

## Midline Watershed: Unusual Border-Zone Infarct of the Corpus Callosum

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### Abstract

Ischemic lesions involving the Corpus Callosum (CC) are rare. Symptoms are nonspecific and clinical findings vary depending on the site and size of the lesions. We describe a rare case of watershed infarct involving the whole corpus callosum but sparing the more common border-zone infarcts between the anterior cerebral artery–middle cerebral artery and the middle cerebral artery posterior cerebral artery and review the relevant literature with special emphasis on the vascular anatomy of the corpus callosum.

**Keywords:** Corpus callosum; Hypercholesterolemia; Exotropia

### Case Report

46-year-old man with diabetes, hypertension, hypercholesterolemia, and end stage renal disease presented with acute confusion. His BP was noted to be 60/28 mm Hg. He opened his eyes to verbal commands. Right pupil was 5 mm and the left pupil 4.5 mm, both reactive to light. Exotropia and hypotropia of the right eye, and roving eye movements were noted. He withdrew all extremities to pain. DTR's were noted as 3/4 in the left upper and 2/4 in the right upper limbs, and absent in the lower limbs. Babinski's sign was absent. MRI of the brain showed hyper intensity involving the entire corpus callosum on DWI (Figures 1 and 2). MRA showed stenosis of the proximal A1 segment of the right ACA at its bifurcation with a hypoplastic right PCA (Figure 3). The patient subsequently had a pulseless electrical activity code and died.

### Discussion

Infarcts of the corpus callosum are uncommon and are attributed to a rich blood supply from three main arterial systems: The anterior communicating artery, the pericallosal artery and the posterior pericallosal artery [1,2]. The pericallosal branches of the Anterior Cerebral Artery (ACA) supply the major portion of the body of the CC. Perforating branches of the ACA supplies the rostrum and genu [3]. The pericallosal branch may reach as far back as the foramen of Monro. It gives rise to four branches: the callosal, cingulo callosal, long callosal, and recurrent callosal arteries [4-5]. There are 3 to 23 cingulo callosal arteries per hemisphere that supply anterior parts of the corpus callosum; and form abundant anastomoses.

The splenium of the CC is supplied by the posterior callosal branch of the Posterior cerebral artery (PCA) or the splenic artery. They take

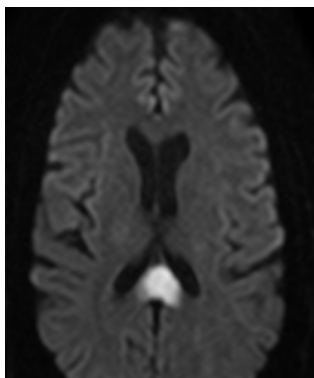


Figure 1: DWI showing infarct of the splenium of the corpus callosum.

off perpendicularly from the PCA [1,6,7]. An accessory posterior pericallosal artery has been described [6]. Anastomosis of the ACA and PCA branches occurs at the tip of the splenium in 75% to 100% of the cases [7,8].

The anterior communicating artery also supplies the CC [3,9]. The subcallosal artery provides blood supply to the medial portions of the rostrum and genu of the CC. The median callosal branches supply the rostrum, genu, and body of the CC and medial aspect of the frontal and parietal lobes [1,10]. The perpendicular origins of the pericallosal vessels make it difficult for the emboli to enter these vessels. Unusual features include arteriole-venule pairs, perivascular fibrous alae, and recurrent companion arterioles, providing resistance to atherosclerosis [10]. Pericallosal pial plexus also adds stability [4,11-13]. Chrysikopoulos et al. described only three diffuse lesions involving the genu and body of the CC [1]. The splenium was the most common site followed by the body and genu. This can be explained by the fact that the final anastomosis between the ACA and PCA occurs at the tip

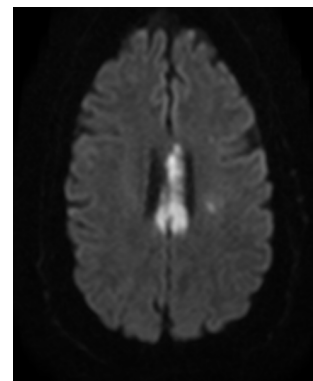


Figure 2: DWI showing infarct of the corpus callosum.

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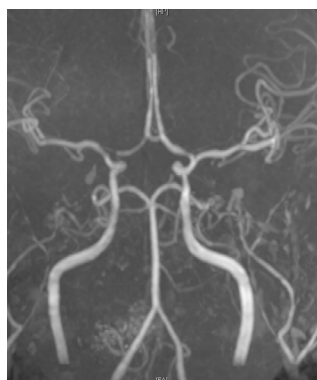


Figure 3: MRA showing stenosis of the proximal right ACA.

of the splenium. Anatomical-clinical correlations are challenging. In most cases, infarcts occur simultaneously in other arterial distributions. A hypoplastic A1 segment may facilitate occurrence of embolism in the anterior cerebral artery distribution [3]. In the described case the stenosis noted in the ACA on MRA along with the hypoplastic PCA may have played a predisposing role in the presence of hypotension.

## Conclusion

Border-zone infarcts involving the whole corpus callosum are rare because of its relative resistance to small-vessel disease and extensive vascular anastomoses [2,14-18]. Certain circumstances including isolated stenosis of arteries and systemic hypotension can precipitate this unusual watershed infarct. Understanding the vascular anatomy of the CC is the key to diagnosis.

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