

Splenium infarction presenting as incongruous homonymous hemianopsia: A case report and narrative review

Shivank¹, Manish Bhartiya²

From ¹Clinical Tutor, Department of Internal Medicine, Armed Forces Medical College, ²Neurologist, Department of Neurology, Command Hospital, Pune, Maharashtra, India

ABSTRACT

Splenium, the posteriormost part of the corpus callosum, has the thickest collection of commissural fibers with most fibers extending into the occipital lobes; however, the exact functions are yet to be known. As such, isolated infarctions of the corpus callosum are rare due to the dual blood supply from the anterior and posterior cerebral arteries. We describe the case of a 52-year-old female who presented with a 1-week history of right-sided incongruous homonymous hemianopsia, with no other evidence of focal deficit. Neuroimaging confirmed acute infarction with intense restriction of diffusion in the left splenium involving the periventricular part. There are a few case reports available of isolated splenium infarction. There is only one case described of a splenium infarct presenting as homonymous hemianopsia, to our knowledge. Our case might shed some light on the clinical presentation in splenium infarcts, with an understanding of the visual processing pathways involving the commissural fibers.

Key words: Corpus callosum, Homonymous hemianopsia, Infarction, Splenium

The corpus callosum is the largest white matter tract in the brain, connecting the two cerebral hemispheres with approximately 180 million fibers passing across [1]. Moving from the anterior to the posterior part of the organ, it is aligned as four anatomical subdivisions: the rostrum, genu, body, and splenium. Corpus callosum infarctions are rarely known due to their abundant blood supply from the pericallosal anastomotic plexus formed by the anterior and posterior cerebral arteries [2]. Splenium is the thickest of all parts, with an abundance of commissural fibers, and is the most commonly affected in corpus callosum infarcts [2]. The described clinical manifestations of splenium infarcts vary from headache to confusion, dysarthria, ataxia, hemiparesis, or increased muscle tone [3]. To the best of our knowledge, there is a single described case of isolated splenium infarction presenting as homonymous hemianopsia [4]. We describe a similar case in a middle-aged female.

CASE REPORT

A 52-year-old female presented with complaints of a dull-aching holocranial headache of 10 days duration, which was later followed by a 1-week history of diminution of

vision of the right-sided temporal field of vision. The patient also gave a history of metamorphopsia (visual lines appearing distorted). There was no other focal deficit. She was a known case of Hypertension and Type 2 Diabetes Mellitus for 5 years, on regular medication.

Clinical examination was suggestive of incongruous right-sided homonymous hemianopia with no macular sparing, which was confirmed on visual perimetry (Fig. 1). The rest of the ophthalmological examination revealed no abnormality.

Her hematological parameters at the time of admission were within normal limits. Blood sugar fasting/post-prandial was 150/165 gm/dL. HbA1c was 7.2%.

Non-contrast computed tomography head showed hypodensity in the splenium of the corpus callosum (left half), likely to be infarction versus demyelination. However, a Magnetic resonance imaging (MRI) brain showed diffusion restriction in the left half of the splenium, suggestive of an acute infarct (Fig. 2). CT angiography showed a calcified plaque in the cervical segment of the left Internal Carotid Artery, causing 50% stenosis. The Holter Electrocardiogram did not show any arrhythmia. 2D Echocardiography was also within normal limits. Evaluation for vasculitis was also normal. Following extensive workup, a diagnosis of atherothrombotic infarction of the Splenium of the Corpus Callosum was confirmed.

Access this article online

Received - 01 November 2025
Initial Review - 22 November 2025
Accepted - 23 December 2025

Quick Response code



DOI: 10.32677/ijcr.v12i1.7933

Correspondence to: Shivank, Department of Internal Medicine, Armed Forces Medical College, Pune, Maharashtra, India. E-mail: shivankafmc94@gmail.com

© 2026 Creative Commons Attribution-NonCommercial 4.0 International License (CC BY-NC-ND 4.0).

With known risk factors of Diabetes Mellitus Type 2 and Hypertension, she was managed with a single antiplatelet drug (Tablet Aspirin 150 mg once a day), statin (Tablet Atorvastatin 20 mg at night), Calcium Channel Blocker (Tablet Amlodipine 10 mg once a day), and biguanide (Tablet Metformin 500 mg twice a day). Our patient showed resolution of headache with improvement in her field of vision over the next 7 days and has been advised for regular follow-up in the Neurology Outpatient Department.

DISCUSSION

The optic pathway in our body is a complicated set of neurons with a specific visual defect described for lesions at any given point in the pathway. It begins from the retinal fibers, followed by the optic nerve, the optic chiasma, the optic tract, the lateral geniculate body, the optic radiation, and the visual cortex in the occipital lobe [5]. Homonymous Hemianopia is known to occur due to a lesion in the contralateral optic tract and beyond. The closer the lesion is to the visual cortex, the more congruous the visual defect [6]. Stroke is the most common underlying cause, with up to 80% of cases involving the occipital lobe [7]. Our patient had incongruous homonymous hemianopia with metamorphopsia and

an isolated splenium infarct. Now, the corpus callosum is generally preserved from any ischemic damage due to the collateral blood supply (Fig. 3). Splenium is the most commonly affected part during an infarction of the Corpus Callosum [8]. A detailed study classifies the etiology of splenial lesions into further heads: Congenital, Infarct, Tumors, Degenerative, and Demyelinating lesions (Table 1). It identifies congenital lesions of the corpus callosum as one of the most common causes, and focal infarctions are rare [3].

It has been hypothesized that it is the disrupted flow of visual information across both cerebral hemispheres that is causing metamorphopsia [9]. Visual field defect is also generally considered to be the result of a similar pathology of disruption of neuronal flow [10]. This would, however, mean that the visual field of any side would be defined by visual information processing in both cortical hemispheres. This fails to explain the laterality of lesions observed in the case reports [4].

We would further hypothesize here the involvement of optic radiation in splenium infarcts, which is the most common site for incongruous visual defects [6]. A coronal section at the level of optic radiation shows

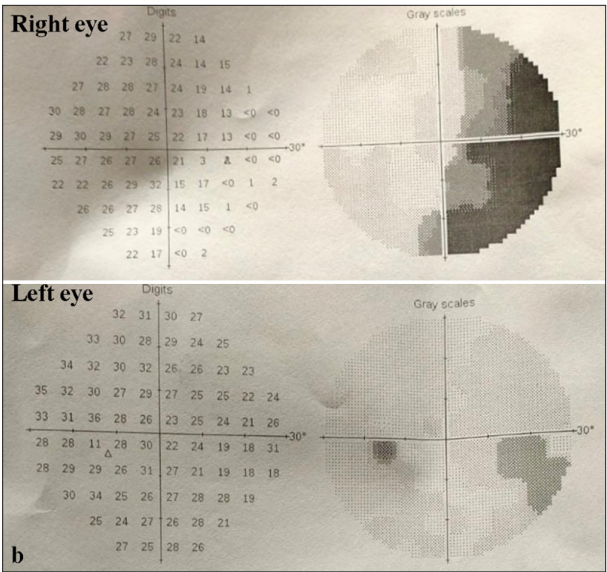


Figure 1: Visual perimetry showing incongruous right-sided homonymous hemianopia

Table 1: Etiology of lesions of splenium of corpus callosum	
Congenital	Agensis (complete or partial) lipoma
Traumatic	Diffuse axonal injury
Ischemic	Infarction
	Hypoxic ischemic encephalopathy
	CO-intoxication
	Hypoglycemic encephalopathy
Tumors	Lymphoma
	Glioma
	Germinoma
Degenerative and Demyelinating	Multiple sclerosis
	Wallerian degeneration
	Acute demyelinating encephalomyelitis
	Posterior reversible encephalopathy syndrome
Toxic Encephalopathy	Marchiafava-Bignami disease
	Metronidazole-induced encephalopathy
Miscellaneous	Transient lesion in splenium
	White matter injury in neonatal viral infection

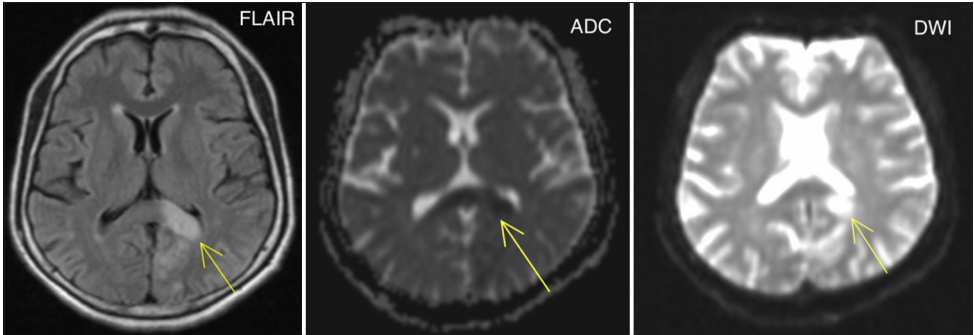


Figure 2: Magnetic resonance imaging sequences of the patient showing Splenium Infarction

Table 2: Reported cases of isolated Splenic infarctions

Author	Year	Age/Sex	Comorbidities	Clinical presentation
Katsuki <i>et al.</i> [4]	2019	85/M	Glaucoma	Right homonymous hemianopia
Zhang <i>et al.</i> [7]	2019	53/M	Not described	Not known
		57/M	Not described	Dizziness
Saito <i>et al.</i> [9]	2014	61/F	Right putamen hemorrhage	Left metamorphopsia
Hashiguchi <i>et al.</i> [11]	2009	63/M	Hypertension, diabetes mellitus	Dysarthria, memory impairment
Katsura <i>et al.</i> [12]	2010	67/F	Not described	Right metamorphopsia
Nagaishi <i>et al.</i> [13]	2015	78/F	Hypertension, post-hysterectomy status	Left metamorphopsia
Chang and Huang[14]	2015	74/F	Nil	Paraesthesia in the right hand
Li <i>et al.</i> [15]	2015	78/M	Not described	Hemiparesis, dysarthria
		74/M	Not described	Hemiparesis, ataxia
		61/M	Not described	Hemiparesis, vertigo
Lai and Kadirji[16]	2017	21/F	Nil	Photophobia
Zhu <i>et al.</i> [17]	2018	53/M	Nil	Left-hand weakness
Barghouthi and El Hussein[18]	2018	67/F	Coronary artery disease, diabetes mellitus, hypertension, dyslipidemia	Prosopometamorphopsia
Sadashivam and Natarajan[19]	2020	24/M	Head injury	Ataxia
Ogawa <i>et al.</i> [20]	2020	?/F	Nil	Prosopometamorphopsia
Zhao <i>et al.</i> [21]	2021	53/M	Nil	Non-convulsive status epilepticus
Samuel Kim <i>et al.</i> [22]	2023	56/M	Nil	Palinopsia
Our case	2023	52/F	Hypertension, diabetes mellitus	Right homonymous hemianopia

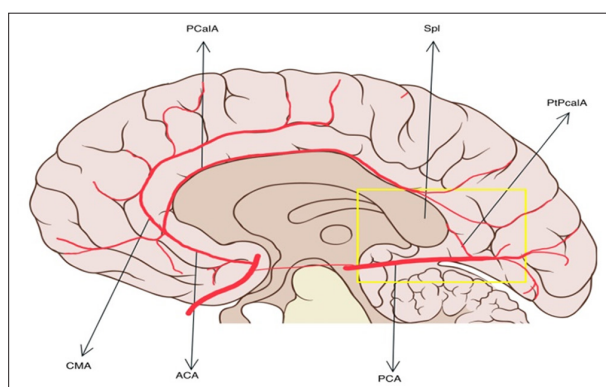


Figure 3: Blood supply of corpus callosum. Spl: Splenium, PCalA: Pericallosal artery, CMA: Callosomarginal artery, ACA: Anterior cerebral artery, PCA: Posterior cerebral artery; PtPCalA: Posterior pericallosal artery.

optic radiation as the lateral wall of the posterior horn of the lateral ventricle with a tapetum of splenium running in the middle of the trigone of the lateral ventricles. This is suggestive of the anatomical proximity of the two structures [5].

Further, isolated Splenic infarcts are rare. We could find only 14 cases that have been described. One case describes Homonymous Hemianopia, and three others describe metamorphopsia. Atherothrombotic infarction has been the underlying etiology in most of these cases (Table 2) [4,7,9,11-22].

CONCLUSION

The exact functions of the Splenic still need to be studied further with a better understanding of the underlying causations. The variation in clinical presentations, with visual complaints in a few cases, highlights the importance of association fibers in optic

pathways. A confirmation of the same would require functional imaging studies.

REFERENCES

- Devinsky O, Laff R. Callosal lesions and behavior: History and modern concepts. *Epilepsy Behav* 2003;4:607-17.
- Kontzialis M, Soares BP, Huisman TA. Lesions in the splenium of the corpus callosum on MRI in children: A review. *J Neuroimaging* 2017;27:549-61.
- Park SE, Choi DS, Shin HS, Baek HJ, Choi HC, Kim JE, *et al.* Splenic lesions of the corpus callosum: Disease spectrum and MRI findings. *Korean J Radiol* 2017;18:710-21.
- Katsuki M, Kato H, Niizuma H, Nakagawa Y, Tsunoda M. Homonymous hemianopsia due to the infarction in the splenium of the corpus callosum. *Cureus* 2021;13:e19574.
- Chen CC, Huang F, Shao HX, Jin JH, Li ZP, Zhang CS. Sectional anatomy of the optic pathways on the coronal plane. *J Chin Med Assoc* 2009;72:515-20.
- Goodwin D. Homonymous hemianopia: Challenges and solutions. *Clin Ophthalmol* 2014;8:1919-27.
- Zhang X, Kedar S, Lynn MJ, Newman NJ, Biousse V. Homonymous hemianopias: Clinical-anatomic correlations in 904 cases. *Neurology* 2006;66:906-10.
- Yamaguchi Y, Iwasaki Y, Wada M, Makita N, Nagasawa H, Yamakawa T, *et al.* Transient lesion of the splenium of the corpus callosum after acute ischemic stroke. *Intern Med* 2019;58:1011-5.
- Saito Y, Matsunaga A, Yamamura O, Ikawa M, Hamano T, Yoneda M. A case of left hemi-facial metamorphopsia induced by infarction of the right side of the splenium of the corpus callosum. *Rinsho Shinkeigaku* 2014;54:637-42.
- Jang SH, Lee HD. Recovery of visual field defect via corpus callosum in a patient with cerebral infarct. *Neuroophthalmology* 2015;39:88-91.
- Hashiguchi A, Yano S, Nitta K, Ide W, Hashimoto I, Kamada H, *et al.* Hemisplenic- accompanied by internal border-zone infarction: Clinical relevance of the splenium of the corpus callosum as a border-zone area between anterior and posterior cerebral arteries. *J Neurol Neurosurg Psychiatry* 2010;81:704-6.
- Katsura N, Konno K, Yamagata M. A case of corpus callosum infarction with unilateral facial metamorphopsia. *Nihon Naika Gakkai Zasshi* 2010;99:1318-20.
- Nagaishi A, Narita T, Gondo Y, Nakane S, Fukudome T,

- Matsuo H. Left-sided metamorphopsia of the face and simple objects caused by an infarction at the right side of the splenium of the corpus callosum. *Rinsho Shinkeigaku* 2015;55:465-71.
14. Chang TP, Huang CF. Unilateral paresthesia after isolated infarct of the splenium: Case report. *Acta Neurol Taiwan* 2010;19:116-9.
 15. Li S, Sun X, Bai YM, Qin HM, Wu XM, Zhang X, *et al.* Infarction of the corpus callosum: A retrospective clinical investigation. *PLoS One* 2015;10:e0120409.
 16. Lai W, Katirji B. Splenium infarct due to cerebral venous thrombosis. *Arch Neurol* 2007;64:1540.
 17. Zhu X, Zhang X, Lu S, Liu Z. Rare etiology for splenium of corpus callosum infarction: Anterior cerebral artery dissecting aneurysm. *Neurology* 2018;91:481-2.
 18. Barghouthi T, El Hussein N. Prosopometamorphopsia secondary to a left splenium of the corpus callosum infarct. *BMJ Case Rep* 2018;2018:bcr-2018-224735.
 19. Sadashivam S, Natarajan A. Isolated infarction of the splenium: A rare presentation of mild head injury. *Asian J Neurosurg* 2020;15:214-7.
 20. Ogawa K, Akimoto T, Takahashi K, Hara M, Morita A, Kamei S, *et al.* A case of prosopometamorphopsia caused by infarction of the splenium of the corpus callosum and major forceps. *Neurocase* 2020;26:264-9.
 21. Zhao Q, Sun L, Hu B, Lin W. Nonconvulsive status epilepticus manifesting as rapidly progressive dementia and infarction in the splenium of the corpus callosum: A case report. *Medicine (Baltimore)* 2021;100:e25263.
 22. Kim US. Palinopsia after splenium infarction. *J Neuroophthalmol* 2024;44:e263-4.

Funding: Nil; Conflicts of interest: Nil.

How to cite this article: Shivank, Bhartiya M. Splenium infarction presenting as incongruous homonymous hemianopsia: A case report and narrative review. *Indian J Case Reports*. 2026; 12(1):42-45.