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Case report

Binasal Hemianopsia Secondary to Bilateral Lesions of the Lateral Geniculate Bodies in Post-ERCP Pancreatitis: A Case Report and Literature Review

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Abstract

Introduction: Endoscopic retrograde cholangiopancreatography (ERCP) is currently the standard procedure for treating pancreatobiliary conditions. As its use has increased, so has the incidence of associated complications, such as pancreatitis, bleeding, and perforation. Post-ERCP acute pancreatitis is the most common complication. While most cases are mild, some can be severe and, in very rare instances, may result in bilateral lesions of the lateral geniculate bodies, as in this case. **Case Report:** A 28-year-old woman with a history of obesity presented with acute cholangitis due to choledocholithiasis. She underwent ERCP with bile duct stone removal and placement of a plastic stent. Forty-eight hours after the procedure, she developed severe post-ERCP pancreatitis and concurrent incongruent binasal hemianopsia. Brain magnetic resonance imaging revealed bilateral lesions of the lateral geniculate bodies (LGB). Ischemic, infectious, hydroelectrolytic, and autoimmune causes were ruled out. The patient's clinical evolution was favorable. **Discussion:** Although the mechanisms underlying lateral geniculate body lesions are not fully understood, in this case, severe post-ERCP acute pancreatitis and multiorgan involvement secondary to systemic inflammatory response syndrome (SIRS) are proposed as the likely etiology.

Keywords

Acute pancreatitis, endoscopic retrograde cholangiopancreatography, geniculate bodies, visual fields, visual acuity.

INTRODUCTION

Endoscopic retrograde cholangiopancreatography (ERCP) is currently considered the standard procedure for managing pancreatobiliary conditions. Various studies have demonstrated its safety and efficacy, leading to an increase in the number of procedures performed. Consequently, this has resulted in a rise in reported complications[(1)]. Among post-ERCP complications, pancreatitis is the most common. Although most cases are mild to moderate (95%) with complete patient recovery, it remains a notable cause of mortality[(2)], including the occurrence of rare complications, as described in this report.

Acute bilateral lesions of the lateral geniculate bodies represent a rare entity with unclear pathophysiology. These lesions are associated with various conditions, such as inflammatory processes and vasculitis[(3)], osmotic demyelination secondary to electrolyte imbalances[(4,5)], and ische-

mia due to systemic hypoperfusion[6]. This report presents the first case described in Latin America of a patient who developed visual deficits and radiological findings indicative of attenuation in the lateral geniculate bodies within the context of severe acute post-ERCP pancreatitis.

A CASE REPORT

The patient is a 28-year-old female with a body mass index (BMI) of 32 kg/m^2 and no other relevant medical history. She was referred from a lower-complexity facility where she had presented with a three-day history of epigastric abdominal pain. The pain was described as sharp, with an intensity of 7/10 on the pain analog scale, radiating to the right dorsal region. The symptoms were accompanied by nausea and three episodes of vomiting, followed by the onset of jaundice and dark urine; however, she did not report pale stools or fever. Upon admission, vital signs were within normal ranges: heart rate (HR), 62 beats per minute [bpm]; blood pressure (BP), 116/82 mmHg; oxygen saturation (SpO_2), 92%; respiratory rate (RR), 18 breaths per minute; and temperature, 36°C. Physical examination revealed mucocutaneous jaundice and tenderness in the right hypochondrium, with no other abnormal findings.

Laboratory tests performed at the referring institution revealed marked leukocytosis (16,280 cells/µL) with neutrophilia (80%), a negative pregnancy test, and, notably, normal bilirubin and alkaline phosphatase levels. As part of the abdominal pain workup, a complete abdominal ultrasound was performed at the referring site, which identified cholelithiasis without signs of cholecystitis and multiple diffuse focal lesions predominantly affecting the right hepatic lobe. To better characterize these findings, a contrast-enhanced magnetic resonance imaging (MRI) of the abdomen was conducted, revealing hypointense areas with geographic contours, primarily in the right hepatic lobe. At our institution, laboratory tests showed alterations in liver biochemistry consistent with a cholestatic pattern (total bilirubin: 4.59 mg/dL, direct bilirubin: 3.38 mg/dL, alkaline phosphatase: 279 U/L, aspartate aminotransferase [AST]: 976 U/L, alanine aminotransferase [ALT]: 1,553 U/L). Tests for hepatotropic viruses were negative, and coagulation times, renal function, and electrolyte levels were within normal limits. Given the persistence of abdominal pain, the cholestatic biochemical pattern, and focal liver lesions, choledocholithiasis was deemed highly probable despite its absence in prior imaging studies. Consequently, endoscopic ultrasound (EUS) was performed, which revealed hyperechoic lesions consistent with microabscesses in the right hepatic lobe and dilation of the distal common bile duct (CBD) to 9 mm, with a 6 mm hyperechoic image in its distal third suggestive of biliary lithiasis. During the same anesthetic session, an ERCP was performed without rectal administration of nonsteroidal anti-inflammatory drugs (NSAIDs). Purulent fluid was observed, and the CBD measured 6 mm in diameter. A 5 mm stone was successfully extracted. As adequate clearance of the contrast medium was not achieved, a plastic biliary stent was placed without complications (**Figure 1**). In light of cholangitis,

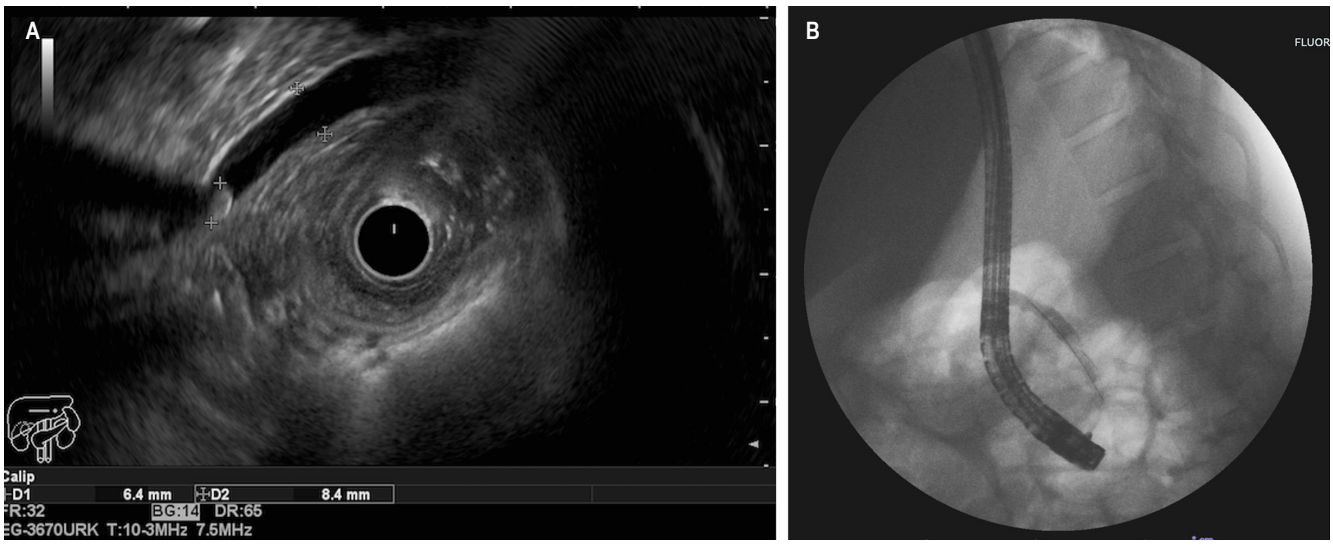


Figure 1. Diagnostic Imaging. **A.** Endoscopic ultrasound of the biliopancreatic region showing distal common bile duct dilation to 8.4 mm with a 6.4 mm biliary stone in its distal third. **B.** Fluoroscopic image from ERCP showing a 6 mm CBD and a 5 mm stone, successfully extracted without complications. Author's File.

antibiotic therapy with intravenous piperacillin/tazobactam 4.5 mg every six hours was initiated.

Forty-eight hours after the procedure, the patient developed upper abdominal pain radiating to the back, requiring opioid analgesics. Serum amylase levels were noted to be elevated to 18 times the upper normal limit, leading to a diagnosis of post-ERCP pancreatitis. On the same day, the patient reported bilateral visual field disturbances, which caused difficulty reading and perceiving objects. A neuro-ophthalmology evaluation revealed normal visual acuity on physical examination but an incongruent binasal visual field defect with left-sided predominance, observed through confrontational visual field testing and better characterized with the Amsler grid (**Figure 2**). A contrast-enhanced brain MRI was requested, showing diffusion restriction in both lateral geniculate bodies (LGBs), with low values on the apparent diffusion coefficient (ADC) map and hyperintensity in fluid-attenuated inversion recovery (FLAIR)/T2 sequences localized to the lateral aspect of the LGBs (**Figure 3**). Infectious and autoimmune causes were ruled out, and no metabolic or electrolyte disturbances were observed during hospitalization to account for the lesions. These findings were attributed to the inflammatory process associated with severe acute post-ERCP pancreatitis, which was also characterized by multiple pancreatic collections on abdominal CT. These collections showed favorable clinical evolution, and the patient's pain resolved completely. Upon discharge, the patient still exhibited persistent incongruent binasal hemianopia. However, during outpatient follow-up at 12 weeks, she reported improvement in her visual field defects.

DISCUSSION

ERCP is a diagnostic and therapeutic tool for various biliopancreatic pathologies[7]. Over the past two decades, the use of ERCP for therapeutic purposes has increased, while its use as a diagnostic tool has declined[8]. ERCP is an invasive procedure associated with adverse events, with an overall incidence rate of approximately 10%, varying across studies based on patient characteristics, procedural complexity and volume, endoscopist experience, and prophylactic measures employed[9].

Among post-ERCP complications, pancreatitis is the most common, occurring in 5%–15% of cases, followed by hemorrhage (0.3%–9.6%), cholecystitis (0.5%–5.2%), cholangitis (0.5%–3%), and perforation (0.08%–0.6%). Rarely, outbreaks of infections linked to contamination of duodenoscope equipment have also been reported[1]. Although the majority of post-ERCP pancreatitis cases are considered mild (95%), this complication can carry a mortality rate of up to 2.8%[2].

The concept of post-ERCP pancreatitis was introduced in 1991, and its definition has since evolved[2]. Currently, the diagnosis is considered in patients who develop new or worsening abdominal pain 24 hours after undergoing ERCP, associated with serum amylase or lipase levels exceeding three times the upper normal limit, or imaging evidence of pancreatitis, requiring hospitalization or prolongation of the hospital stay, as observed in the present case[10]. Notably, this patient presented symptoms 48 hours after the procedure, which is unusual and suggests the need

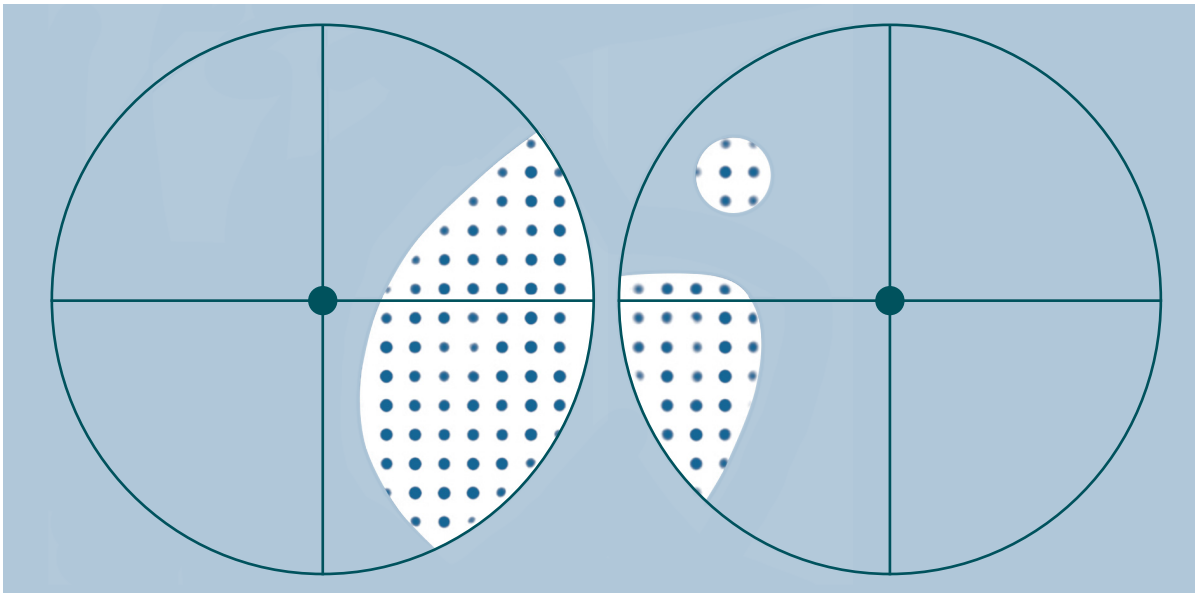


Figure 2. Diagram of the Patient's Visual Fields Based on the Amsler Grid. Author's own research.

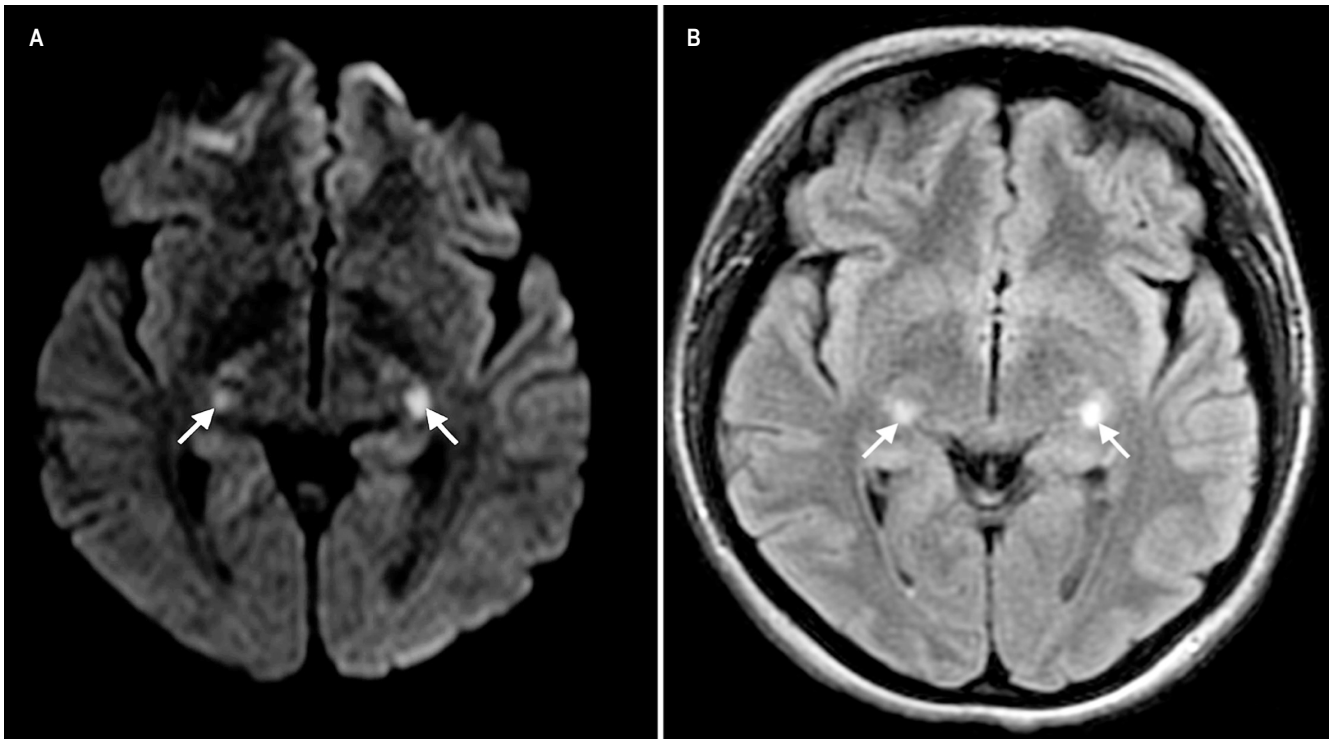


Figure 3. Brain MRI Findings. **A.** Diffusion-weighted imaging (DWI) showing signal enhancement in the lateral geniculate bodies, predominantly on the left side (arrows). **B.** FLAIR images with hyperintensities in the same distribution. Author's File.

for longer observation periods for such patients, particularly in outpatient settings.

Post-ERCP pancreatitis should be suspected in cases of post-procedural abdominal pain, with evaluation of serum amylase or lipase levels to distinguish it from other post-ERCP complications, such as perforation, cholangitis, or unresolved biliary lithiasis. Diagnostic imaging may also be warranted to rule out structural causes of pancreatitis[2]. In confirmed cases, the revised Atlanta classification is recommended to describe local and systemic complications, as it may better predict the severity and mortality of post-ERCP pancreatitis compared to consensus criteria[2].

While the pathophysiological mechanisms of acute pancreatitis are not fully understood, intra-acinar signaling events play a critical role. These include elevated cytosolic calcium levels, mitochondrial depolarization, and cytochrome C leakage, alongside nuclear factor-κB activation via IkB dissociation and proteasomal degradation, culminating in nuclear translocation of p65. This process promotes the synthesis of inflammatory mediators (cytokines and chemokines), leading to neutrophil infiltration in the pancreas[11]. Concurrently, lysosomal dysfunction, zymogen granule alterations, and increased oxidized glutathione due to reactive oxygen species (ROS), coupled with concurrent deleterious mechanisms and impaired autophagy, perpetuate acinar injury[11]. Additional mechanisms include loss of apical microvilli, inhibition of apical secretion, F-actin reorganization, basolateral vesicle formation, release of damage-associated molecular patterns (DAMPs) activating inflammasomes, and leakage of exocrine enzymes such as trypsin and pancreatic lipase. These enzymes cause unregulated hydrolysis of adipocyte triglycerides, resulting in fat necrosis and the production of unsaturated fatty acids that inhibit mitochondrial complexes I and V, thereby reducing adenosine triphosphate (ATP) production. This exacerbates local injury and systemic inflammatory responses[11]. These insights elucidate the multi-organ involvement of acute pancreatitis and the emergence of unusual manifestations, as seen in the present case.

In the presented clinical case, the diagnosis of post-ERCP pancreatitis was confirmed based on the onset of abdominal pain, marked elevation of serum amylase, and imaging findings showing bilateral lesions of the lateral geniculate bodies (LGBs), which coincided with acute visual field deficits.

The LGBs are thalamic nuclei located in the dorsolateral thalamus[12] and are critical in the visual pathway. They receive retinal projections and send signals to the primary

visual cortex[13]. Measuring 4–6 mm in diameter[14], the LGBs are structured into six cellular layers. Layers 2, 3, and 5 receive ipsilateral input (temporal retina), while layers 1, 4, and 6 receive contralateral input (nasal retina). Each layer is composed of distinct neuronal populations characterized by their size: magnocellular neurons are found in layers 1 and 2, while parvocellular neurons are located in layers 3 through 6[15]. The layers have a retinotopic organization, with macular vision represented centrally, the superior visual field in the lateral horn, and the inferior visual field in the medial horn of the LGB[16]. The LGBs are connected to retinal ganglion cells through their axons, which travel via the optic nerves and optic tracts[16].

Projections from the LGBs predominantly terminate in the primary visual cortex via optic radiations, with a smaller proportion reaching extra-striate cortical areas such as the thalamic reticular nuclei[16]. The LGBs are vascularized by dual arterial supply: the anterior choroidal artery, a proximal branch of the internal carotid artery, which supplies the medial and lateral horns, and the posterior choroidal artery, a branch of the posterior cerebral artery, which irrigates the hilum of the LGB[16]. These structures are also considered centers of signal modulation and integration[17], with roles that extend beyond simple signal relay. They regulate the flow and strength of visual information sent to the cortex, control extraretinal afferent input to the LGB[18], encode visual attention[19], and perform additional integrative functions[17].

Lesions of the LGBs are rare. Unilateral involvement typically causes congruent bilateral homonymous hemianopia[20] or partial homonymous anopia. In the context of multiple sclerosis, such lesions result from demyelinating processes. However, thromboembolic events have also been described, leading to variability in visual defects due to the anatomical diversity of LGB vascularization and anastomoses[21].

As presented in this case, bilateral lesions of the lateral geniculate bodies (LGBs) have been infrequently described in the literature (**Table 1**), and the underlying mechanisms for their involvement remain poorly understood. The first known report was published in 1933 by MacKenzie and colleagues, who described these lesions in the context of anterior choroidal arteritis secondary to syphilis[22]. Since then, various mechanisms have been proposed to explain such injuries. Among these are hypoperfusion and ischemia resulting from hemodynamic changes secondary to massive hemorrhage or anaphylactic shock, as the LGBs lie in a vascular border zone supplied by the anterior, middle, and posterior arterial territories[22]. Other proposed mechanisms include posterior circulation cerebral infarcts in patients with microangiopathic hemolytic anemia, traumatic brain injury, and methanol-induced cytotoxicity (pancreatitis and renal necrosis). Due to the histological similarity between the LGBs and the brainstem pons, it has been hypothesized that the tightly interwoven neuronal and oligodendroglial networks in the LGBs hinder cellular expansion during rapid sodium level increases, resulting in bilateral extrapontine geniculate myelinolysis due to overly rapid correction of hyponatremia[23]. Additional etiologies reported include aseptic lateral geniculitis, H1N1 influenza, severe preeclampsia, inflammatory bowel disease, and anaphylactic shock[24].

Interestingly, recent reports of such lesions frequently occur in the context of acute abdominal pain or pancreatitis, often as complications of ERCP. Six cases were identified in the reviewed literature[23,24,28–31]. Another notable case was reported by Pun and colleagues, where vision loss occurred as a complication of ERCP, though central nervous system lesions could not be documented because the patient declined neuroimaging (**Table 1**)[32].

It is noteworthy that, to date, since the first case reported in 1933, 92% of patients in the literature with bilateral LGB lesions have been female; only a single case has been reported in a male patient[29]. The reason for this relationship remains unknown, or it could represent an incidental finding. In our patient, the only preexisting condition was obesity, similar to the case reported by Murugesan and colleagues[31], which also identified acute pancreatitis as a possible etiology. However, unlike our case, their patient was diagnosed with associated thrombotic microangiopathy. In our case, there were no hemodynamic disturbances or electrolyte imbalances requiring correction, no history of traumatic brain injury, and infectious, autoimmune, and autoinflammatory causes were excluded. Based on these findings, we propose severe post-ERCP acute pancreatitis and multiorgan involvement secondary to the underlying systemic inflammatory response as a plausible etiology. The damage to the blood-brain barrier caused by the release of trypsin, elastase, lipase, and phospholipase A2 may allow the infiltration of leukocytes and proinflammatory cytokines (tumor necrosis factor-alpha [TNF-α], interleukin-1β, and interleukin-6), potentially explaining neuronal edema, demyelination, or hemorrhagic necrosis in these structures, as has been suggested in cases of pancreatic encephalopathy[33].

Conflict of Interest

The authors report no conflicts of interest in relation to this article.

Table 1. Reported Cases of Bilateral LGB Lesions

Year	Author	Age (years)	Sex	Proposed Etiology	Remarks
1933	Mackenzie and colleagues[34]	No data	Female	Syphilitic arteritis	Motor deficit resolved completely; pathology revealed syphilitic gummas
1972	Merren[25]	37	Female	Pancreatitis, microangiopathy, methanol toxicity (?)	Associated renal failure; LGB pathology showed coagulative necrosis
1995	Donahue and colleagues[4]	37	Female	Myelinolysis due to rapid correction of hyponatremia	Postoperative case in a patient with alcoholic cirrhosis and chronic hyponatremia
1996	Greenfield and colleagues[6]	28	Female	Lateral geniculitis due to infectious diarrhea	Diarrhea
2002	Imes and colleagues[26]	33	Female	Ischemia; shock from uterine hemorrhage	Respiratory distress was noted
2002	Moseman and colleagues[27]	21	Female	Ischemia, vasoconstriction	Patient with eclampsia
2004	Lefebvre and colleagues[22]	31	Female	Ischemia, anaphylactic shock	
2006	Baker and colleagues[28]	29	Female	Pancreatitis, transient hypotension	Post-ERCP case; high sedation dose required; history of migraine with typical aura; vision improved by day 13.
2007	Mudumbai[23]	18	Female	Pancreatitis, microangiopathy	Oral contraceptive use; acute renal failure (hemodialysis); visual acuity recovered at 32 months.
2015	Viloria and colleagues[29]	40	Male	Pancreatitis, osmotic demyelination due to hydration	Post-cholecystectomy; visual acuity recovered in 6 weeks.
2015	Bartel[24]	22	Female	Pancreatitis, ischemia, transient hypotension	Post-normal ERCP; received rectal indomethacin.
2016	Mathew et al.[30]	42	Female	Ischemia, bilateral LGB arteritis (?)	Unconfirmed systemic bacterial infection; history of psoriasis.
2023	Murugesan et al.[31]	22	Female	Pancreatitis, microangiopathy	Obesity; recent initiation of paleo diet.

Author's own research.

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