Title: Painful abdomen, painless blindness: an unusual presentation of pancreatitis

Authors: Udupi Raksha Mallya, Raghunandan Nadig, Vishal Sharma, GRK Sarma, Thomas Mathew, Sagar Badachi, Sonia Shivde, Akshata Huddar, Somdatta Ray

Departmemnt of Neurology, St Johns's Medical College Hospital, Bengaluru, India

INTRODUCTION

Neurological complications in systemic illnesses, particularly renal failure and pancreatitis, are often under-recognized. We present the case of a young male with epilepsy and recurrent pancreatitis who presented with acute, painless, bilateral vision loss.

HISTORY AND EXAMINATION

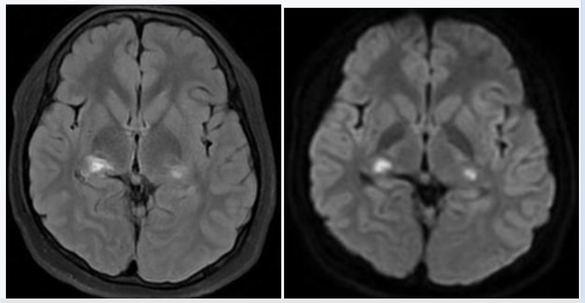
A 19-year-old male with a history of scar epilepsy since childhood secondary to birth asphyxia and hypogonadotropic hypogonadism presented with recurrent episodes of acute pancreatitis since 2016. During the current episode, he was found to have a recurrence of pancreatitis along with oliguric renal failure and azotemia. He was initially admitted to an outside hospital, where he developed **sudden-onset, painless loss of vision in both eyes**. There was no history of headache or seizures. He was subsequently transferred to our center for further management.

On examination, the patient was conscious and oriented to time, place, and person. Blood pressure was 130/90 mmHg, and pulse was 90/min. Vision: there was no perception of light**. Pupils were 3–4 mm and sluggishly reactive to light.** Bilateral relative afferent pupillary defect (RAPD) was present. Fundus examination was normal. The range of eye movements was full. There were no other neurological deficits

Initial blood investigations revealed anemia with iron and vitamin B12 deficiency. Urea and creatinine levels were elevated. Serum amylase and lipase were also elevated

TEST	VALUE
Haemoglobin	9g/dl
Total count	14400 cells/mm^3
Platelet count	82000/mm^3
Peripheral smear	normocytic normochromic with a few normocytic hypochromic cells and elliptocytes
Urea	104mg/dl
Creatinine	7.3mg/dl
Amylase	939U/L
Lipase	917U/L
Serum B12	19.2pmol/dl
Serum	16mcg/dl
Serum TIBC	242mcg/dl
Transferrin saturation	6.61%
Serum ferritin	406.7ng/dl

MRI of the brain at admission showed restricted diffusion in the **bilateral medial temporal lobes and lateral geniculate bodies (LGB),** raising differentials of MELAS, hypoxic-ischemic encephalopathy (HIE), and metabolic encephalopathy (Fig 1,2). CT abdomen revealed features suggestive of **acute edematous pancreatitis**, with a Modified CT Severity Index (CTSI) score of 4/10. The main pancreatic duct (MPD) was seen opening into the minor papilla, raising concern for pancreatic divisum



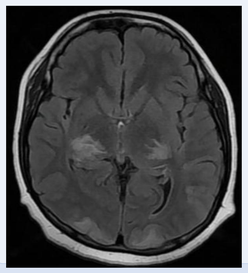
(FIG 1,2) MRI OF BRAIN SHOWING DWI RESTRICTION AND FLAIR HYPERINTENSITY IN BILATERAL LGB

COURSE IN HOSPITAL

He was initiated on hemodialysis (HD) in incremental sessions. A renal biopsy revealed acute tubular necrosis. His urine output gradually improved during the hospital stay, and he was subsequently discharged.

However, he was readmitted the following day with a **severe bifrontal, throbbing headache** that was not relieved by medications. On examination, his blood pressure was **155/100 mmHg**. Fundus examination remained normal. A follow-up MRI revealed T2/FLAIR hyperintensities in the bilateral parieto-occipital and temporal lobes, suggestive of **posterior reversible encephalopathy syndrome** (PRES) (Fig 3).

He was treated with antihypertensives, and his blood pressure was brought under control. Whole exome sequencing and mitochondrial genome sequencing revealed no pathogenic variants.
On follow-up, vision improved to PL. MRI brain showed resolution of the imaging abnormalities.



(FIG 3) MRI OF BRAIN SHOWING FLAIR HYPERINTENSITIES IN BILATERAL LGB AND PARIETOOCCIPITAL REGIONS SUGESSTIVE OF PRES

DIFFERENTIAL DIAGNOSIS

Causes of sudden painless loss of vision in both eyes

- Bilateral occipital lobe infarcts
- Bilateral retinal artery occlusion
- Toxic- methanol poisoning
- Bilateral ischemic optic neuropathy
- PRES
- Bilateral lateral geniculate body infarcts

Causes of bilateral lateral geniculate body infarcts

- MELAS (Mitochondrial Encephalopathy, Lactic Acidosis, and Stroke-like episodes)
- Bilateral Ischemic Infarction of the LGB
- Hypoxic-Ischemic Encephalopathy (HIE)
- Toxic/Metabolic Encephalopathy
- Inflammatory/Demyelinating Disease- ADEM
- Infectious Encephalitis- West Nile virus, herpes
- Posterior Reversible Encephalopathy Syndrome (PRES)
- Traumatic Brain Injury / Diffuse Axonal Injury
  Injury to thalamic relay nuclei including LGB

Causes of bilateral vision loss in pancreatitis

- Purtscher's-like retinopathy
- Bilateral LGB infarcts

DISCUSSION

This case emphasizes the rare occurrence of bilateral LGB infarction in a young patient with pancreatitis. On literature review, the exact reason for LGB involvement is not known. Several hypothesis, like microangiopathy, hypovolemic shock, osmotic pathology have been mentioned.

The LGBs have a very **high metabolic rate** and a dual blood supply. The anterior choroidal artery, a branch of the internal carotid artery, supplies the anterior part; while rest of the LGB is supplied by the posterior choroidal artery, a branch of the posterior cerebral artery. Vascular occlusion due to microangiopathic disorders or arteritis have been proposed as a mechanism for the vascular compromise in the LGB. The hemorrhagic infarcts seen in these lesions are thought to be due to the disruption of the blood–brain barrier in the distribution of anterior and posterior choroidal arteries.

DISCUSSION CONTINUED

MRI findings confirmed bilateral infarcts of the LGB. A subsequent scan revealed imaging features consistent with PRES.

Other potential causes, including mitochondrial disorders, were systematically ruled out. The episode was most likely secondary to acute pancreatitis and associated metabolic derangements in the setting of renal failure and hypertension.

A literature review of similar cases was performed

Author / Year	Age	Etiology
Meena et al., 2023	10Y M	Acute pancreatitis with coagulopathy → hemorrhagic infarction of bilateral LGBs
Srichawla et al., 2024	17Y F	Acute pancreatitis + Purtscher-like retinopathy → hemorrhagic LGB infarcts
Viloria et al., 2015	40Y M	Acute pancreatitis → osmotic demyelination (LGB myelinolysis)
Lefèbvre et al., 2004	31Y F	Anaphylactic shock (amoxicillin) → hypoperfusion injury to LGBs
Puppala et al., 2019	22Y F	H1N1 influenza A → hemorrhagic infarction of bilateral LGBs
Silva et al., 2014	10Y F	Severe febrile gastroenteritis → dehydration/hypoperfusion leading to bilateral LGB infarction
Murugesan et al., 2022	22Y F	Acute pancreatitis → bilateral LGB necrosis (possible ischemic-hypoxic)
Callaway et al., 2006	29Y F	Classic migraine → isolated bilateral LGB infarction
Stem et al., 2014	Adult female	Posterior reversible encephalopathy syndrome (PRES) in preeclampsia → bilateral LGB lesions
Case report, 2021 (Rani et al.)	40Y F	Post-ERCP pancreatitis → bilateral LGB infarction (ischemic)
D'Ambrosio et al., 2010	14Y M	Inflammatory bowel disease with recurrent pancreatitis → bilateral LGB infarct
Thakkar et al., 2015	27Y M	Severe dehydration (hypovolemia) from gastroenteritis → bilateral LGB ischemia
Kale et al., 2016	19Y M	Acute viral illness (fever, dehydration) → LGB ischemia with hemorrhagic component

- **Dehydration/hypoperfusion** was the common mechanism, in these studies
- Both **ischemic** and **hemorrhagic** changes are reported.
- **Other non-vascular** pathologies osmotic demyelination, PRES, migraine were also reported.

CONCLUSION

Bilateral LGB infarction is a rare but important differential diagnosis in patients presenting with acute, painless bilateral vision loss, especially in the context of systemic illnesses such as pancreatitis. Further research is needed to elucidate the precise mechanisms involved and to understand the spectrum of neurological complications in pancreatitis.

REFERENCES

Mathew T, Delon D, Nadig R, Sarma GR. Bilateral lateral geniculate body hemorrhagic infarction: A rare cause of acute bilateral painless vision loss in female patients. Neurology India. 2016 Jan 1;64(1):160-2.

Mudumbai RC, Bhandari A. Bilateral isolated lateral geniculate body lesions in a patient with pancreatitis and microangiopathy. Journal of Neuro-Ophthalmology. 2007 Sep 1;27(3):169-75.