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Deep Cerebral Venous Thrombosis Associated with Callosal Hemorrhage-A Rare Case Report

Dr. Prabhjot Singh[1], Dr. Kumar Sambhav[2], Dr. Renu Gupta[3], Dr Ashish Kumar Nayyar[4], Dr. Surajit Ghatak[5]

[1] Junior Resident, Department of Anatomy, All India Institute of Medical Sciences, Jodhpur 342005, Rajasthan, India

[2]Senior Resident, Department of Anatomy, All India Institute of Medical Sciences, Jodhpur 342005, Rajasthan, India
ORCID ID: 0000-0003-0012-3994
Corresponding author Email: *drkrsambhavaiims[at]gmail.com*
+918877195979

[3]Additional Professor, Department of Anatomy, All India Institute of Medical Sciences, Jodhpur 342005, Rajasthan, India ORCID ID: 0000-0002-5581-1615

[4]Additional Professor, Department of Anatomy, All India Institute of Medical Sciences, Jodhpur 342005, Rajasthan, India
ORCID ID: 0000-0001-9937-4472

[5]Professor and Head, Department of Anatomy, All India Institute of Medical Sciences, Jodhpur 342005, Rajasthan, India
ORCID ID: 0000-0003-2768-7984

Abstract: ***Deep cerebral venous thrombosis manifesting as corpus callosal haemorrhage is a rare entity. Such involvement is best explained anatomically. We describe a case of a 56-year-old female presenting with headache, significant impairment of sensorium and nonambulatory paraparesis. The patient succumbed immediately after being put on mechanical ventilation. Brain dissection showed significant callosal haemorrhage with extensive disruption. There were signs of venous involvement affecting rostrum and genu with defacement of margins. The right hemisphere unlike the left was defaced entirely with no signs of vessels or the anterior part of corpus callosum. Anatomical observation revealed signs of deep cerebral vein thrombosis with a larger hemorrhagic zone in the right hemisphere with visible vessels and involved venous channels on the contralateral side. The case was ascertained to be a case of corpus callosal haemorrhage attributing to deep cerebral venous thrombosis. A rare event such as this should always be followed up with imaging to improve our document for further understanding.***

Keywords: deep cerebral venous thrombosis, callosal hemorrhage, corpus callosum

1. Introduction

Corpus callosum (CC) being the largest white mattercommissural fiber is composed of 150-200 million fibers which connects homotopic parts of contralateral cerebral hemispheres with one another. CC is divided into four parts, namely; rostrum, genu, trunk and splenium [1]. CC receives it blood supply from three main sources: the anterior cerebral artery (ACA), pericallosal artery and posterior pericallosal artery [2]. The latter two arteries serve as the source of main arterial supply to CC [2]. The hemorrhage of CC is rare but possible. Main causes of callosal hemorrhage are – trauma, hypertension, arteriovenous malformations [3]. Deep cerebral venous thrombosis (DCVT) is an extremely rare entity which leads to the callosal hemorrhage [4]. There are just a handful of cases of DCVT reported which led to callosal hemorrhage.

In this case report, we have reported a possible case of callosal hemorrhage associated with DCVT in which the hemorrhage was found to be extending to corpus callosum and lateral ventricles.

2. Case Presentation

A 56-year-old female who had presented to the emergency with history of increasing generalised headache for seven days and drowsiness for four days. There was significant impairment of sensorium and non-ambulatory paraparesis. General examination revealed unstable vitals, severe pallor, signs of dehydration, left hemiparesis with GCS E2V3M3. She succumbed within minutes of admission shortly after being out on mechanical ventilation. Blood analysis, CT/MRI werenot performed. There was however no history of Diabetes Mellitus, Hypertension, hypothyroidism, coronary artery disease. No significant family history was recorded. The deceased had previously pledged herself for body donation upon death to the Voluntary body donation programme run by Department of Anatomy, All India Institute of Medical Sciences (AIIMS), Jodhpur. Upon dissection of the brain, Callosal hemorrhage with extensive disruption was observed.

The haemorrhage was localised to the anterior part of corpus callosum – rostrum, genu and anterior part of body of CC. The limits of the anterior part of CC were defaced whereas limits of posterior part of body of CC and splenium were intact (Figure 1). The haemorrhage was seen to be extending into the bilateral lateral ventricles whereas the third and the fourth ventricles were spared. The arteries which supply the CC appeared intact and no evidence of rupture was observed. Since these are present along the periphery of CC and seemed intact, arterial haemorrhage can be ruled out. The superior and inferior sagittal sinus appeared intact whilst brain extraction.

The main venous draining the corpus callosum is internal cerebral vein. The anterior and posterior septal veins draining the splenium were not visualised as the septum pellucidum was stripped during dissection. The septal veins join the ICV which in turn unite to form the Vein of Galen which also seem. Upon further study of the base of skull, there was no sign of rupture of basal vein of rosenthal. No vessels were visualised in the right callosal region along with extensive defacement of margins of CC which fails us to provide an accurate anatomical analysis (Figure 2). No evidence of ICV and a greater hemorrhagic zone anterposterioly leads us to conclude that the ICV, Vein of Galen along with septal veins was most probably involved to a larger extent in the right hemisphere. Though the left hemisphere appears involved but also presents with few intact vessels. The most probable cause of this hemorrhage is DCVT of internal cerebral vein.

At the site of hemorrhage, in the left cerebral hemisphere indentation was found along with the ruptured vessel. Whereas, on the contralateral hemisphere protrusion was observed. The total length of left and right cerebral hemisphere from frontal pole to the occipital pole was noted to be 140mm and 136mm respectively. The depth of indentation of left hemisphere was measured at three points, at the centre of indentation, towards the anterior end of indentation and towards the posterior end of indentation and was found to be 9.18mm, 7.57mm and 14.65mm respectively. The dimensions of hemorrhagic zoneon right hemisphere were 65.52mm * 28.90mm and on left hemisphere were50.56mm * 34.11mm (anterior-posterior * superior-inferior direction) respectively. Discoloration was also observed on right hemisphere extending beyond the hemorrhagic zone in superior and inferior directions, the total length of discoloured zone was 52.72mm. The distance from anterior pole of frontal lobe to the posterior margin of splenium on right and left hemisphere was 97.77mm and 97.58mm. The distance from superior most point on cerebral hemisphere to the point just inferior to the site of hemorrhage on right and left hemisphere was 74.42mm and 80.45mm respectively.

3. Discussion

Hemorrhage of CC is uncommon and is usually related to damage of corpus callosum due to extension of hemorrhage from other parts of cerebrum [5]. Handful of cases have been reported regarding hemorrhage of CC secondary to DCVT [4, 6]. Various common causes of hemorrhage of CC have been reported till date and these include head injury [3], cerebral infarction in the area supplied by ACA secondary to rupture of arteriovenous malformation [5], tumors of brain [7], secondary to corpus callosotomy done for intractable seizures [8], moyamoya disease [9], Marchiafava-Bignami disease [7], multiple sclerosis [10] and many others.

Kulkarni *et al*, studied 63 cases of DCVT over approximately six years. Out of 63 cases only two of them led to callosal hemorrhage. They concluded that although rare but DCVT could also serve as the cause of callosal hemorrhage. One case was associated with DCVT of internal cerebral veins, vein of Galen and straight sinus with the hemorrhagic damage of genu of CC. Other case was associated with DCVT of left transverse sinus and deep cerebral vein with hemorrhagic damage to genu of CC [4]. Ganeshbhai *et al* also reported a case of venous thrombosis which led to the hemorrhage of CC involving the genu of CC [6]. The findings of involvement of genu of CC secondary to DCVT by Kulkarni *et al* and Ganeshbhai*et al* match our observation.

Kasahara *et al*, published a case report regarding a case of hemorrhage restricted to CC. In a case of 87-year-old female patient who has cerebral hemorrhage which was restricted to CC. The patient had history of hypertension, which the authors believe could had led to bleeding from pericallosal artery. On admission, GCS was E4, V4 and M6 with no motor ataxia but the movements of left arm of the patient were synchronised with the movements of her right arm. They labelled these symptoms as dyspraxia. Neurological findings suggested that the lesion of CC was restricted to the body of CC only, unlike in the current study where entire CC was involved except the splenium [11]. Similar symptoms were also reported by Nishikawa *et al [*12].

Damage to posterior part of CC, mainly the splenium is associated with development of diagnostic dyspraxia as well as callosal apraxia. Multiple authors have studied the involvement of splenium and have established these facts [7, 13–19]. In current study posterior part of body and complete splenium was found to be normal along with normal occipital lobe. Diagnostic dyspraxia was not ascertained in our case.

Kim *et al*, studied a case of callosal hemorrhage involving the genu and body of corpus callosum following hemorrhage from anterior communication artery. Diffusion tensor tractography (DTT) showed extensive disruption of CC including the genu and whole body of CC. Further, their MRI scans revealed intracranial and sub duralarachanoid hemorrhage of genu and body of CC [20]. Coordination between posterior parietal lobe, supplementary motor area and transcallosal inhibitory influence is essential for making appropriate movements [21]. Alien hand syndrome (AHS) attributed only to callosal disconnection occurs with lesion to middle of body of CC. MRI is highly sensitive and specific tool for determination of level of brain injury but sometimes it is difficult to establish the degree of injury due to various reasons, such as, perilesional edema, penumbra lesion and volume effect of stroke [20]. In our case too there was severe disruption of callosal fibers of genu and body with no reported signs of involuntary movements. Further clinical picture of AHS wasn't assessed owing to low GCS. Kim *et al*, found hypermetabolism of right hemisphere which they presumed was due to extensive injury to left hemisphere and interruption of transcallosal inhibition [20]. In our case the involvement of right hemisphere appears significantly more.

Leiguarda *et al*, in their study, reported finding of parapares is as common in cases of callosal hemorrhage [5]. Our case also had presented with non-ambulatory paraparesis on admission.

Such constellation of clinical signs and symptoms along with findings of brain dissection lead us to conclude it to be a case of DCVT manifesting as corpus callosal hemorrhage.

4. Conclusion

Corpus callosal hemorrhage is ascertained as a rare implication of deep cerebral venous thrombosis. DCVT is tedious to diagnose with varied signs and symptoms with CT/MRI imaging aiding in diagnosis.

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Conflict of Interest

There are no potential conflicts of interest to declare by the authors of this study.

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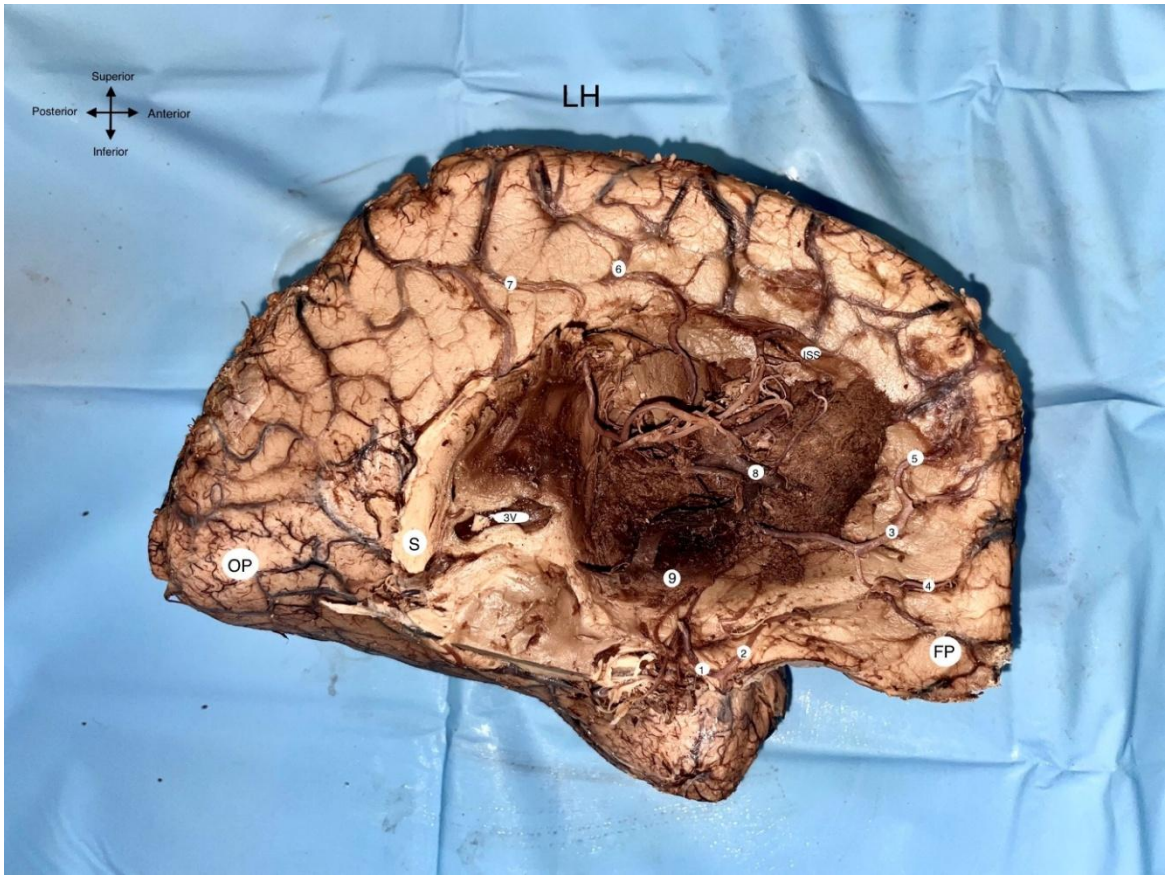

Figure 1: Sagittal view of dissected left brain hemisphere showing extensive disruption of CC
1Anterior Cerebral artery; 2Orbitofrontal artery; 3Callosomarginal artery; 4 Frontopolar artery; 5Posteroinferior frontal artery; 6Superior Parietal artery; 7Inferior Parietal artery; 8Internal Cerebral Vein; 9Vein of Galen; ISS-Inferior sagittal sinus; 3V-Third Ventricle; S-Splenium; OP-Occipital pole; FP-Frontal pole; LH-Left hemisphere

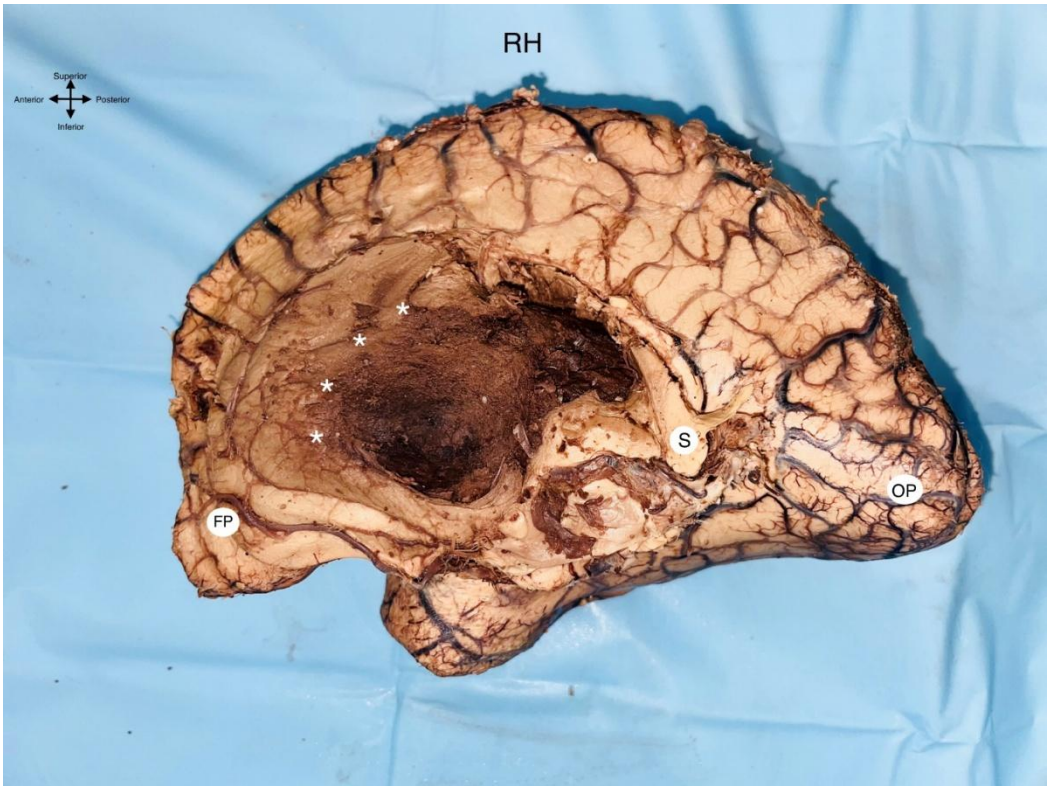

Figure 2: Sagittal view of dissected right brain hemisphere showing extensive defacement of CC and non visualisation of vessels
S-Splenium; OP-Occipital pole; FP-Frontal pole; RH-Right hemisphere; *-Denoting extensive defacement of margins of CC