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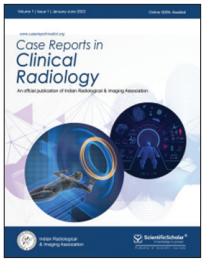

Case Report

Hemorrhagic infarction of bilateral lateral geniculate bodies – unusual complication of pancreatitis

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ABSTRACT

We report a case of 10-year-male child diagnosed as acute pancreatitis with deranged liver and renal functions, who presented with sudden onset bilateral painless vision loss 2 days after initial diagnosis. Neuro-ophthalmological examination including deep tendon reflex, plantar reflex, visual acuity, pupillary reflexes, and fundus was performed. Magnetic resonance imaging brain demonstrated features of hemorrhagic infarction involving bilateral lateral geniculate bodies (LGBs) and posterior end of optic tracts. Two major causes of bilateral LGBs lesions include vascular ischemia and osmotic demyelination. Possible mechanism of involvement of bilateral LGBs in present case is coagulopathy in the settings of acute pancreatitis attributed to pro-thrombotic state leading to microvascular occlusions.

Keywords: Lateral geniculate bodies infarction, Pancreatitis, Painless vision loss, Extrapontine demyelinosis, Disseminated intravascular coagulation

INTRODUCTION

Lateral geniculate body (LGB) is visual sensory projection nuclei located in the posteroventral region of the thalamus. It is a relay center for the visual pathway where optic tract terminates and optic radiation originates (Meyer's loop, central bundle, and Baum's loop) that project through the internal capsule to the primary visual cortex.[1] LGB receives dual blood supply from lateral posterior choroidal arteries branch of posterior cerebral artery and anterior choroidal artery branches of the internal carotid artery.[2] LGB may be involved with unilateral or bilateral lesions. Visual acuity is preserved with unilateral lesions while severely compromised with bilateral lesions. LGB may be involved in arteriovenous malformations, spinocerebellar ataxia Type 2, extra-pontine myelinosis, and microangiopathies secondary to systemic illness. Causes of unilateral lesions include neoplasm, aneurysm, vascular malformation, head trauma, neurosurgical procedures stroke, and inflammation. Bilateral hemorrhagic infarction of LGBs is rare and many hypothesis has been provided including osmotic demyelination, ischemic arteritis, and anaphylactic shock. Interestingly, there is an increased incidence of this condition in patients with acute pancreatitis. The author mentions coagulopathy as a case in this case, which may be a valid hypothesis. It is difficult to prove and disprove. Isolated LGB lesion is a rare cause of visual loss. As per literature search, very few cases of bilateral LGBs lesions associated with visual symptoms have been reported. Syphilitic anterior choroidal arteritis was the first reported pathology of LGB involvement. Other etiologies were microangiopathy secondary to pancreatitis, extrapontine myelinosis, hemorrhagic infarction in preeclampsia, and anaphylactic shock.[3] Here,

we report the clinical and radiological feature of a 10-year-old male patient diagnosed as acute edematous pancreatitis which lead to sudden onset bilateral painless vision loss.

CASE REPORT

A 10-year-old male child was admitted with complaints of severe acute epigastric pain and multiple episodes of bilious vomiting. He was diagnosed with acute pancreatitis and oliguric acute kidney injury. On admission, patient was conscious and oriented. Vitals of patient recorded were BP – 126/80, Heart rate – 118/min, Respiratory rate – 22/min, SpO_2 – 98% on room air, and afebrile. Blood investigation showed normocytic normochromic anemia with Hb – 9.6 g/dl, increased white blood cells (WBCs) – 15600 cells/mm^3 with 71% of neutrophils and decreased platelet counts – 75000cells/mm^3. He had deranged liver and renal function tests – serum amylase – 1050 U/L, serum lipase – 602 U/L, total bilirubin – 1.2 mg/dl, alanine transaminase – 14 U/L, aspartate transaminase – 70 U/L, alkaline phosphatase – 248 U/L, serum creatinine – 1.1 mg/dL, and blood urea – 94 mg/dL. Prothrombin time was 20 s (10–13 s) and activated partial thromboplastin clotting time was 55 s (24–36 s). The D-dimer was 3900 ng/mL (<500 mg/mL).

Ultrasound abdomen revealed diffusely bulky and hypoechoic pancreas [Figure 1]. No evidence of any pancreatic or peripancreatic necrosis/collection was noted. Multiple small calculi of approximately 4–5 mm average size were seen in gall bladder lumen. No features suggestive of cholecystitis were present. On imaging, diagnosis of acute edematous pancreatitis with cholelithiasis was made.

Two days after initial evaluation, the child complained of acute painless bilateral vision loss associated with headache. His Glasgow coma scale score was E4V5M6 and rest of central nervous system examination was normal. Ophthalmological examination documented perception of light and sluggish reactive pupil in bilateral eye fields with relative afferent pupillary defect in left eye. The fundus examination was normal in both eyes with normal and free extraocular movements. Urgent magnetic resonance imaging (MRI) brain was done with the possible differential clinical diagnoses of retrobulbar optic neuritis and drug-induced optic neuritis. MRI depicted bilateral symmetrical T2/FLAIR hyperintense lesions confined in the posterolateral thalamus in the region of LGBs and extending laterally along the posterior end of optic tracts on both sides. Areas of T1WI hyperintensity were seen within lesions which showed blooming on gradient echo sequence, suggestive of hemorrhage [Figure 2] and bilateral normal optic nerves [Figure 3].

Patient was managed with intravenous fluids, analgesics, and antiemetics. Intravenous antibiotic course was given to the patient including vancomycin and ceftriaxone. Dexamethasone was started, after ophthalmology opinion. WBC count, platelet count, liver, and renal functions were normalized within 2 weeks; however, no improvement in vision could be noted uptill 3 months.

DISCUSSION

Acute bilateral vision loss is infrequently encountered in children and can be due toretinal detachment, ischemic optic neuropathies, posterior cerebrovascular accidents involving optic radiations, primary visual cortex, and associated visual

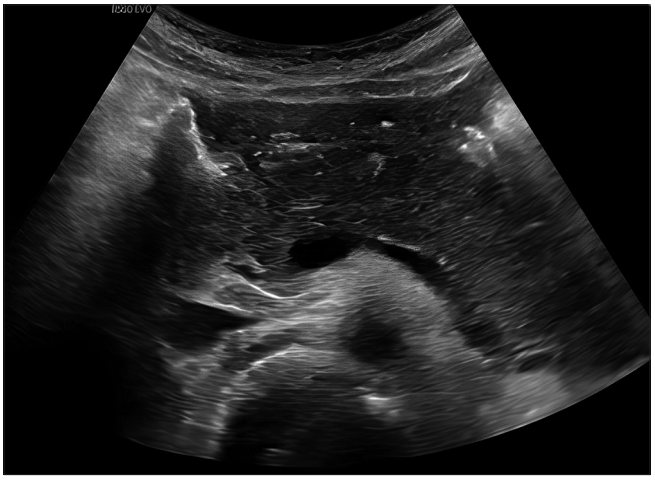

Figure 1: Ultrasound image shows diffusely bulky hypoechoic pancreas. No evidence of any pancreatic or peripancreatic collection seen.

Table 1: Causes of LGB infarction from previous case reports.

Name of the study	Chracteristics of study population	Possible etiologies
Mackenzie I *et al*.[5] 1933	(--)	Syphilitic involvement of LGB along with thalamus.
Donahue *et al*.[7] 1995	37/F	Central pontine myelinosis
Greenfield *et al*.[8] 1996	28/F	Lateral geniculitis
Moseman CP *et al*.[9] 2002	21/f	Severe vasoconstriction due to pre-eclampsia.
Lefebvre *et al*.[10] 2004	31/F	Ischaemia with systemic hypotension
Mudumbai RC *et al*.[3] 2007	18/F	Acute pancreatitis with thrombotic microangiopathy
Mulholland C *et al*.[11] 2010	14/M	LGB infarction with Inflammatory bowel disease and recurrent pancreatitis.
Mathew T *et al*.[6] 2016	42/F	Vascular occlusion due to microangiopathic disorders

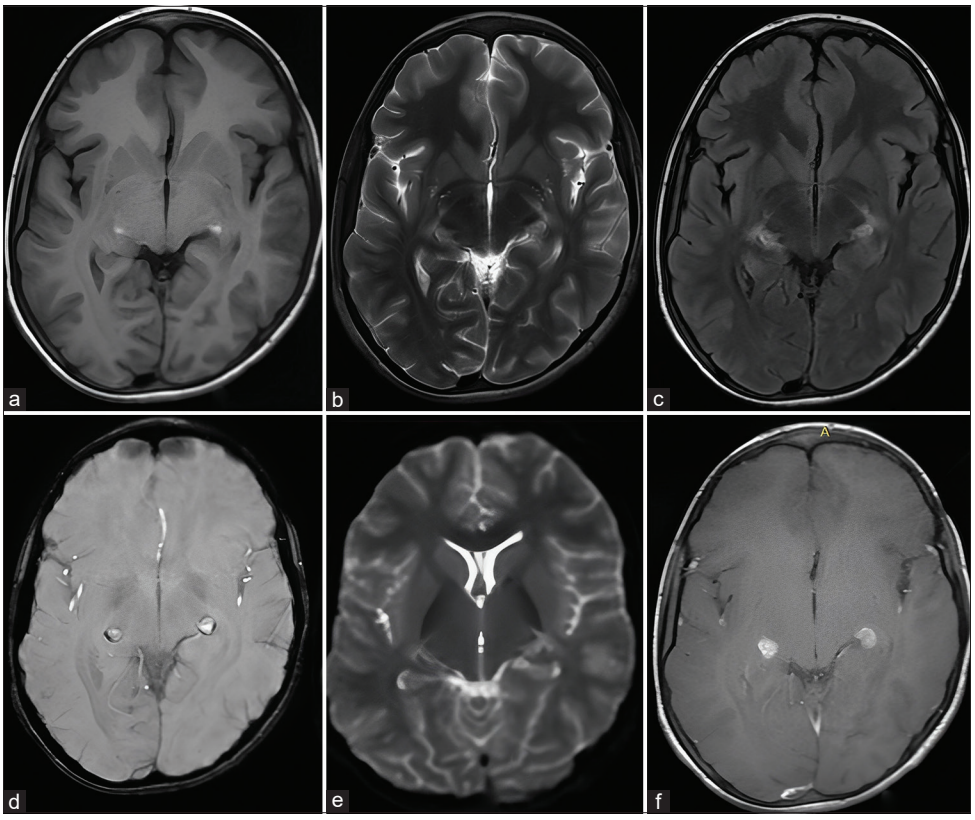

Figure 2: Axial MRI brain images show area of altered signal intensity in bilateral lateral geniculate bodies extending into optic radiations which appears bright on T1(a), hyperintense on T2/FLAIR(B&C), show peripheral blooming on FFE(d), no evidence of any diffusion restriction(e) and show solid contrast-enhancement on post-contrast imaging(f).

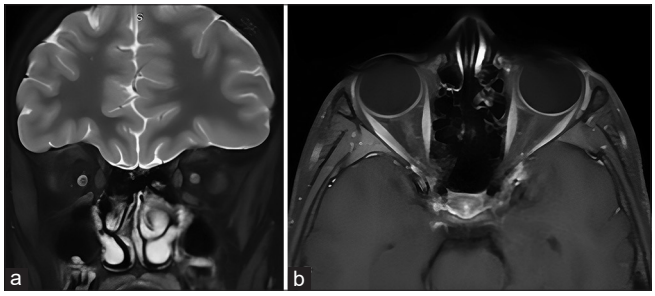

Figure 3: Coronal (a) T2 fat suppressed image and post-contrast axial T1 fat suppressed image (b) show normal bilateral nerves.

cortices.[4] Isolated LGB involvement causing bilateral vision loss is rare with very few literature available on the topic. First case described in the literature dates back to 1933 by Mackenzie *et al*.[5] where LGB involvement was attributed to anterior choroidal syphilitic arteritis. While the most recently described case involves hemorrhagic infarction of bilateral LGBs in a patient of acute infective systemic illness with renal and hepatic dysfunction.[6]

Other reported causes of LGB involvement are extrapontine myelinolysis following rapid correction of hyponatremia or infarct due to severe vasoconstriction in pre-eclampsia and anaphylactic shock.[7-10] [Table 1]. Predilection of isolated LGB involvement in these cases has been partially understood only. LGB has high metabolic rate making it susceptible to hypoxia. In addition, it has grid like arrangement of gray and white matter similar to pons, and tight connection between oligodendrocyte and neurons making it difficult to expand after rapid correction of hyponatremia leading to increased sensitivity of LGB to osmotic demyelination amounting to rapid dehydration of oligodendrocyte and further cell shrinkage.[3] All patients involved in these reported cases were middle aged females. Reason for this skewed affliction is not known.

The mechanism of involvement of bilateral LGBs in our case is more likely deranged coagulation profile that can be attributed to pro-thrombotic state. Acute pancreatitis leads to DIC and has been responsible for microvascular occlusions. There was no documented hyponatremia, so possibility of osmotic demyelination could be excluded. The literature similar to our case of acute onset painless vision loss due to coagulative necrosis of LGB induced by acute pancreatitis with microangiopathic hemolytic anemia was described by Mathew *et al*.[6] and Mudumbai and Bhandari.[3]

It is interesting to note that among previously documented cases in the literature with bilateral LGB infarctions, mostly were middle aged female patients while our case is the male and peditaric patient with bilateral LGB infarction. Similar patient profile was seen in study by Mulholland *et al*.[11] Further studies are needed in this direction to establish or refute the skewed association of this entity with particular gender and age group.

CONCLUSION

Hemorrhagic infarction of bilateral LGBs is rare cause of sudden painless vision loss. Show skewed predilection in females. Extrapontine myelinolysis and thrombus due to pro-thrombotic state are most common etiologies.

Teaching Points

1. LGB is important relay station in visual pathway.
2. Isolated infarction of LGB is rare.
3. Osmotic demyelination and microvascular occlusion are the most common proposed etiologies.

MCQs

1. True about lateral geniculate bodies are except
   a. Relay center in visual pathway
   b. Located in anteromedial thalamus
   c. Dual blood supply from branches from posterior cerebral artery and internal carotid artery
   d. Located in posteroventral thalamus

Answer Key: b

2. Pathologies commonly affecting lateral geniculate bodies are except-
   a. Osmotic demyelination
   b. Ischemic arteritis
   c. Microangiopathies due to pro-thrombotic state
   d. Alzheimer's disease

Answer Key: d

3. True about lateral geniculate bodies infarct are –
   a. Females are more commonly affected.
   b. Males are more commonly affected.
   c. Not seen in pediatric age group.
   d. Present as acute painful vision loss.

Answer Key: a

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

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