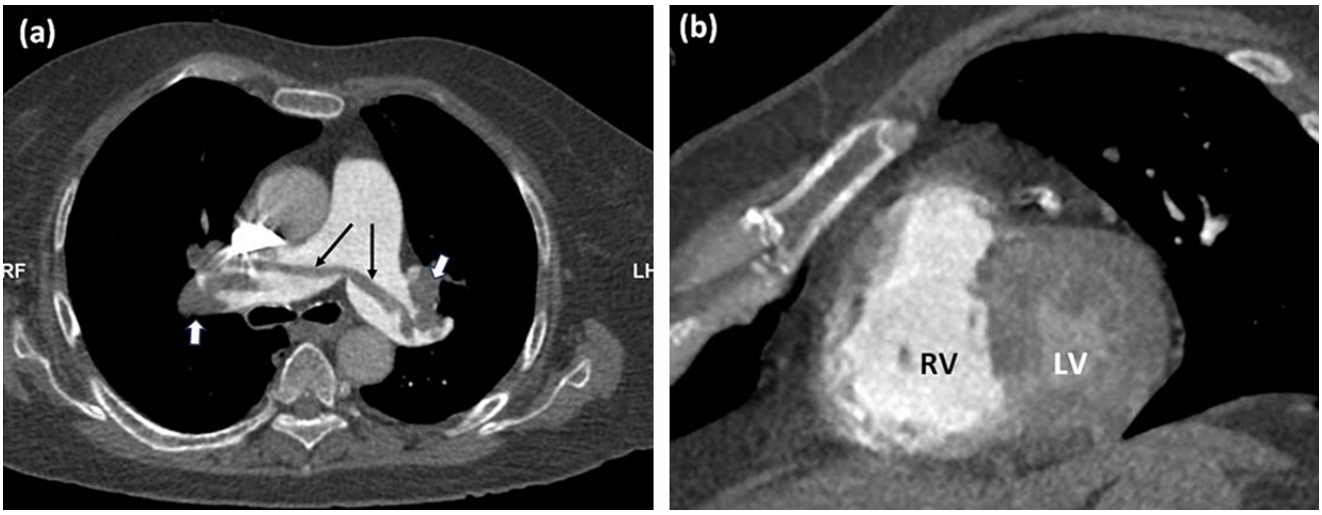

Figure 1: (a) Axial CT pulmonary angiography at the level of pulmonary artery bifurcation demonstrates dilated pulmonary trunk with presence of saddle embolus extending within both main pulmonary arteries (thin arrows). Large emboli are also seen within both segmental branches of pulmonary arteries (thick arrows). (b) There is straightening of interventricular septum indicating presence of pulmonary arterial hypertension.

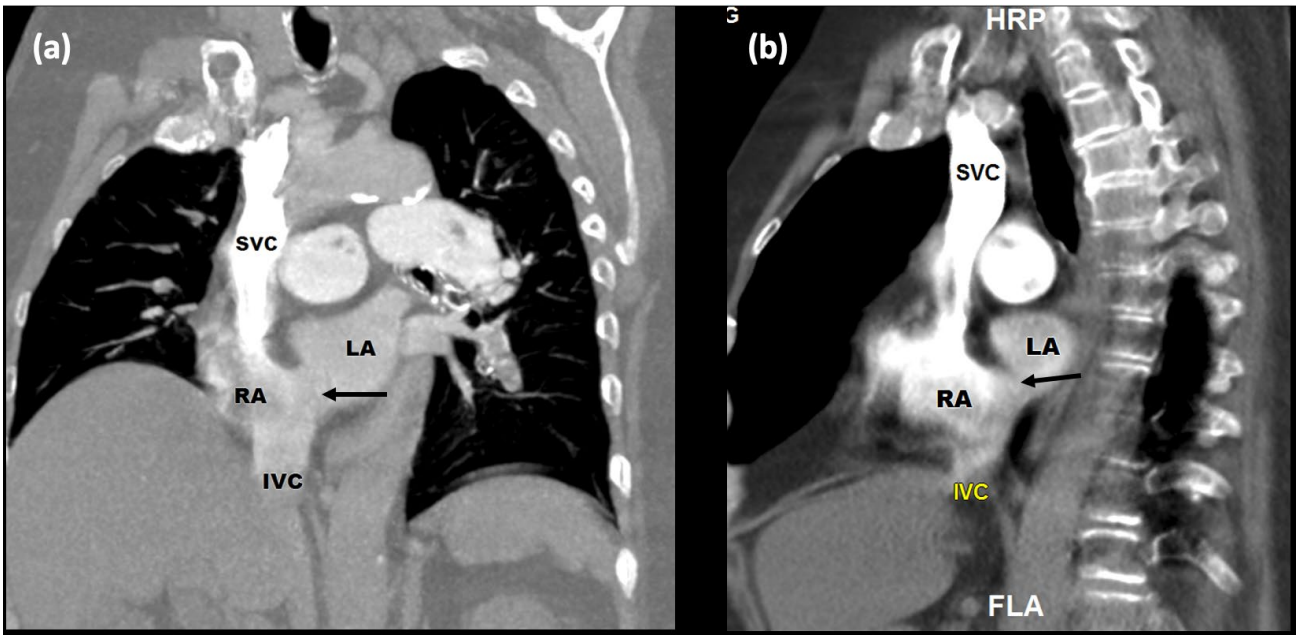

Figure 2: CT pulmonary angiography images in (a) oblique coronal and (b) oblique sagittal planes demonstrate the inferior sinus venosus defect (black arrow). Note the opacification of the left heart chambers during pulmonary arterial phase indicating interatrial right-to-left shunt. The IVC nearly overrides the defect, a characteristic feature of this anomaly, best illustrated in (a).

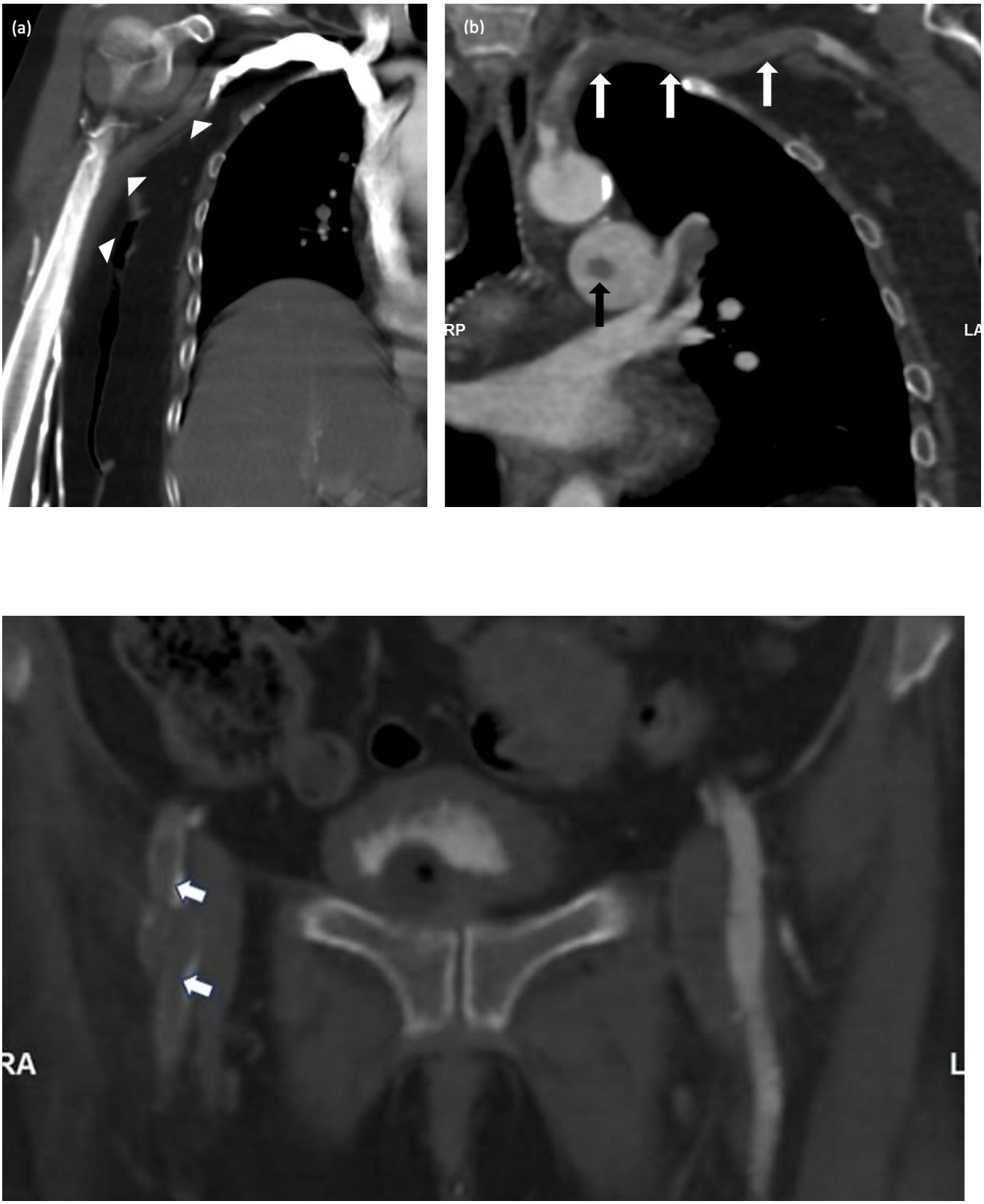

Figure 4: Coronal reformatted CT angiography image of the lower limbs demonstrates long segment filling defect within the right common and superficial femoral artery (arrows). The left femoral artery is well-opacified.

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FETAL POSTERIOR COMMUNICATING ARTERY AS A CONDUIT FOR CONCURRENT ANTERIOR AND POSTERIOR CIRCULATION INFARCT: A CLINICAL CASE REPORT

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DOI: https://doi.org/10.32896/cvns.v3n4.7-11
Published: 31.12.2021
Article History: Received October 30, 2020; Revised October 07, 2021; Accepted October 28, 2021

ABSTRACT
Fetal type posterior cerebral artery (FTPCA) is a variant of posterior circulation of brain, in which the distal part of posterior cerebral artery (PCA) is perfused by a branch of internal carotid artery (ICA) via fetal posterior communicating artery (fetal PCOM). In the presence of fetal PCOM, a paradoxical concurrent infarction of anterior and posterior circulation may happen. We report a 67-year-old man who presented with sudden onset right sided weakness and aphasia, with National Institutes of Health Stroke Scale (NIHSS) score of 22 and clinically diagnosed to have left total anterior circulation infarct (TACI). Subsequently, he received IV Alteplase as a standard hyperacute ischemic stroke treatment. Computed tomography angiography (CTA) of brain showed left FTPCA with prominent left fetal PCOM. Subsequent computed tomography (CT) of brain showed concurrent left middle cerebral artery (MCA) and PCA territories infarct. CTA brain is commonly done in ischemic stroke cases to assess presence of large vessel occlusions and intracranial or extracranial atherosclerotic disease. However, this case depicts its additional role in detecting anatomical variants of cerebral circulation. In terms of clinical importance, presence of multiple territories infarction portends a poorer neurological outcome.

Keywords: Infarct

BACKGROUND
Posterior cerebral artery (PCA) is an important branch of basilar artery in posterior circulation of the brain. It functions to supply the occipital lobe, the inferomedial temporal lobe, and portions of the posterior inferior parietal lobe. [1] Fetal origin of the PCA is a common variant in the posterior cerebral circulation, with an estimated prevalence of 15-32% of individuals. [2] This common variant is mainly detected only after a patient has suffered an ischemic stroke or when a non-invasive or invasive cerebral angiography is performed for various indications.

Fetal type PCA (FTPCA) denotes bulk of blood supply to PCA territory arising from internal carotid artery (ICA) via posterior communicating artery (PCOM) with absent or hypoplastic P1 segment of PCA. This variant of PCOM is called fetal PCOM. Van Raamt et al. have proposed the term full fetal type PCA (full FTPCA) for total absence of P1 segment, while partial FTPCA for hypoplastic P1 segment. In both circumstances, the posterior communicating artery appears larger than usual on CTA brain. [3] Although FTPCA is a normal variant; it results in the lack of proper collaterals and larger than usual posterior communicating artery may pose an

increased risk for ischemic injury to the anterior as well as posterior cerebral regions. [4] A concurrent infarct involving multiple arterial territories herald a poorer neurological outcome. We report a case of concurrent left middle cerebral artery (MCA) and PCA territory infarction, sparing the anterior cerebral artery (ACA).

CASE PRESENTATION

A 67-year-old man presented with sudden onset of right sided body weakness and slurred speech at 11.30am. Otherwise, he didn't complain of headache, blurred vision, vomiting or numbness. There was no seizure. He neither complained of chest pain nor palpitation. He had underlying schizophrenia diagnosed in 2015, on chlorpromazine 100mg on night. He had a laparotomy 3 years ago, which the cause remained uncertain. He is an active smoker but not known to have diabetes mellitus, hypertension or dyslipidaemia.

Upon arrival, his Glasgow Coma Scale (GCS) was E4V3M5, pupils bilaterally equal and reactive. Initial blood pressure 138/87mmHg, pulse rate 57 beats per minute, respiratory rate 16 breaths per minute, temperature 37ºC, pain score 0. His oxygen saturation was 95%, and capillary blood glucose 5.7mmol/L. His lungs were clear on auscultation, no murmur and no carotid bruit. Examination of abdomen revealed a midline laparotomy scar. Neurological assessment revealed normal tone with right upper limb and lower limb muscle power grading 5/5, while left upper limb and lower limb power 2/5 based on medical research council scale for muscle strength. Otherwise, reflexes were normal with extensor plantar response bilaterally.

His initial NIHSS score was 22. Electrocardiography revealed sinus rhythm. Blood investigations revealed hemoglobin count 16.2g/dL, white blood cell count 13 X 10^3/μL, platelet count 269 X 10^3/μL, hematocrit 48%, sodium 141 mmol/L, potassium 4 mmol/L, urea 4.1 mmol/L , creatinine 105 μmol/L , corrected calcium 2.07 mmol/L, phosphate 1.43 mmol/L, prothrombin time 13 sec, activated plasma thromboplastin time 41 sec, international normalised ratio (INR) 0.99.

Non contrasted CT brain showed as below:

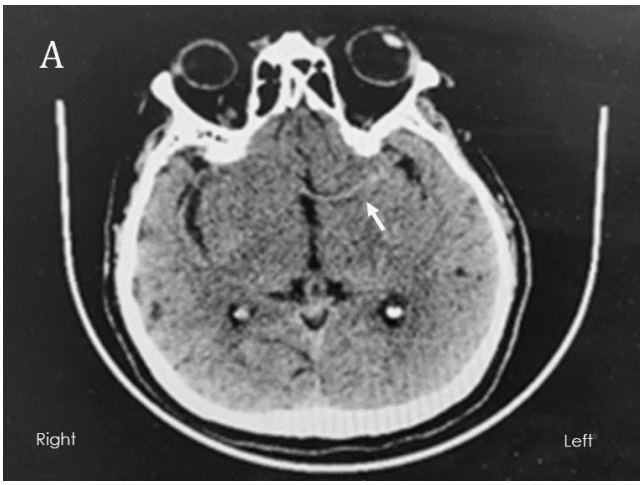

Figure A. Hyperdense left MCA sign. (arrow)

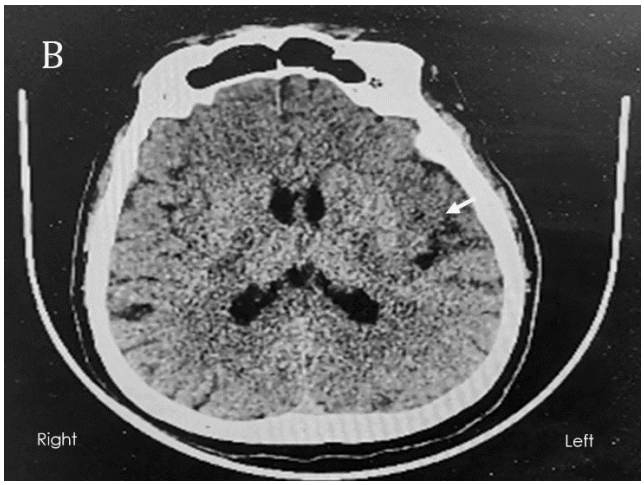

Figure B. Hypodense of left parietal and insular cortex. (arrow)

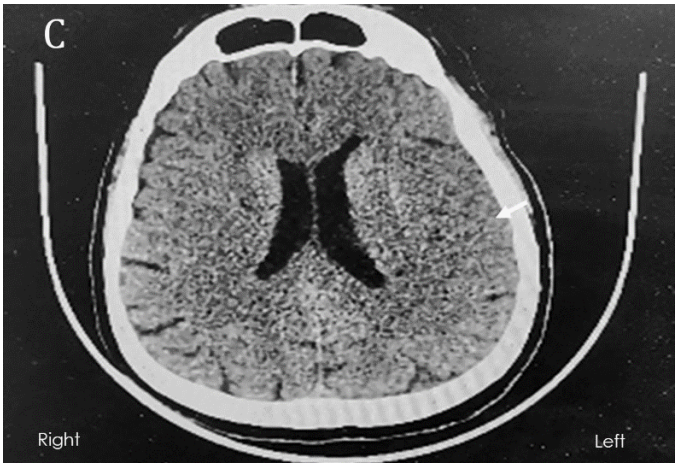

Figure C. Loss of sulci over left parietal area. (arrow)

The ASPECT score was 5, involving left M1, M2, M3, M4, and insular cortex.

Based on clinical symptoms and imaging, he was diagnosed as left total anterior circulation infarct (TACI) involving left MCA territory, based on Oxfordshire Community Stroke Project (OCSP) classification. Intravenous Alteplase 50mg (based on body weight) was given at 3hour 20minutes from symptom onset.

Subsequently, a cerebral CT angiogram was performed. The imaging as follows:

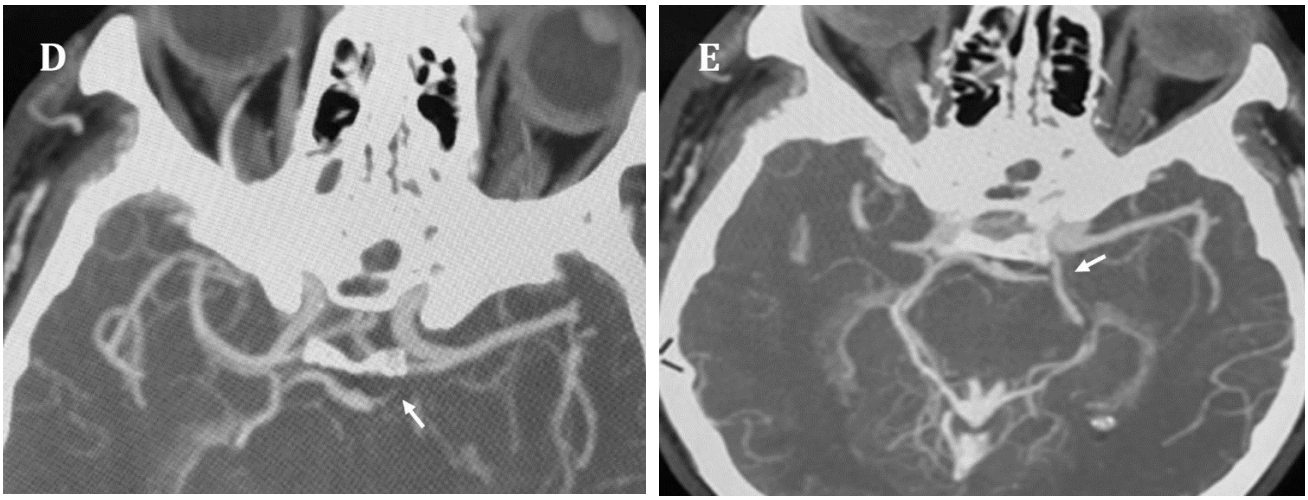

Figure D: Hypoplastic left P1. (arrow)

Bilateral MCA and ACA were patent. Intracranial part of bilateral ICA was patent. However, unable to comment on extracranial ICA as our CT angiography was not extended down to arch of aorta.

He was admitted into intensive care unit for close monitoring. Systolic blood pressure remained in the range of 150-180 during and after alteplase infusion. Unfortunately, 2 hours later he developed respiratory distress due to acute pulmonary oedema. He was intubated and ventilated, after initial intravenous frusemide failed to improve his clinical state.

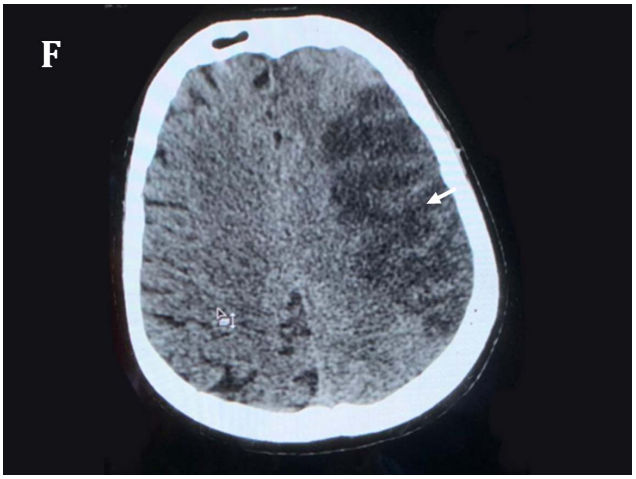

Figure F: Infarct over left MCA territory. (arrow)

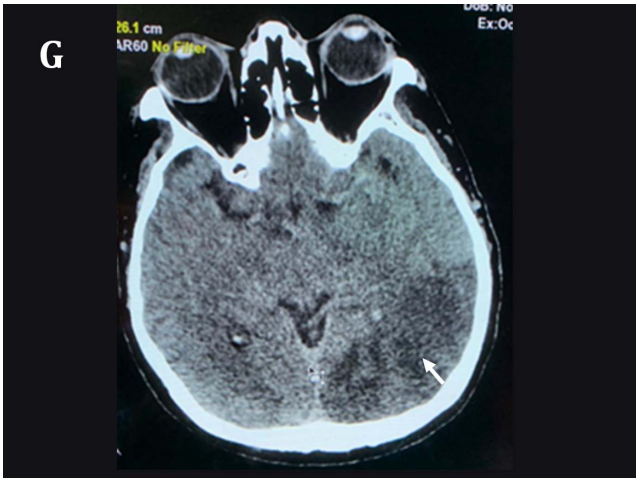

Figure G: Infarct over left PCA territory. (block arrow)

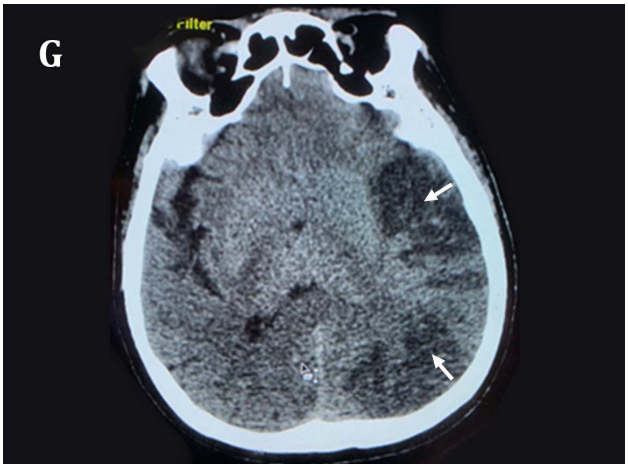

Figure H: Infarct over both left MCA (arrow) and PCA territory. (block arrow)

Repeated CT brain at day 4 of stroke revealed well established concurrent right MCA and PCA territory infarctions. Ipsilateral mild cerebral oedema *was* present as well. Hence, IV mannitol 20% 200cc 12-hourly was given for one day. His Glasgow Coma Scale (GCS) was E2VTM5 at day 5 of stroke despite 24 hours without sedation. Subcutaneous heparin 5000unit BD was started for deep vein thrombosis prophylaxis and oral aspirin was initiated at day 5 of stroke. He was planned for trachaeostomy and weaning off ventilatory support. However, he developed pneumonia and acute kidney injury requiring high ventilator setting and hemodialysis respectively. Eventually, succumbed to sepsis on day 13 of stroke.

DISCUSSION

MCA infarct is one of the devastating forms of stroke. Clinically they can come in the form of Partial Anterior Circulation Infarct (PACI) or Total Anterior Circulation Infarct (TACI). Our patient presented as TACI, however to our surprise he had a concurrent MCA and PCA territory infarcts.

A fetal origin of Posterior Cerebral Artery is a common variant in posterior circulation, estimated to be of 15-32% of population. This means the PCOM will be larger than P1 segment of PCA, which the latter will be hypoplastic or at times absent. Hence, bulk of blood supply to PCA comes via PCOM, from ipsilateral ICA. This type of PCOM is named fetal PCOM.

Our patient suffered a concurrent left MCA and PCA territory infarcts due to the presence of left fetal PCOM (fetal origin of PCA). We postulate an artery-to-arteries embolization (from extracranial part of left internal carotid to left PCA-P2 segment and left MCA).

Nevertheless, CT angiography did not show acute thrombus in either vessel, which could be due to lysed clot (post intravenous thrombolysis) or the clots could have migrated further distally. Distal occlusions are not readily visible on CT angiography.

Otherwise, a concurrent infarct could be as well of cardiac origin, whereby a thrombus from intracardiac region could have travelled up to intracranial artery via internal carotid, causing occlusion of left MCA and PCA, with left PCOM

serving as a conduit for cross embolization from the ICA to the PCA P2 segment or its distal branches. Unfortunately, 24 hours cardiac activity recording (Holter examination) and echocardiography was not done to confirm possibility of cardioembolism.

According to van Raamt et al, a full FTPCA carries a higher vascular insufficiency risk than a partial FTPCA because leptomeningeal anastomoses do not form between the anterior and posterior circulation if a person has a full FTPCA.[3] A series of cases reported by Nico et.al showed patients with fetal origin of PCA is more likely to have a posterior circulation infarct, rather than concurrent anterior and posterior circulation infarct [5]. This is supported by a theory of increased ICA-PCA pressure gradient due to hemodynamic changes from stenotic lesions in vertebrobasilar system or significant artherosclerosis of proximal ICA.[6]

This case of fetal-type PCA (FTPCA) and concurrent PCA-MCA territory infarction illustrates the variability in anatomy and clinical significance of PCA variants. FTPCA is clearly the mechanism of paradoxical infarction in this patient. To our knowledge, reports of concurrent multiple territories infarct are rare and adds on significantly to poor outcome of major acute ischemic strokes (TACI and PACI). This case illustrates a typical presentation of TACI and subsequent hyperacute stroke reperfusion therapy as the treatment for TACI. Also, this highlights the importance of CT angiography of brain in aiding management of acute ischemic stroke. Furthermore, it has served to explain why patient developed concurrent multiple territory infarcts. FTPCA can increase the extent and severity of anterior circulation strokes by allowing additional infarction in the PCA territory. Whether FTPCA increases the overall risk of stroke independent of other risk factors is not clear. The optimal stroke prevention regimen for individuals with FTPCA and one or more stroke risk factors is also unclear. Risk factors control remains the mainstay in stroke prevention.

The authors acknowledge the generosity of Mr M's family in providing permission to write about Mr M's treatment in our facility.

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PUTRA ACUTE STROKE PROTOCOL

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DOI: https://doi.org/10.32896/cvns.v3n4.12-14
Published: 31.12.2021
Article History: Received September 16, 2021; Revised September 26, 2021; Accepted October 28, 2021

Keywords: Acute Stroke, Magnetic Resonance Imaging, Stroke protocol.

Computed Tomography (CT) is by default the first line neuroimaging method for acute stroke in many institutions; however, early infarct signs on CT can be subtle. Many experiences have proved that magnetic resonance imaging (MRI), especially diffusion weighted imaging (DWI), is significantly more sensitive than CT in the identification of the infarct core, with better correlation to the final infarct volume (1). Recent scepticism about the current understanding of the ischemic core further supports the need for a more detailed tissue information from imaging in hyperacute stroke (2). MRI provides significantly more valuable information in hyperacute stroke and does not compromise the door to needle (DTN) time (3-5). Sakamoto Y et al have shown that a MRI-first policy is feasible in tertiary general academic teaching hospitals, and significantly reduces the door to reperfusion time (2). Centres adopting the MRI first policy in acute stroke have shown that it is feasible, safe, with satisfactory DTN times (5-7).

Our centre is a newly established teaching hospital and are among few centres which embrace the MRI-first policy for hyperacute stroke. We adopt the Putra Acute Stroke Protocol, with an initial 8-minutes first 3 sequences consisting of DWI, fluid-attenuated inversion recovery (FLAIR) and magnetic resonance angiography (MRA). If DWI shows suspected haemorrhage, the order of the sequences is modified, so that susceptibility weighted imaging (SWI) then comes after DWI (Figure 1). Imaging is paused after the first 3 sequences for the clinical team to determine if intravenous tissue plasminogen activator (IV-tPA) treatment should be administered, or preparations for mechanical thrombectomy (MT) should begin. The order in which subsequent sequences are completed during the scan depend very much on the clinical indication at the time; this usually consists of ASL (arterial spin labelling), SWI (if not included in the first 3 sequences) and MRA of the carotid arteries. Contrasted perfusion MRI and black blood (BB) imaging are performed if contrast is decided to be given.

The adopted protocol enables us to achieve a decision after the third sequence. Aided by the capability to thrombolyse inside the MRI suite, this helps to ensure that the average DTN time is

less than 60 minutes, as recommended by the majority of current guidelines (3,4).

From January to July 2021, a total of 198 patients underwent the MRI-first policy for hyperacute stroke, adopting the Putra Acute Stroke Protocol. Of these, 40 patients received IVT, while 22 patients underwent MT, representing 20% and 11% of the total patients, respectively. Eighty percent of the IVT patients had a DTN time of less than 60 minutes. There were a total of 20 patients who presented with haemorrhage – all of which were identified on the DWI sequence and subsequently confirmed on the SWI sequence. Additionally, the fact that the Putra Acute Stroke Protocol incorporates both SWI and contrasted BB (black blood) imaging enables us to identify haemorrhage, masses, or infection in most instances. Our early observation shows that DWI with ADC is able to identify haemorrhage in acute stroke, even by less experienced radiologists.

From June to December 2020, we had 14 stroke mimics (33% metabolic causes, 25% epilepsy, 25% neuroinfections; the rest due to other causes). Our limited experience showed that all the stroke mimics were accurately diagnosed, thus avoiding inadvertent thrombolysis. Transient ischemic attacks (TIAs) were also clearly identified in our MRI-first policy, avoiding unnecessary treatment. The data is available and is planned to be published but is not yet ready at this juncture.

The DWI and ADC sequences pick-up haemorrhage very well. This is followed by confirmation with the SWI sequence, ensuring that haemorrhagic stroke is not missed when utilizing a MRI-first policy in hyperacute stroke practice. At the moment, we are working on our data of DWI and ADC in identifying haemorrhage in hyperacute stroke. Preliminary findings show an almost 100% pick-up rate, however, the data is not finalised yet. We hope to publish the findings soon.

There were challenges in establishing a MRI-first policy in acute stroke, as highlighted by a few studies. In our limited experience, the commitment of the core team to solve initial hurdles, aided by good diagnostic outcomes, strengthens the belief of other stakeholders to embrace MRI for acute stroke. We hope to present these issues in a more comprehensive manner in another study.

Cost and benefit is one of the main issues touched by many people when discussing about MRI services in acute stroke. There is no cost of x-ray tube wear and tear in MRI, therefore, the more cases that are performed should not increase the cost of replacement for parts. The actual cost of MRI is relatively fixed even with examinations performed up to 24 hours a day, 7 days a week. The cost of heat management is also relatively fixed, bar quenching. Many centres leave the MRI machine idle outside of standard working hours, which increases cost inefficiency. A significant percentage of acute strokes present outside of standard working hours, which occupies the idle MRI slots and averages down the MRI overall cost.

The MRI examination market price set previously in our country was set during the early introduction of MRI, reflecting the high purchase price of the MRI scanner then, and the complexity of performing a MRI examination. With price reduction, increased efficiencies, and faster scanning times, a tremendous increase in the capability to perform more examinations per scanner is seen; however, the savings are not reflected well to the price charged per MRI examination - making it appear more costly than it actually is. We are hoping to evaluate, in more detail, the cost versus benefit in future undertakings.

In conclusion, our MRI-first policy for hyperacute stroke adopting the Putra Acute Stroke Protocol did not result in delay of DTN times and did not reduce the adoption for hyperacute stroke treatment, be it either IVT or MT.

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Figure 1:

The Putra Acute Stroke Protocol. The MRI examination will be stopped for IV thrombolysis after the third sequence and administered inside the MRI suite, if indicated. The patient will then be transferred to the angiography suite (after the MRI examination), if indicated for mechanical thrombectomy. If hemorrhage is suspected on the DWI sequence, the SWI sequence will be the sequence that ensues DWI, instead of FLAIR.

SEQUENCES	ACQUISITION TIME
DWI	01:47
FLAIR	02:40
MRA	04:09
SWI	03:42
PERFUSION IMAGING (CONTRAST)	02:05
BLACK BLOOD VESSEL WALL IMAGING	05:00
OTHER SEQUENCES	

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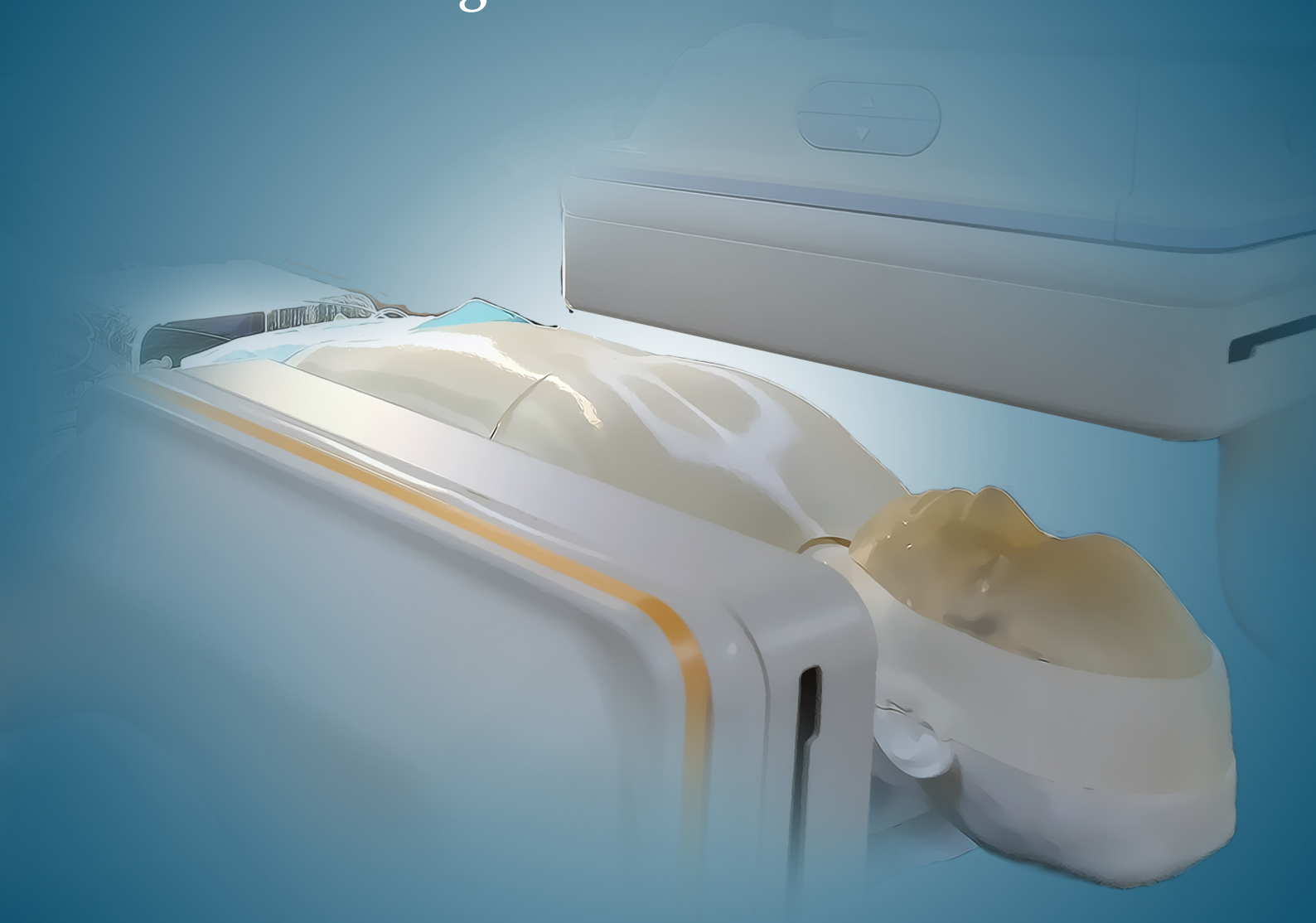

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