

# Cerebral Infarction Following Traumatic Carotid Cavernous Fistula in Adolescent Male; a Case Report

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## Abstract

*Carotid cavernous fistula (CCF) is a pathologic communication between carotid artery and the cavernous sinus. It can cause a wide range of symptoms that could be life-threatening in rare cases. A 15-year old male was diagnosed with right sided CCF after severe road traffic accident and underwent surgical coiling procedure. Two years later, while doing investigations to determine the cause of recurrent syncopal attacks that the patient was suffering, he was diagnosed with cerebral infarction together with postural orthostatic tachycardia syndrome. The current report highlights the importance of follow up of patients with CCF.*

## I. BACKGROUND

Carotid cavernous fistula (CCF) is a pathologic communication between carotid artery and the cavernous sinus, causing intracranial blood to shunt from arterial to venous circulation[1]. Communications originating directly from the internal carotid artery are called direct (high flow) CCF while those originating from meningeal branches of internal or external carotid arteries are called indirect (low flow) CCF[2]. Radiologically, CCF is characterized by asymmetrical enlargement of cavernous sinus and typically associated with dilation of ipsilateral superior ophthalmic vein [3]. Etiologically, CCF frequently happens after trauma (75%) and sometimes happens spontaneously (25%)[2].

Traumatic CCF is considered a rare complication of craniofacial injuries accounting for only 0.2 to 0.3% of the cases [4]. More than 90% of traumatic CCF is of the direct type and usually have an acute presentation [5]. The majority of traumatic CCF develops one or more of the following cardinal symptoms; proptosis, chemosis, orbital bruits, and headache. This is usually accompanied by visual disturbances, orbital pain, or multiple cranial nerves dysfunction. While the course is usually benign, life-threatening complications such as intracranial hemorrhage, epistaxis, or cerebral ischemia may happen in rare cases. We are describing a rare case of CCF who was complicated with cerebral infarction.

## II. CASE REPORT

A 17-year old male patient had severe road traffic accident (RTA) involving the head in 2013. The patient was admitted to the intensive care unit in coma. After two-weeks the patient was conscious and transferred to the neurosurgery ward. The patient was complaining of headache over right temporoparietal area of the head associated with decreased visual acuity of the right eye. On examination, the patient was conscious and has stable vital signs. Heart and lung examination was unremarkable. The patient had proptosis, orbital bruits, and 6th and 7th cranial nerves palsy. History revealed benign rolandic epilepsy 5 years ago that was treated with tegretol. Catheter angiogram through right common carotid showed opacification of the aneurysm sac in right cavernous sinus and very early filling of bilateral cavernous sinuses as well as internal jugular veins. Additionally, there was grossly enlarged right superior ophthalmic vein which was also opacified with retrograde flow. There was no flow seen in middle or anterior cerebral arteries (Figure 1). The patient was then diagnosed with right sided CCF and underwent surgical coiling procedure. Post-surgical catheter angiogram through left vertebral artery showed no more filling of the aneurysm sac, cavernous sinus, superior ophthalmic vein or any other venous structure (Figure 2). The patient became clinically stable and was discharged home. The patient was followed at neurosurgery clinics for approximately two years with serial MRIs showing stable condition.

In 2016, the patient presented to the neurology department with a history of pressure headache not responding to regular analgesics associated with dizziness and unsteadiness. Due to previous history, the patient was admitted as a case of seizure for further investigations. During admission, the patient experienced frequent (at least once/day) transient loss of consciousness episodes that was witnessed by his family. The episodes were aggravated by walking, standing from seating position, and when turning the head to the right side while standing. Neurologic examination showed horner syndrome on the right side of the face (including miosis and partial ptosis) with right lower motor neurone (LMN) facial palsy. The

patient had persistent decrease of sensory functions of all modalities over left side of the face and upper and lower limbs. Electroencephalography over 30 minutes did not show any epileptiform discharges. Echocardiogram was normal and 24 hours of holter monitoring showed sinus tachycardia rhythm. Ear, nose, and throat (ENT) examination was not remarkable. Tilt table test showed postural orthostatic tachycardia syndrome (POTS), with dizziness and profuse sweating.

Imaging investigation was done to further clarify the diagnosis. Non-contrast brain CT brain showed ipsilateral focal hypodensity of the frontal lobe involving both grey and white matter, suggestive of the presence of infarction (Figure 3). Brain magnetic resonance imaging (MRI) showed an area of acute ischemia in right frontal lobe (Figure 4). Catheter angiogram through common carotid arteries and left vertebral artery showed no evidence of recurrence or residual of the CCF. The right hemispheric supply comes from right posterior communicating artery and the anterior communicating artery (Figure 5). The patient was then diagnosed with POTS and ipsilateral cerebral infarction following right CCF. The patient was started on fludrocortisone 0.2 mg/day for 10 days to control the POTS. The patient significantly improved with no more syncope and then discharged home. In follow up visits at the neurology clinic, the patient was stable and reported no more dizziness or recurrent syncopal attacks.

### III. DISCUSSION

We presenting here a case of unilateral CCF complicated with ipsilateral cerebral infarction in a 17-year adolescent male. The infarction of the right frontal lobe was discovered approximately two-years after traumatic CCF and during the investigations done to determine the cause of recurrent syncopal attacks. Neurological, cardiac, and ENT causes of dizziness and syncopal attacks were ruled out. POTS was confirmed using the Tilt table test. The disappearance of dizziness and syncopal attacks following fludrocortisone intake further confirm the POTS diagnosis. While it difficult to determine the exact cause of POTS, it could be due to sympathetic activation following reduced cerebral blood flow as happen in cerebral infarction [6].

We believe that the current case is rare and worth reporting because cerebral infarction is a rare complication of CCF. For example, in a series of 24 patients with traumatic CCF who underwent endovascular embolization, only one was complicated with infarction [7]. In another series of 28 patients with CCF who underwent endovascular embolization, none

of the patients developed infarction [8]. Additionally, infarction at young age is uncommon. For example, in the few case reports showing infarction following traumatic CCF, one was in 24-year old male [9] and the rest were in old aged males and females [7, 10].

The infarction occurred in our case was not due to failure of the surgical coiling procedure or recurrence of the CCF. It was most probably caused by the cerebral hypoperfusion because of early venous filling rather than arterial. The blood supply of the ischemic side comes from right posterior communicating artery and the anterior communicating artery. The delayed diagnosis of the infarction may indicate gradual course of the disease or missed diagnosis during the follow up.

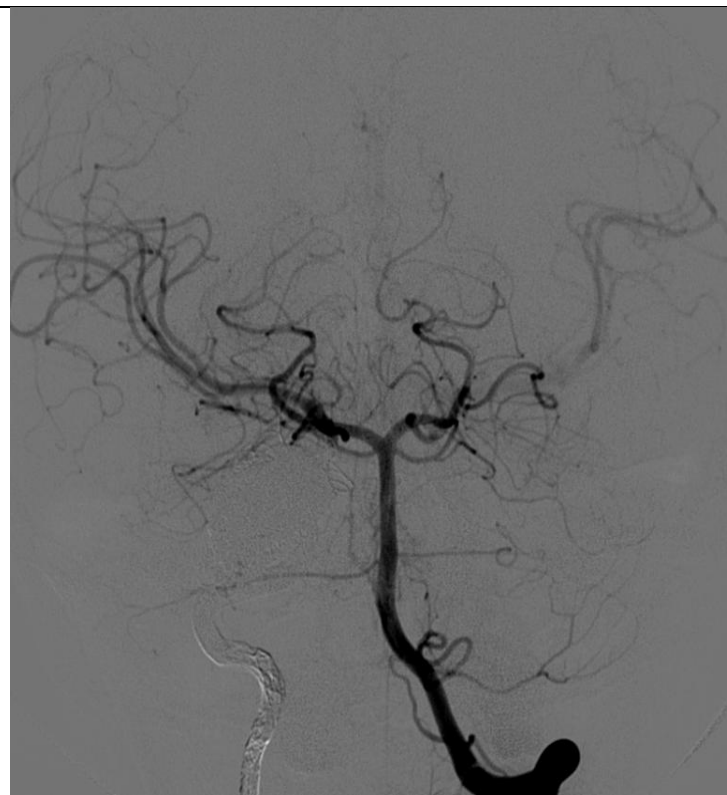
In conclusion, we are reporting a rare case of cerebral infarction diagnosed in an adolescent male two years after traumatic CCF. The current report highlights the importance of follow up of patients with CCF not only post-operatively but also over a longer period.

### REFERENCES

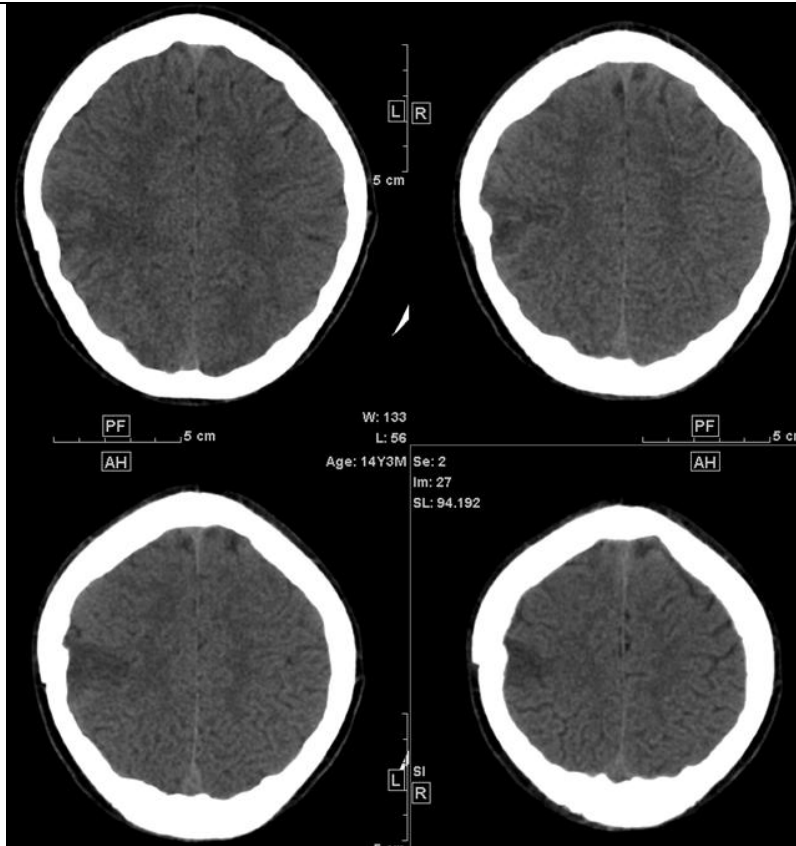
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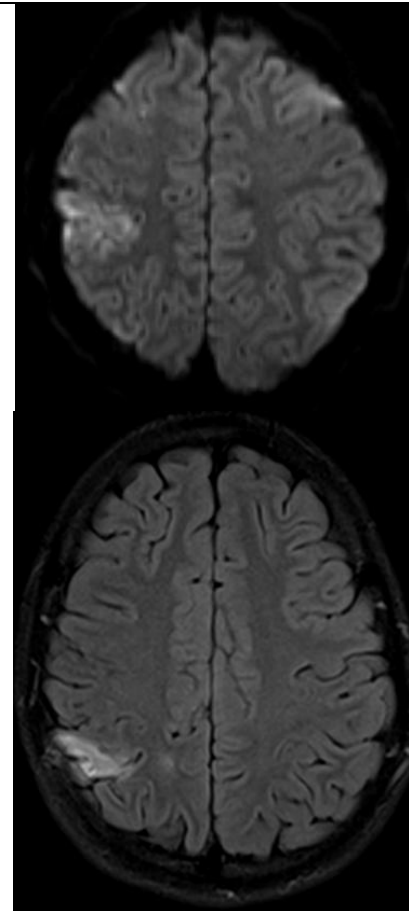
**Figure 1: Catheter Angiogram Through Right Common Carotid Injection. There Is Opacification Of The Aneurysm Sac In Right Cavernous Sinus And Very Early Filling Of Bilateral Cavernous Sinuses As Well As Internal Jugular Veins. Grossly Enlarged Right Superior Ophthalmic Vein Is Also Opacified With Retrograde Flow. No Flow Is Seen In Middle Or Anterior Cerebral Arteries.**



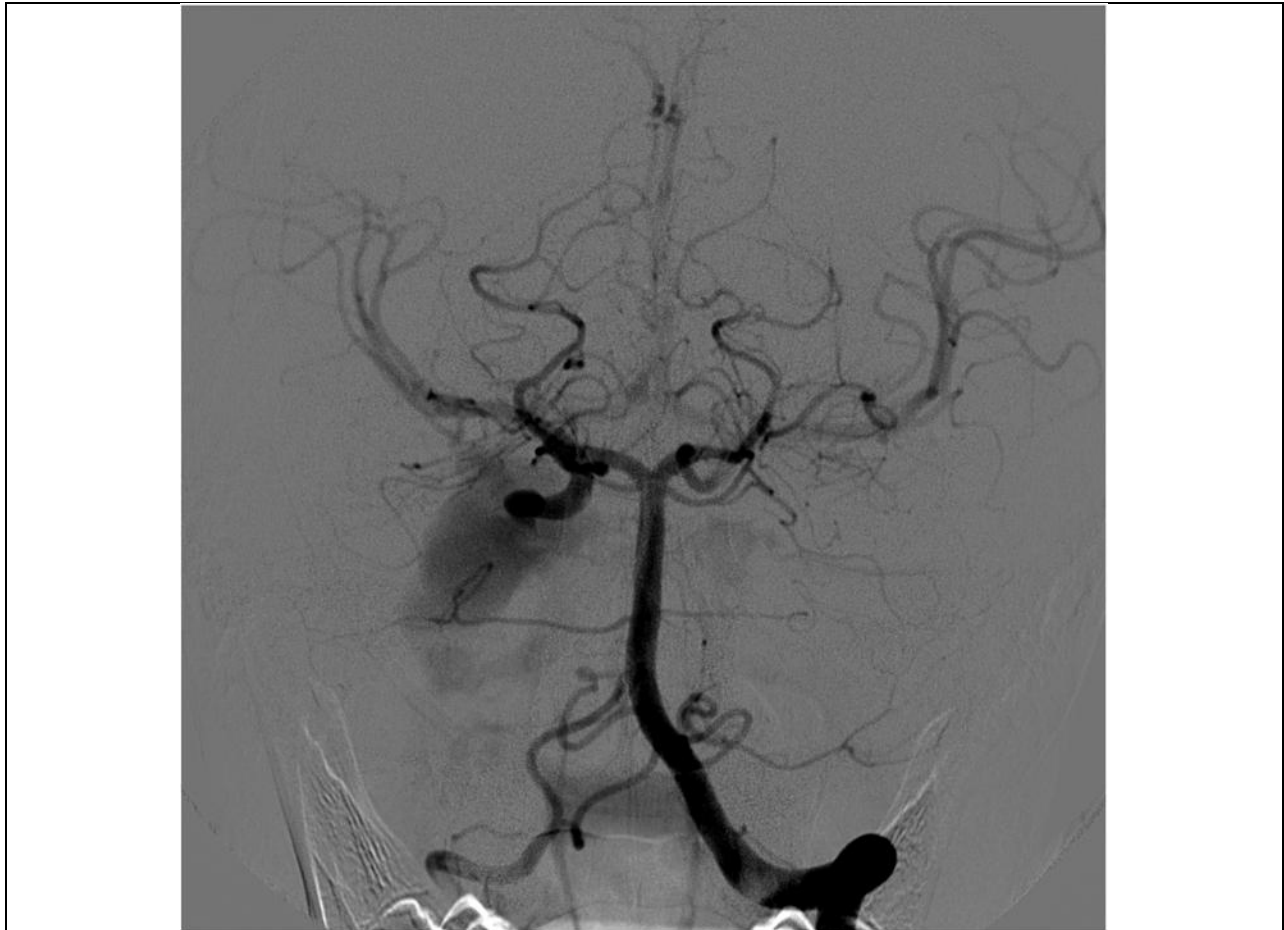
**Figure 2: Post Coiling Of The Aneurysm. Catheter Angiogram Through Left Vertebral Artery Injection. No More Filling Of The Aneurysm Sac, Cavernous Sinus, Superior Ophthalmic Vein Or Any Other Venous Structure.**



**Figure 3: Non-Contrast CT Brain After Traumatic CCF Showed Focal Low Density In Upper Right Frontal Lobe Involving Grey And White Matter Suggestive Of Infarction.**



**Figure 4: Diffusion Weighted Images Shows Areas Of Acute Ischemia In Upper Right Frontal Lobe On Left And FLAIR Images Shows Focal Abnormal Areas In Upper Right Frontal Lobe Involving Grey And White Matter Suggestive Of Infarction On Right**



**Figure 5: Catheter Angiogram Through Left Vertebral Artery Injection. Opacification Of Bilateral Anterior, Middle And Posterior Cerebral Arteries Indicates Adequate Flow Through Circle Of Willis. Aneurysm Sac Is Again Filling Due To Anteriorly Directed Flow Through Right Posterior Communicating Artery and The Anterior Communicating Artery.**