CASE REPORT

INTRACRANIAL ATHEROSCLEROSIS PRESENTING AS SUB ARACHNOID HEMORRHAGE - A UNIQUE SCENARIO

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ABSTRACT

Intracranial atherosclerotic disease (ICAD) represents the most common cause of ischemic stroke worldwide. Here, we present a unique manifestation of ICAD presenting as headache in a 53 years old hypertensive and diabetic patient. CT demonstrated sulcal sub-arachnoid hemorrhage (sSAH), with no infarct. He was referred to Dow University Hospital for CT Angiography examination, which revealed > 90% narrowing of the supraclinoid segments of Atherosclerotic internal carotid arteries (ICA) with extensive leptomeningeal collaterals, supplied by posterior circulation. To the best of authors' knowledge, only single case analogous to it, on DSA has been published in the International Radiology literature.

Key words: CT angiography, intracranial arteriosclerosis, neuroradiology, non-aneurysmal sulcal sub arachnoid hemorrhage, sSAH.

Introduction

ICAD represents the most common cause of ischemic stroke worldwide. Digital subtraction angiography (DSA) is regarded as the gold standard in assessing degree of stenosis in intracranial vessels. However, it is invasive and carries a risk of complications. We present the imaging features and rare manifestation of ICAD on cross sectional non-invasive imaging.

Case Report

A 53 year old male, known hypertensive and diabetic, presented to the Dow University Hospital with symptom of headache. According to the patient, the headache initiated during sexual intercourse, was continuous, severe in intensity, only partially relieved by analgesics. There was no history of motor or sensory deficit in past or at present time. His MRI Brain Plain showed subarachnoid hemorrhage with no intra ventricular extension (Fisher Grade 3) seen over the right fronto-parietal lobe with gyral swelling and sulcal effacement. No evidence of Diffusion restriction focus. There was a suspicion of peripheral cerebral aneurysm/ cortical venous sinus thrombosis and the patient proceeded to CTA (angiography) and CTV (venography) of the cerebral vessels.

CTA revealed multiple atherosclerotic calcified mural plaques are seen along the cavernous segment, petrous segments as well as in the left distal cervical segment of the internal carotid arteries causing significant greater than 90% luminal narrowing with non-visualization of the supraclinoid segments and luminal attenuation of the M-1 and M-2 segments of the middle cerebral artery, A-1 segment of the anterior cerebral artery and anterior communicating artery with fill-in of the distal M-3 segment of the middle cerebral artery and distal anterior cerebral artery with collateral branches form the posterior circulation.

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There is non-significant luminal narrowing less than 50% of the distal basilar and left posterior cerebral artery P-1 segment. Hypoplastic / non visualized bilateral posterior communicating artery also seen. Extensive leptomeningeal collaterals seen along with cerebral convexity, also in the suprasellar cistern along the branches of circle of wills. No evidence of contrast extravasation. No evidence of aneurysm, arteriovenous malformation or fistulous communication noted.

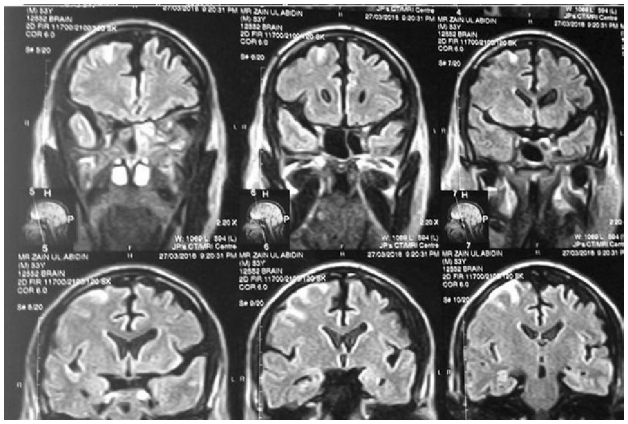

Figure 1: MRI coronal FLAIR images showing right convexity sSAH.

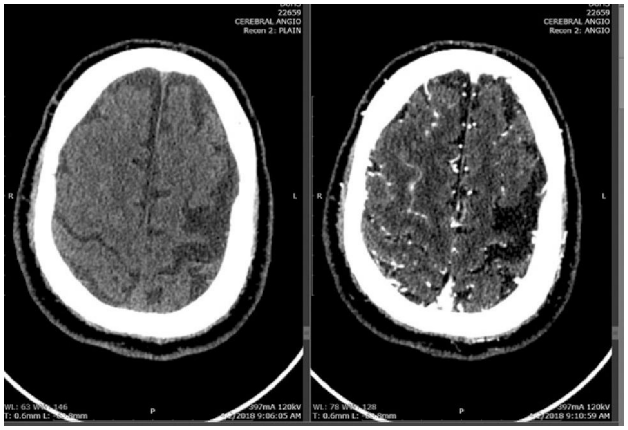

Figure 2: CT Brain axial images and CTA source images showing SAH and leptomeningeal collaterals.

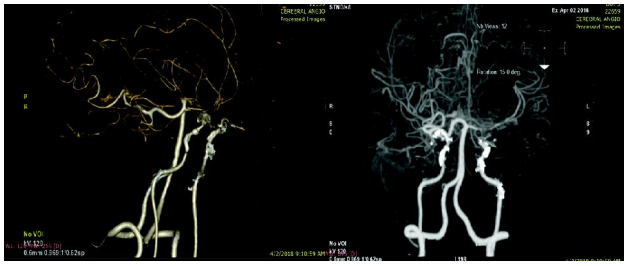

Figure 3: CTA MIP images showing significant ICA, MCA and ACA stenosis.

Discussion

Nonaneurysmal spontaneous sulcal subarachnoid hemorrhage (sSAH) is defined as an hemorrhage located in the convexity of the brain, without involvement of adjacent parenchyma or extension into inter hemispheric fissure, sylvian fissure, basal cisterns or ventricles. It is a rare cause of cerebrovascular disease and represents 5% of SAH.[1] There are multiple known etiologies for sSAH,[2] including vascular causes, namely, reversible cerebral vasoconstriction syndrome (RCVS), cerebral amyloid angiopathy (CAA), cerebral venous thrombosis, vascular malformations and Moyamoya disease or syndrome; and nonvascular causes as brain tumors, abscesses and coagulopathy. In older patients CAA is more prevalent.

Computed tomography angiography (CTA) is a rapidly developing technology with great potential. This is particularly true for evaluating neurovascular disease.[3] Compared to DSA, CTA has high sensitivity and specificity for detecting 50% stenosis of large intracranial arterial segments. CTA is minimally invasive;[4] performed quickly; less susceptible to motion artifacts than MRA; less dependent on hemodynamic effects compared to MRA; and more widely available in the general community than MRA and DSA.

Geraldes R in 2014,[5] et al conducted the study of imaging findings, etiologies, and long-term outcomes of case series of sSAH and found that only 2.3% of the 210 non traumatic SAH patients had clinical significant internal carotid artery (ICA) atheromatous stenosis, ipsilateral to sSAH.

Gouveia A et al[6] at 10th World Congress on Controversies in Neurology, Portugal in 2016 reported the case of a 54-year-old male patient, who presented with severe abrupt headache, followed two days later by right visual field defect and dysphasia. Head CT revealed left peri-rolandic sSAH and two hypodense lesions (left cortical parieto-occipital and left subcortical parietal). DSA found a severe MCA atherosclerotic stenosis.

This case report describes ICAD with extensive leptomeningeal collaterals over the cerebral convexities on CTA, resulting in non-aneurysmal sSAH at watershed zone. Possible pathophysiological mechanisms, being similar to that of Moyamoya disease that severe arterial stenosis may trigger the development of fragile

and dilated pial collaterals. Surprisingly, no infarct was seen and there were complete periventricular ischemic free zones, despite of severity of disease. DSA of the internal carotid arteries was in plan as confirmatory evidence. However, due to invasiveness of the procedure, it was abstained. Our patient got recovered by regime of analgesics and proper control of hypertension, and was followed for 6 weeks and remained symptom free. He was continued on conservative management.

Conflict of interest: None

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