

# Border-zone Infarct of the Corpus Callosum: A Case Report, Vascular Anatomy & Review of Literature

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**Abstract** Corpus callosum infarctions (CCI) are rare, accounting for only 3% of all ischemic strokes. CC derives its rich blood supply from anterior and posterior circulations with extensive anastomosis near the splenium tip, providing inherent protective redundancy to reduced blood flow. The presence of intracranial atherosclerotic disease (ICAD) impairs this cerebrovascular reserve, increasing susceptibility to ischemia and subsequent infarction. The border zone is more hemodynamically unstable when both circulations are affected simultaneously or in the presence of congenital anatomical variations in the cerebral vasculature. In such circumstances, accentuated hypoperfusion of the cerebral border zones secondary to systemic hypotension results in the development of border-zone infarct of the CC. We present a 47-year-old male with a midline border-zone infarct of CC due to diffuse ICAD with superimposed systemic hypoperfusion in the peri-procedural period of coronary artery bypass grafting (CABG). Due to the peculiar vascular involvement by ICAD, the infarction was isolated to CC, sparing the more common cortical border zones. Following the case report is a review discussing various anatomical locations, classifications, and pathophysiology of border-zone infarcts, with a specific focus on the vascular anatomy of the corpus callosum.

**Keywords:** *corpus callosum, border zone infarct, systemic hypoperfusion, intracranial atherosclerotic disease*

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## 1. Introduction

Corpus callosum infarction (CCI) is less frequently reported in the literature, accounting for only 2.9% to 8% of all ischemic strokes. [1,2,3,4] Strokes isolated to the corpus callosum (CC) are even rarer, ranging from 0.4% to 7%. [1] CC derives its rich blood supply from anterior and posterior circulations with extensive collateralization between the two circulations near the tip of the splenium. [1,4,5,6] This anterior cerebral artery (ACA)-posterior cerebral artery (PCA) anastomosis provides inherent protective redundancy to reduced blood flow, making CCI less common. [3,4,5,6] However, in the presence of significant intracranial atherosclerotic disease (ICAD), zone of impaired cerebrovascular reserve is formed, increasing susceptibility to ischemia and subsequent infarction. Border-zone is more hemodynamically unstable when both circulations are affected simultaneously or in presence of congenital anatomical variations in the cerebral vasculature. In such circumstances, accentuated hypoperfusion of the cerebral border-zones secondary to systemic hypotension results in a development of border-zone infarct of the corpus callosum. [7,8] Although most of the CC lesions are attributed to toxic-metabolic insufficiencies, lesion

developed in the peri-procedural period with the potential for prolonged and severe systemic hypotension such as Coronary artery bypass grafting (CABG), and especially in patients with significant ICAD, border-zone infarct must always be considered as one of the differential diagnoses. Moreover, CCI presents as clinically challenging to recognize neurological symptoms evolving over a few days to weeks with radiological features of mass-like effect or enhancement across the midline, often resembling the presentation of a tumor. [9,10,11] Knowledge of the vascular anatomy of the CC is the key to accurate diagnosis and avoiding unnecessary and potentially invasive workups such as brain biopsy. Also, a better understanding of the various locations and underlying mechanisms of border-zone infarction would help identify those patients at high-risk and provide an evidence-based rationale for preventing the occurrence and progression of border-zone infarction.

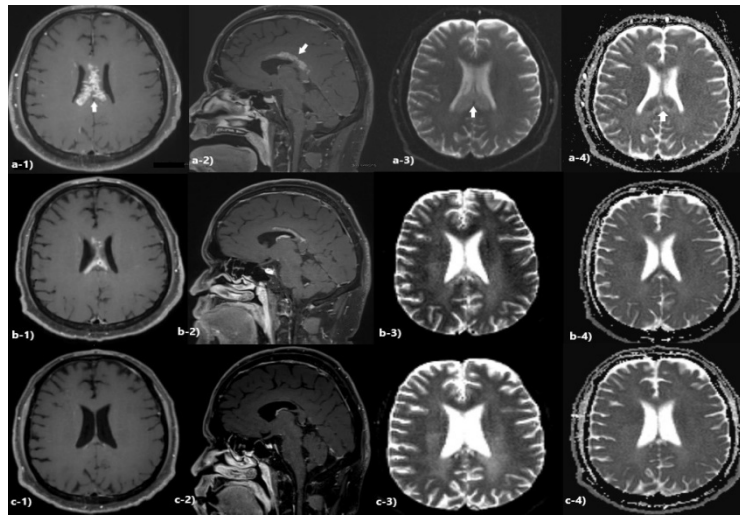
This review article outlines an unusual case of midline border-zone infarct in the corpus callosum during the peri-procedural period of CABG. Unlike typical border-zone infarcts between ACA-MCA and MCA-PCA, this case spared those areas due to unique vascular involvement related to intracranial atherosclerotic disease. Following this case report, the review discusses relevant literature, detailing anatomical locations, classifications, and pathophysiology of border-zone infarcts, with a specific

focus on the vascular anatomy of the corpus callosum.

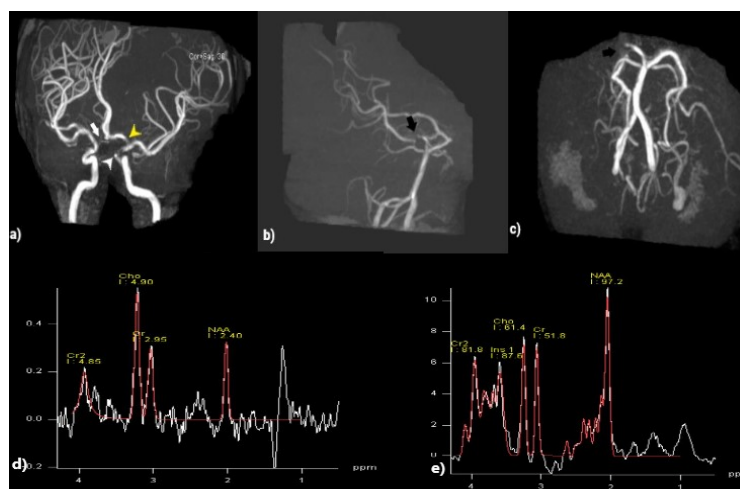
## 2. Case Report

A 47-year-old right-handed man with vascular risk factors of Hypertension (HTN), Diabetes Mellitus (DM), Hyperlipidemia (HPLD), Coronary Artery Disease (CAD) post Percutaneous Coronary Interventions (PCI) and Coronary Artery Bypass Grafting (CABG) and End Stage Renal Disease (ESRD) requiring hemodialysis presented with progressive left hemiparesis and facial droop first noted a day after he underwent an elective CABG. Neurological examination revealed left facial droop, left hemiparesis, symmetric 2+ deep tendon reflexes in all four extremities with a positive Babinski sign on the left. The National Institute of Health Stroke Scale score (NIHSS) was 4. MRI brain showed diffusion-restricting and contrast-enhancing lesions in the CC (splenium and body of the corpus callosum bilaterally, R>L) and minimal periventricular white matter hyperintensities of presumed

vascular origin. (Figure-1A) MRA head and neck showed diffuse intracranial atherosclerosis resulting in critical steno-occlusive disease of the right distal P1/proximal P2 segment, right ACA A1 segment and left supraclinoid internal carotid artery (ICA). (Figure 2a-c) MR spectroscopy reported "decreased NAA with reverse choline to creatinine ratio and lactate peak which can be seen in intracranial neoplasms such as high grade glioma vs lymphoma. (Figure 2d,e) However, subsequent MRI brain after 1 month showed an interval decrease in the lesion size, restricted diffusion and contrast-enhancement representing an evolving infarct rather than a tumor. The patient was started on dual antiplatelet therapy for secondary stroke prevention. The patient's neurological deficit improved with mild residual deficits of left sided weakness with NIHSS score of 3 at a 3-month follow-up. Repeat MRI brain (02/27/2023) showed near complete resolution of contrast enhancement and diffusion restriction (Figure 1) along with reversal of hunter's angle on MRS which is suggestive of neuronal cell death and regeneration further supporting the diagnosis of infarct.



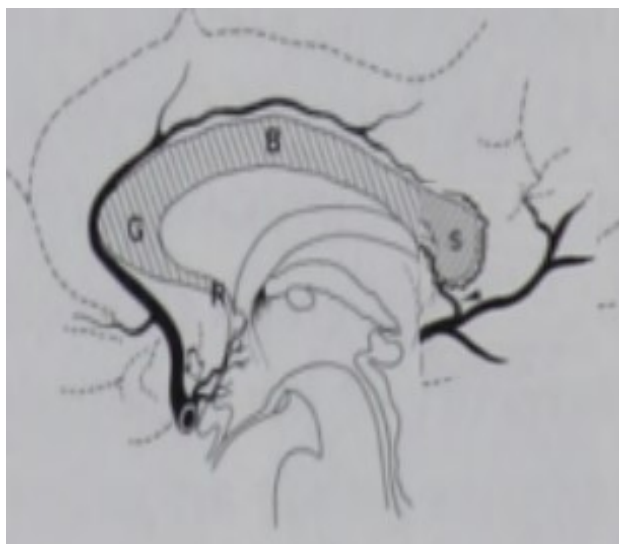
**Figure 1.** Series a), b) and c) are the MRI brain w/o contrast images taken at 0,1 and 3 month follow up showing bilateral corpus callosum lesion R>L with post contrast enhancement on axial T1 (a-1), sagittal T1 (a-2) and restricted diffusion on DWI (a-3) along with ADC correlate (a-4). Repeat MRI brain after 1 month showed interval decrease in contrast enhancement (b-1,2) along reduced diffuse restriction (b-3,4). Follow up MRI brain at 3 months showed near complete resolution of contrast enhancement and diffusion restriction



**Figure 2.** Magnetic Resonance Angiography (MRA) 3D reconstruction images showing a) right A1 stenosis (white arrow), left A1 focal stenosis (white arrow head), and left ICA stenosis (yellow arrow head), b) right P1 stenosis (black arrow) on posterior spin sequence, and c) right P1 stenosis (black arrow) on posterior tumble sequence. MR spectroscopy at 0 (d) and 3 months (e) follow up showing reversal of hunter's angle which is suggestive of neuronal cell death and regeneration

### 3. Discussion

CC derives its rich blood supply from both anterior and posterior circulations with extensive collateralization from both the circulations. [1,2,3,4,5,7,8] Pericallosal artery, a branch of ACA supplies the body of CC, subcallosal and medial callosal arteries. Branches of anterior communicating artery (AcomA) supply genu and rostrum. Whereas the posterior pericallosal artery (branch of PCA) supplies the splenium. The anastomosis of the ACA and PCA branches occurs at the tip of the splenium. [1,4,5,6,7,8] (Figure 3) This ACA-PCA anastomoses provides innate protective redundancy, making CCI less likely. [2,3,7] However, when diffuse intracranial atherosclerosis with predominant/selective involvement of these arteries result in impaired cerebrovascular reserve and increased susceptibility to ischemia. The hemodynamic compromise of further accentuated when arteries from both circulations are affected simultaneously or in case of anatomical variations. [7] In such circumstances, any disturbance in cerebral circulation such as systemic hypotension might result in border-zone infarct of CC.



**Figure 3.** vascular anatomy of corpus callosum showing anastomosis between anterior pericallosal artery (branch of ACA) and posterior pericallosal artery (branch of PCA). [8] R: rostrum; G: genu; B: body; S: splenium

In the present case, Large artery atherosclerosis (LAA) resulting in proximal right A1 segment stenosis was noted along with hypoplastic right P2 segment, thus involving both anterior and posterior circulation resulting in focal hypoperfusion of the right CC. This made CC vulnerable to reduced cerebral perfusion secondary to systemic hypotension in the intra/peri-procedural period of Coronary artery bypass grafting (CABG) resulting in border-zone infarct of CC. Selective involvement of cerebral vasculature by ICAD made border-zone involving CC more susceptible to infarction as compared to traditionally known superficial and deep border-zones as noted in this case. In our case, the splenium was affected as compared to the body of CC, which can be hypothesized by the fact that final anastomosis between the ACA and PCA occurs at the tip of the splenium,

making it the first area susceptible to hypoperfusion. [1,4,5,6,7] A similar case was reported by Ambadi et al., [7] who described a patient with bilateral border-zone infarct of CC in the setting of systemic hypotension secondary to ESRD. Patient was found to have stenosis of the proximal right A1 segment along with hypoplastic right PCA predisposing the patient to develop border-zone infarct of CC.

Border-zone infarctions are seen more frequently in patients with postcardiac surgery stroke than in the general stroke population (over 40% versus 2% to 5%, respectively). [9] In general, unilateral border-zone infarctions are more common in global hypoperfusion states, often occurring on the side of focal vascular stenosis. [13] However, bilateral border-zone infarctions can also occur in the event of prolonged severe hypotension such as CABG even in the absence of underlying stenosis. [9,14,15] Moreover, prolonged CABG was proven to be a risk factor for bilateral border-zone infarctions, perhaps due to a greater incidence of hypoperfusion during prolonged CABG. [9] In the present case, pt underwent CABG during which he was severely hypotensive requiring multiple blood transfusions and intra aortic balloon pump, thus explaining significant and prolonged cerebral hypoperfusion on top of impaired cerebrovascular reserve attributed by ICAD. The extent of infarction depends on the severity and duration of hypoperfusion, the location and severity of occlusive vascular disease, and the adequacy of collateral blood supply. [9] More prominent right sided focal stenosis explains the greater involvement of right CC compared to left.

Lesions in CC can present with a variety of clinical manifestations including focal neurological deficits including disconnection syndrome, seizures, encephalopathy and headache. [1,2] It can also present with non-specific symptoms, slowly evolving over a few days to weeks, often resembling the presentation of a tumor. [6,10,11,12] Our patient presented with L-sided hemiparesis with facial droop with a progressive clinical course. DWI showed diffusion restriction and contrast-enhancing lesion of the bilateral CC. MR spectroscopy showed decreasing NAA with reverse choline to creatinine ratio and lactate peak. Hence, neoplasms such as lymphoma were high on the initial differentials. However, a repeat MRI after four weeks showed an interval decrease in the lesion size with resolving edema and contrast-enhancement representing an evolving infarct rather than a tumor. A similar case was reported in the literature by Kasow et al [10] where the patient developed CCI, however, was misdiagnosed with a tumor and underwent biopsy. Hence, anatomical-clinical correlations of CCI are challenging. Knowing the vascular anatomy of the CC is the key to correct diagnosis and avoiding invasive workup such as biopsy with significant morbidity and mortality. [10,11,12]

Diffusion restricted lesions in CC has a wide differential diagnosis. While vascular causes are the most common, other less common etiologies include neoplasm, toxic metabolic derangements, trauma, infection, demyelination and inflammation. [16] Majority of CCI were secondary to Cardio-embolism and ICAD, with other mechanisms like small vessel disease being less common. [17,21].

## 4. Border-zone Infarcts of Cerebral Hemispheres

Border-zone infarcts occur at the borders between two neighboring cerebral arterial territories where the tissues are vulnerable to reductions in perfusion. They constitute approximately 10% of all cerebral infarcts. [13,17,20] They may be precipitated by several different mechanisms, however cerebral hypoperfusion is the most frequent cause. [13,14,17,20,21] They are classified into two broad categories (a) external or superficial (cortical) and (b) internal or deep (sub-cortical) border-zone infarcts. (Figure 4)

### (a) Cortical border-zone infarcts:

The external or cortical border zones are located at the junctions of the three main cerebral arteries namely, 1) Anterior border-zone infarcts (between ACA&MCA), and 2) Posterior border-zone infarcts (between MCA&PCA), and 3) Paramedian white matter (between ACA&MCA). [13,14,17,20,21]

Conventionally, the hemodynamic compromise produced by episodes of profound hypotension in the presence of a severe ICAD is believed to cause impaired cerebral perfusion preferentially affecting border zones resulting in ischemia and subsequent infarction. [14,20,21,22] However, recently published studies found no evidence of hemodynamic impairment in cortical border-zone infarcts. [23] A new mechanism has been emphasized where the formation of microemboli in presence of ICAD has been associated with cortical border-zone infarcts. [13,17,22] Cortical border-zones are under perfused in the presence of ICAD resulting in reduced clearance of the microemboli when they propagate through these areas, thus enabling ischemia to develop. [17,22] Therefore, even in the absence of severe stenosis, reduction of blood flow can result in ischemia due to decreased clearance of microemboli. Recently, there were many studies published that directly compared cortical border-zone infarcts to the internal border-zone infarcts. [17,21,22,23,24,25,26,27] According to these studies, cortical border-zone infarct is invoked by a combination of two interrelated processes i.e. hypoperfusion and embolization. [21,22,23,26] Isolated cortical border-zone infarcts in ICAD are believed to be caused by an embolic phenomenon, whereas cortical border-zone infarcts coexistent with internal border-zone infarcts are believed to be secondary to focal cerebral hypoperfusion. [21,22] As MCAs and ACAs arise from the ICAs, steno-occlusive disease of the ICA makes anterior border-zones more susceptible resulting in anterior border-zone infarcts more common than posterior border-zone infarcts. Any contralateral ICA disease and/or inefficient collateralization, particularly affecting the anterior portion of the circle of Willis, will further add into this intrinsic vulnerability of the anterior border-zone infarcts. [22] Conversely, stenosis or occlusion of the vertebrobasilar system, or a fetal-type PCA, may favor the involvement of the posterior border-zone infarcts, which probably accounts for individual observations of preferential involvement of the posterior border-zone infarcts. [22]

On MR imaging, the cortical border-zone infarcts usually appear to be wedge-shaped or ovoid. [13,17,21,22] However, their location may vary with anatomical

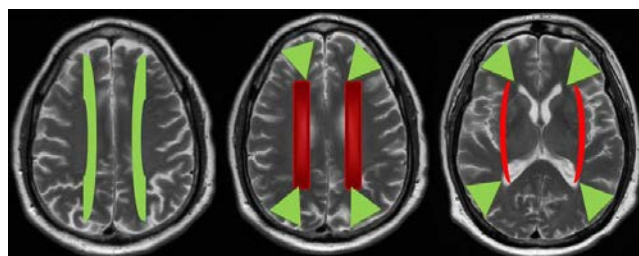
variations of arterial supply and the development of leptomeningeal anastomosis. [13,22] Patients with cortical border-zone infarcts have a more benign clinical course and a better prognosis than those with internal border-zone infarcts. [13,17,22] This is because of the vicinity of the cortical border-zone infarcts to the cortical surface where the chances of leptomeningeal anastomosis formation are high. However, when cortical border-zone infarcts occur in association with internal border-zone infarcts, there is a higher probability of hemodynamic impairment, and the prognosis is usually not favored. [17,28]

### (b) Internal border-zone infarcts:

Internal border-zone infarcts are located between the superficial systems of the MCA&ACA or between the deep and the superficial arterial systems of the MCA namely, 1) lateral and medial lenticulostriate-MCA and ACA, 2) perforators arising from PCA-p1 segment, 3) recurrent artery of Heubner-ACA, 4) anterior choroidal-ICA, and 5) posterior choroidal-PCA. [13,17,21,22]

In contrast to cortical border-zone infarcts, internal border-zone infarcts are more vulnerable to hemodynamic compromise in the presence of vascular stenosis/occlusion. [12,17,21,22] This is explained by the anatomic characteristics of the cerebral arterioles within these border-zones. The deep perforating lenticulostriate arteries being end arteries are devoid of significant anastomoses from superficial cortical medullary arteries. Moreover, the medullary penetrating vessels of the ICA are the most distal branches with small diameters and less density resulting in the lowest perfusion pressure. Therefore, the sub-cortical structures like corona radiata are more susceptible to ischemic insults and repeated episodes of hypotension in the presence of ICAD facilitating internal border-zone infarcts. [13,17,22]

Based on the radiological appearance on MRI, they are classified as either partial or confluent. [15] Partial infarcts are usually caused by brief episodes of hypotension and appear as large, cigar-shaped, arranged in a pattern resembling the beads, parallel and adjacent to the lateral ventricle. Whereas the confluent infarcts are believed to be caused by prolonged/long-lasting impairments of cerebral perfusion resulting in a rosary-like pattern. Internal border-zone infarcts are associated with a less favourable prognosis compared to cortical border-zone infarcts. [13,17,22]



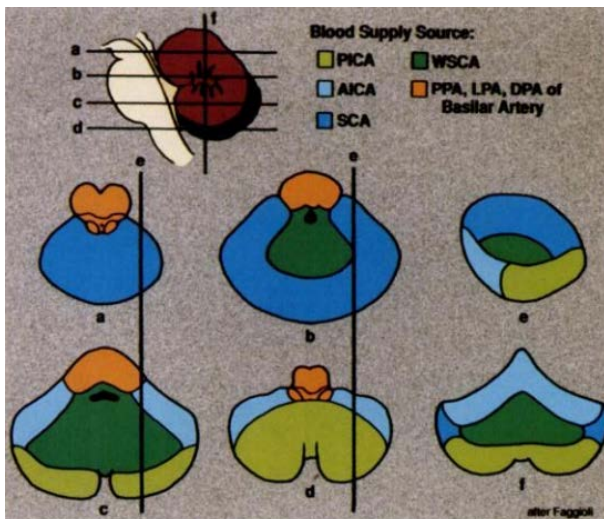
**Figure 4.** Axial T2-weighted magnetic resonance (MR) images of normal cerebrum showing probable locations of cortical (green) and internal (red) border-zone infarcts

### Cerebellar border-zone infarcts:

Border-zone infarcts of the cerebellum are usually <2 cm in size and are described as ‘very small cerebellar infarcts. [29,30] They remain under recognized and under published, with scattered case reports and case series with

varied etiology like large artery atherosclerosis, microembolism, hypercoagulable states, or hemodynamic impairment. [29,30,31] However, hypoperfusion states in the presence of vascular stenosis superimposed by hemodynamic impairment are the most commonly reported etiology. [29,30,31,32]

The border-zone infarcts between Posterior Inferior Cerebellar Artery (PICA) & Superior Cerebellar Artery (SCA) are the most commonly reported in the literature. (Figure 5) A study conducted by Zheng et al, [29] reported 76% of the border-zone infarcts of the cerebellum occurring in the border-zone of PICA&SCA. The proposed mechanism is vertebro-basilar atherosclerosis superimposed by hemodynamic compromise. The second most common border-zone infarct between penetrating branches of Anterior Inferior Cerebellar Artery (AICA), PICA and SCA is usually described to be associated with occlusion of small branch arteries due to emboli either from the ruptured plaque or cardiac source. Cerebellar border-zone infarcts have a favorable prognosis. [30,33]



**Figure 5.** Cross-sectional schematic shows the vascular territories in the cerebellum and brain stem in the axial (a-d), sagittal paramedian (e), and coronal (f) sections [34]. DPA = dorsal penetrating artery, LPA = lateral penetrating artery, PPA = paramedian penetrating artery, WSCA = watershed area in deep white matter (mostly supplied by the SCA)

## 5. Conclusions

Despite the abundant vascular supply, corpus callosum infarction in the border-zone can occur with pre-existing arterial steno-occlusive disease, particularly the event of systemic hypotension and subsequent cerebral hypoperfusion in the peri-procedural periods. In patients with corpus callosum lesions in the peri-procedural period, border-zone infarct should be considered. Corpus callosum infarction (CCI) exhibits less commonly recognized neurological symptoms, evolving slowly over days to weeks, often with complex radiological features, raising concerns for neoplastic causes. Recognition of infarction patterns and comprehensive radiographic studies may minimize the need for invasive procedures like brain biopsy. Improved knowledge of anatomical and pathophysiological mechanisms of border-zone infarction can identify high-risk patients and help strategies for prevention.

## Acknowledgements

None

## Statement of Competing Interests

The authors have no competing interest.

## List of abbreviations

CC: corpus callosum, CCI: corpus callosum infarction, ACA: anterior cerebral artery, MCA: middle cerebral artery, PCA: posterior cerebral artery, CABG: coronary artery bypass grafting, ICAD: intracranial atherosclerotic disease, HTN: hypertension, DM: diabetes mellitus, HPLD: hyperlipidemia, CAD: coronary artery disease, PCI: percutaneous coronary interventions, ESRD: end stage renal disease, NHSS: National Institute of Health Stroke scale score, ICA: internal carotid artery, LAA: large artery atherosclerosis, PICA: posterior inferior cerebellar artery, SCA: superior cerebellar artery, AICA: anterior inferior cerebellar artery.

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