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Intracranial dural arteriovenous fistula mimicking ischemic stroke and treated with intravenous thrombolysis

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Abstract

Stroke patients are evaluated with a non-contrasted brain computed tomography (CT) scan for decision-making for intravenous thrombolysis. We report a case of a patient who presented with receptive aphasia, and was administered IV alteplase after the non-contrasted brain CT showed no contraindication. However, a dural arteriovenous fistula (dAVF) was detected on the subsequent CT angiography performed in the consideration for endovascular therapy. The patient developed fatal symptomatic intra-cerebral hemorrhage, despite subsequent cessation and reversal of thrombolysis. This case highlights how early CT angiography can be useful in hyper-acute stroke patients beyond the detection of large vessel occlusions, with its ability in revealing stroke mimics such as dural arteriovenous fistula and other contraindications to thrombolysis, which may be missed on the non-contrasted brain CT.

Keywords: Dural arteriovenous fistula, thrombolysis, stroke mimics, angiography, intracranial hemorrhage

CASE REPORT

A 66-year-old Chinese lady with no prior medical history, presented acutely with receptive dysphasia. On examination, her blood pressure was 133/76 mmHg, pulse rate was regular at 97 per minute. The initial National Institutes of Health Stroke Scale (NIHSS) score was 6 (2 each for the components of loss of consciousness questions, commands and best language). A non-contrast computed tomography (CT) brain was ordered, showing neither hemorrhagic nor early ischemic changes (Figure 1). Intravenous alteplase was administered an hour from the onset of symptoms, with an initial bolus of 10% of the intended dose of 0.9mg/kg bodyweight, followed by infusing the remaining 90% over an hour.

In the search for large vessel occlusions whilst assessing her suitability for endovascular therapy, she immediately underwent a CT angiography (CTA) after the bolus of alteplase. The CTA was negative for large vessel occlusions, and the patient was not subjected through endovascular therapy. The CTA was later reviewed by a senior radiologist on duty who observed dilated cortical in the posterior aspect of the left cerebral hemisphere, associated with increased contrast density in the left transverse sinus and severe stenosis at the transverse-sigmoid sinus junction. These findings were consistent with an underlying dural arteriovenous fistula (dAVF) (Figure 2 and 3). The IV alteplase infusion was nearing completion when the dAVF was noticed, with the patient having already received more than 90% of the intended dose. Despite the immediate cessation of the infusion and the prompt administration of fresh frozen plasma, she deteriorated rapidly to a Glasgow Coma Score (GCS) of 3 within an hour. An urgent non-contrasted CT brain scan showed a large intra-parenchymal hematoma within the left cerebral hemisphere, associated with mass effect and consequent left subfalcine and uncal herniations (Figure 4). She subsequently passed away 5 hours from the time when alteplase was administered.

DISCUSSION

DAVFs account for 10-15% of intracranial arteriovenous malformations[1], with differing rates of neurological complications and consequences. Crude detection rates of dAVFs in Japan and Finland were reported to be 0.29 and 0.51 per 100,000 adults per year respectively.[2,3]

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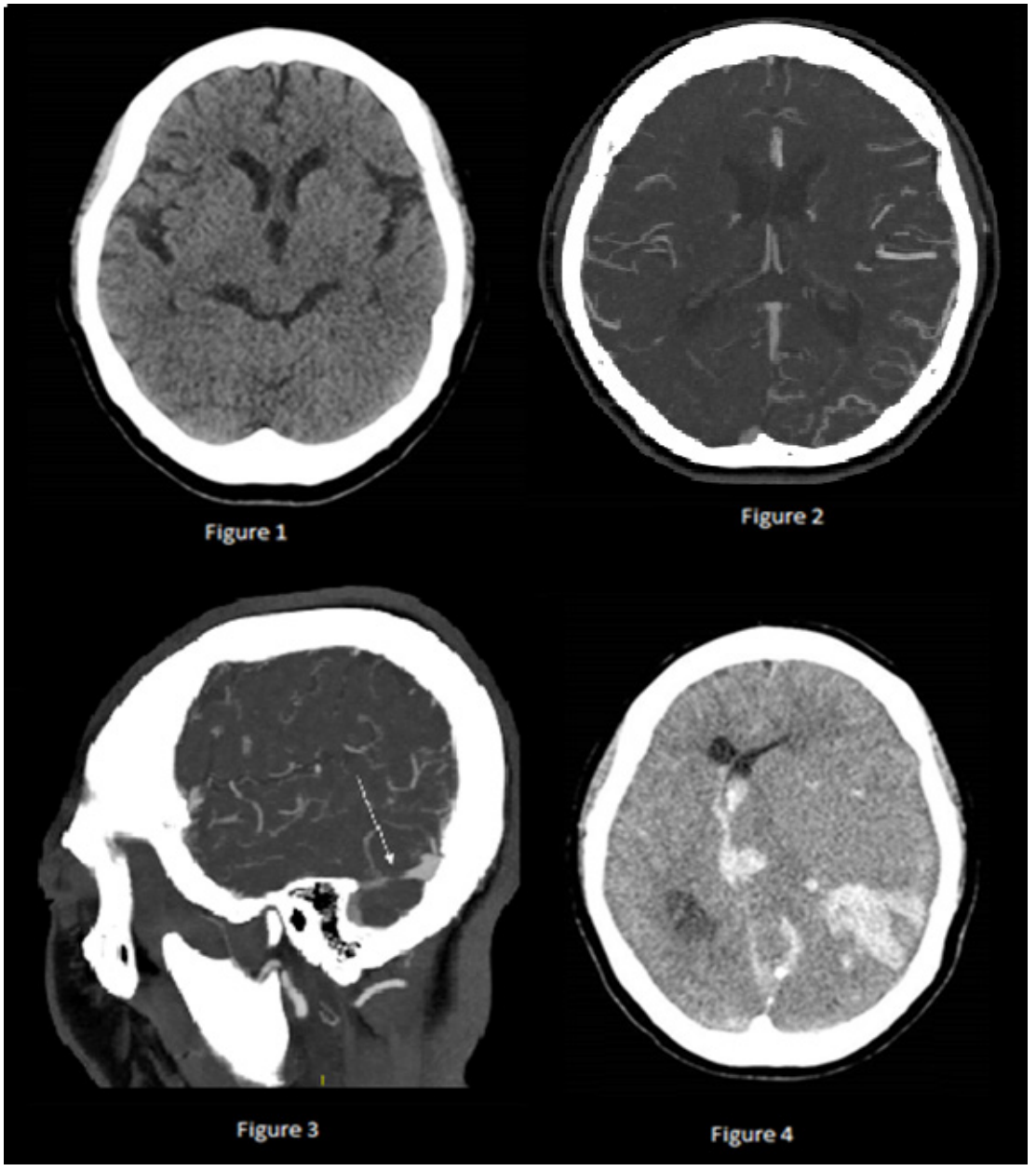

Figure 1. The initial non-contrasted CT brain (Figure 1) was negative for early ischemic changes and intracranial hemorrhage.

Figure 2. Subsequent CTA detected tiny wriggly vessels in the cerebral sulci (arrow), predominantly over the posterior aspect of the left cerebral hemisphere.

Figure 3. The left transverse sinus was irregular, with a large dilated portion at the lateral aspect as it drained into the sigmoid sinus, and severe stenosis at the junction of the left transverse sinus to the left sigmoid sinus.

Figure 4. A non-contrasted CT brain done 3 hours after IV alteplase showed a large intra-parenchymal hematoma in the left cerebral hemisphere, with associated mass effect causing midline shift to the right and intracranial herniation, and intra-ventricular extension of the bleed.

Adults diagnosed with dAVFs are more likely to be middle-aged, with a slight female predominance.[11,12,19] Despite the relative rarity, aggressive variants of dAVFs confer an annual risk of non-hemorrhagic neurological deficits of up to 6.9%.[4,5] Non-hemorrhagic neurological deficits include seizures, cerebellar dysfunction and focal neurological deficits which may mimic an ischemic stroke.[17]

The search for intracranial dAVF with non-contracted CT brain scans can be difficult, unless guided by the presence of hemorrhagic or thrombotic sequelae. The utilization of CTA allows better identification of dAVFs, with sensitivity and specificity at 93% and 98% respectively in a recent Taiwanese study[6], demonstrating abnormally enlarged, tortuous and dilated cortical veins, enlarged and engorged external carotid artery or transosseous vessels, asymmetric sinus enhancement (with arterialization of contrast phase) and the occlusion of dural sinuses.

Current treatment recommendations in the initial management of hyper-acute ischemic strokes indicate the incidental presence of an intracranial arteriovenous malformation as a relative contraindication to IV alteplase[8], based on case reports of patients with myocardial infarction[7,8] and massive pulmonary embolism[10] who underwent thrombolytic treatment.

Clinical history alone is of limited utility in detecting patients with dAVFs, as the pathophysiological basis for the formation of dAVFs remains unclear and the majority of cases were devoid of significant prior precipitating events[16], although a small number of patients reported a history of prior craniotomy, head trauma or dural venous sinus thrombosis.[13-16]

The case described herein, illustrated how dAVFs can present as stroke mimics, may be missed by a non-contrasted brain CT, and can be better detected by CTA. The detection of the dAVF would have rendered the attending physician especially wary of the devastating hemorrhagic consequence of alteplase administration. Reassuringly, the incorporation CTA in the assessment of patients with acute stroke has not been shown to significantly contribute to delaying intravenous thrombolytic therapy in multiple studies.[17,18]

The two main learning points from this case are: (1) It is important to maintain an index of suspicion for dAVF-related non-hemorrhagic neurological deficits as potential stroke mimics, especially in middle-aged patients without cardiovascular risk factors. (2) The utility of CTA in EVT goes beyond the detection of large vessel occlusion, as it provides an opportunity to search for vascular abnormalities such as untreated intracranial arteriovenous malformations and giant intracranial aneurysms, which may predispose the patient to severe hemorrhagic complications as illustrated in our report.

Our institution's protocol in managing hyper-acute strokes recommends performing a non-contrast CT brain prior to administering the bolus dose of alteplase. This is closely followed by a CTA whilst the alteplase infusion is ongoing. Our experience with this patient emphasizes the need to look for other vascular abnormalities beyond large vessel occlusion, and if present, to dispense immediate corrective measures (such as the cessation of alteplase infusion, and the administration of frozen plasma) to ameliorate the risk of catastrophic complications.

DISCLOSURE

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