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Case Report

When the liver fails, vision fades: A case of hepatic encephalopathy leading to cortical blindness

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Abstract

Cortical blindness is an uncommon but recognized neurological complication of hepatic encephalopathy, and its occurrence has been linked to several underlying pathophysiological mechanisms. The aim of this report was to describe the clinical presentation of cortical blindness in a patient with hepatic encephalopathy and to discuss the potential mechanisms and diagnostic implications associated with this condition. A 50-year-old woman presented with sudden, complete bilateral visual loss after four days of abdominal pain, bloating, nausea, constipation, and abdominal distension. Progressive and painless visual deterioration had been experienced over the preceding two weeks. A four-year history of cirrhosis with poor treatment adherence was noted. On examination, the Glasgow Coma Scale (GCS) score was 13, and scleral icterus was not observed. Both pupils were round and reactive to light. However, no light perception was detected in either eye. The anterior segment examination was unremarkable, whereas ascites and peripheral edema were identified. Abdominal ultrasonography revealed hepatic cirrhosis, splenomegaly, and ascites. Electroencephalography and magnetic resonance imaging were not performed. Cortical blindness secondary to grade II hepatic encephalopathy was diagnosed, and therapeutic paracentesis was performed. However, neither the level of consciousness nor visual function improved after two days of hospitalization, and the patient was subsequently referred to a tertiary hospital. The patient died three days after admission to the referral hospital. In this patient, visual loss occurred in the setting of hepatic encephalopathy and decompensated cirrhosis. Although visual recovery may occur following prompt management, prolonged metabolic disturbance may result in irreversible cortical injury.

Keywords: Cortical blindness, hepatic encephalopathy, liver cirrhosis, vision loss, neurotoxicity

Introduction

Liver he global incidence and prevalence of cirrhosis and hepatic encephalopathy have continued to rise, and despite advances in hepatology, substantial morbidity and mortality continue to be observed, with cirrhosis estimated to affect approximately 2–3% of the global population [2,3]. These manifestations have been attributed to multiple interrelated processes, including hepatocellular injury, neurotoxin accumulation, systemic inflammation, and metabolic imbalance, all of which may adversely affect neurological status [4,5]. Among the principal mechanisms proposed, hyperammonemia, impaired neurotransmission, and blood-brain barrier

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dysfunction have been implicated in the development of altered consciousness and cognitive impairment [6]. Owing to central nervous system involvement, rare but clinically important neurological complications should also be considered in the setting of advanced liver disease [7].

One rare but clinically relevant neurological manifestation is cortical blindness, which is defined as loss of vision resulting from dysfunction of the occipital cortex despite anatomically preserved ocular structures [8]. In patients with hepatocellular disease, elevated blood ammonia levels may exert neurotoxic effects through metabolic disturbance, cytotoxic edema, and astrocytic injury [9]. Several case reports have suggested a possible association between cortical blindness and liver disease, particularly because visual loss has been reported to improve following correction of the underlying metabolic derangements [10]. Nevertheless, scientific evidence regarding the prevalence, mechanisms, and prognosis of cortical blindness associated with liver disease remains limited [11]. Consequently, an important knowledge gap persists regarding the relationship between the severity of hepatic dysfunction and the development of visual impairment [12]. This gap may partly be explained by limited clinician awareness of the clinical characteristics and potential mechanisms underlying cortical blindness in hepatic disease [10]. Further investigation is therefore warranted to better characterize this uncommon manifestation.

The aim of this study was to report a case of cortical blindness occurring in the setting of hepatic encephalopathy and to describe its clinical manifestations, possible etiological mechanisms, and diagnostic implications. The findings presented herein may support the concept that metabolic disturbances secondary to liver failure can result in cortical visual impairment. In addition, this report may assist clinicians and researchers in improving recognition and management of this condition, broadening understanding of its pathogenesis, and informing strategies for earlier diagnosis and appropriate treatment.

Case

A 50-year-old woman was brought to the hospital because of bilateral visual loss. Progressive visual deterioration had been experienced for approximately two weeks, whereas complete loss of vision in both eyes was reported to have occurred four days before presentation. During this period, abdominal pain and a sensation of abdominal fullness were also reported. According to family members, before the onset of progressive visual decline, the patient had remained physically active and independent in daily activities. A four-year history of cirrhosis secondary to hepatitis C had been documented before presentation (Child-Pugh class B), and propranolol, spironolactone, and turmeric had been taken intermittently.

On arrival at the emergency department, the recorded vital signs were as follows: blood pressure 118/68 mmHg, pulse rate 84 beats/minute, respiratory rate 20 breaths/minute, temperature 36.2°C, and oxygen saturation 100% on room air. The Glasgow Coma Scale (GCS) score was 13 (E4M5V4), indicating mild impairment of consciousness. Scleral icterus was not observed, and both pupils were round, isocoric, and reactive to light. Despite preserved pupillary reflexes, no light perception was detected in either eye. No abnormality was identified on anterior segment examination. On general physical examination, ascites and bilateral lower-extremity edema were observed, consistent with progression of chronic liver disease and grade II hepatic encephalopathy.

Laboratory evaluation showed anemia, with hemoglobin level of 9.8 g/dL and hematocrit of 30.8%, leukocyte count of 7,700/µL, and thrombocytopenia, with a platelet count of 98,000/µL (**Table 1**). Coagulation studies demonstrated a prothrombin time of 17.5 seconds, an activated partial thromboplastin time of 36.9 seconds, and an international normalized ratio of 1.36. Additional biochemical findings included albumin 3.8 mg/dL, sodium 144.1 mmol/L, potassium 5.1 mmol/L, chloride 106.8 mmol/L, blood glucose 253 mg/dL, and glycated hemoglobin (HbA1c) 6.0%. Abdominal ultrasonography demonstrated hepatic cirrhosis, splenomegaly, and ascites. Electroencephalography and magnetic resonance imaging were not performed, thereby limiting further evaluation of cortical function and structural abnormalities.

Table 1. Laboratory findings of the patient

Parameter	Result
Hematology	
White blood cell count (WBC), /µL	7,700
Hemoglobin, g/dL	9.8
Hematocrit, %	30.8
Platelets, /µL	98,000
Coagulation	
Prothrombin time (PT), second	17.5
Activated partial thromboplastin time (APTT), second	36.9
International normalized ratio (INR)	1.36
Biochemistry	
Albumin, mg/dL	3.8
Sodium, mmol/L	144.1
Potassium, mmol/L	5.1
Chloride, mmol/L	106.8
Blood glucose, mg/dL	253

Based on the clinical findings of sudden bilateral visual loss with preserved pupillary reflexes, an unremarkable ocular examination, hepatic dysfunction, and altered mental status, cortical blindness secondary to hepatic encephalopathy was diagnosed (**Figure 1** and **Figure 2**). Initial management consisted of fresh frozen plasma transfusion, analgesics, antiemetics, furosemide, antibiotics, and therapeutic paracentesis. Despite these interventions, neither visual function nor the level of consciousness improved during the first two days of hospitalization. The patient was therefore referred to a tertiary care center. Following three days of treatment at the referral hospital, the patient died.

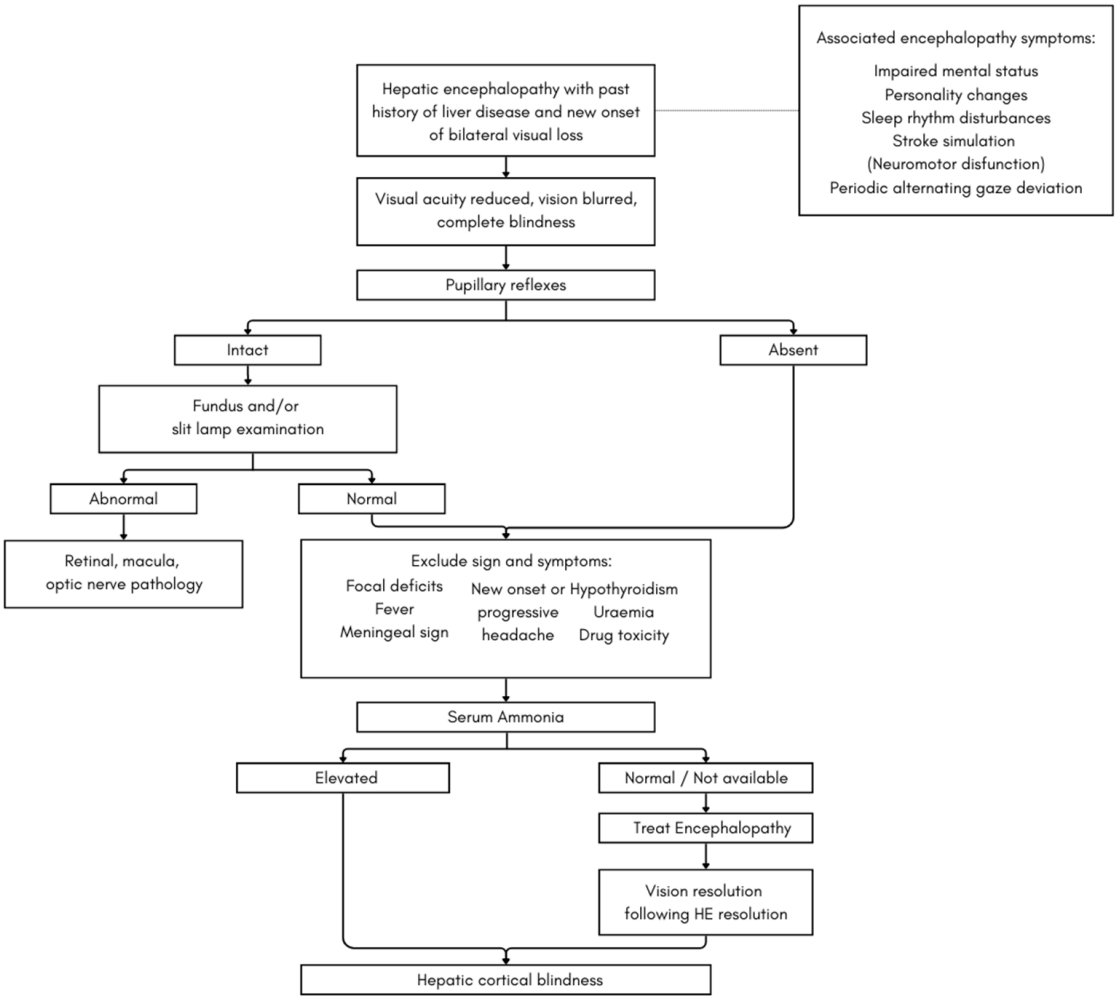

Figure 1. Diagnostic approach to cortical blindness associated with hepatic encephalopathy in this case report.

Discussion

Cortical blindness represents an uncommon but clinically important neurological manifestation in patients with hepatic encephalopathy and advanced liver disease. This report is consistent with previously published cases of acute-on-chronic liver failure in which visual impairment occurred in the absence of detectable ocular abnormalities [10,13]. Cortical blindness has been described in several case reports as a rare neurological complication of hepatic encephalopathy, frequently in association with hyperammonemia and other metabolic disturbances. For example, a previous case report showed that visual impairment was reversed after correction of the underlying metabolic abnormalities [10]. Similar observations have been described in other report, in which improvement in visual function was observed following early recognition and appropriate treatment of hepatic encephalopathy [13]. In contrast, no immediate improvement in visual function was observed in the present patient before referral, which may indicate more severe or more prolonged metabolic injury involving the occipital cortex. This difference may have been attributable to delayed treatment, progression of chronic liver disease, or persistent neurotoxicity. Collectively, these findings suggest that the underlying process was systemic and localized to the cortical visual pathways rather than to the ocular tissues [16]. The visual disturbance may be explained by elevated ammonia levels, which are known to interfere with neurotransmission and promote cellular swelling [17]. In addition, neurological impairment may have been exacerbated by poor treatment adherence, metabolic instability, and progressive deterioration of liver function [18]. Therefore, the present case supports the view that the pattern of visual loss observed was compatible with the atypical neurological manifestations previously reported in patients with acute-on-chronic liver failure [19].

Pupillary reflexes were preserved on clinical examination, and the anterior segment appeared normal. No focal neurological deficits were identified. These findings suggest that the visual loss was more likely attributable to cortical dysfunction than to ocular or optic nerve pathology. This interpretation is consistent with published reports describing complete visual loss despite normal ocular findings, in which impaired cortical visual processing secondary to metabolic disturbance was subsequently considered the most plausible explanation [20,21]. Additional reports have similarly supported this dissociation between normal ocular examination findings and profound visual loss, thereby indicating cortical involvement [22,23]. In the present case, the findings may suggest heightened vulnerability of the occipital cortex to ammonia-related neurotoxicity [24]. Because advanced diagnostic investigations, such as magnetic resonance imaging and electroencephalography, were not available, definitive confirmation of cortical involvement could not be established. Nevertheless, several alternative diagnoses were considered. Occipital infarction was considered less likely because focal neurological deficits were absent and pupillary reflexes were preserved. Posterior reversible encephalopathy syndrome was also considered less likely because acute hypertension, seizures, and typical predisposing factors were not present. Toxic-metabolic encephalopathy of non-hepatic origin was also contemplated; however, the presence of chronic liver disease together with clinical features of hepatic encephalopathy strongly supported hepatic-related cortical dysfunction. This case illustrates that severe visual loss in a patient with decompensated liver disease, particularly in the absence of ocular abnormalities, should prompt consideration of cortical dysfunction.

The pathogenesis of this disorder has been proposed to involve ammonia neurotoxicity and astrocytic dysfunction, which may lead to excessive conversion of ammonia to glutamine, intracellular glutamine accumulation, and selective vulnerability of the primary visual cortex to neuronal injury [25,26] (**Figure 2**). In addition to these cellular alterations, impaired astrocytic function may directly disrupt the transmission and processing of visual signals within the visual cortex and associated regions, including the lingual gyrus and fusiform gyrus [27,28]. Accordingly, although the visual pathway may remain intact through subcortical connections, thereby preserving the pupillary light reflex, conscious visual perception may be lost, resulting in cortical blindness despite preserved ocular function [29]. Additional indirect mechanisms may also have contributed, including electrolyte imbalance, systemic inflammation, cerebral hypoperfusion, and poor adherence to treatment, all of which may further increase metabolic stress on cortical tissue [30]. Diagnostic investigations such as electroencephalography and diffusion-weighted or fluid-attenuated inversion recovery magnetic resonance imaging may

theoretically help clarify these mechanisms, although such examinations were not available in the present case [31]. Taken together, these mechanisms provide a biologically plausible explanation for how impaired hepatic metabolic function may damage the structure and function of the visual cortex and thereby produce visual loss in hepatic encephalopathy [26,28].

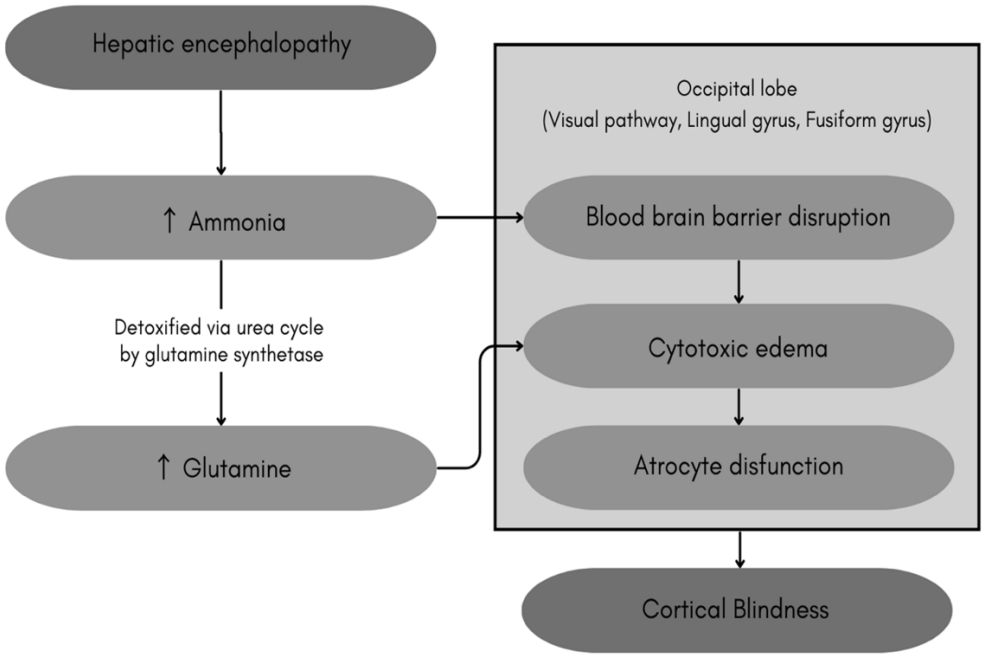

Figure 2. Proposed pathogenesis of cortical blindness associated with hepatic encephalopathy.

This case report has some important clinical implications. First, it indicates that profound visual loss in hepatic encephalopathy may originate from cortical dysfunction even when ocular structures appear normal. This point is clinically important because preserved pupillary reflexes and an unremarkable anterior segment examination may falsely reduce suspicion of a central neurological cause. Recognition of this possibility may therefore support more accurate diagnosis and prevent the visual disturbance from being attributed solely to primary ocular pathology. Second, the present case underscores the importance of early clinical recognition, particularly in settings where advanced diagnostic modalities are not readily available. In such circumstances, careful interpretation of the clinical presentation remains essential, and the coexistence of severe bilateral visual loss, normal ocular findings, and hepatic dysfunction should prompt consideration of cortical blindness related to hepatic encephalopathy. Earlier recognition may facilitate more timely metabolic correction and supportive management. Third, this case highlights the need for further investigation into factors that may determine visual recovery. In particular, the duration of metabolic disturbance, the severity of encephalopathy, and the timeliness of treatment may all influence whether cortical dysfunction is reversible or progresses to more persistent injury.

This study however has some limitations. Potential confounding factors, including metabolic instability, poor treatment adherence, subclinical infection, and electrolyte fluctuation, may have influenced the interpretation of the findings. The absence of advanced diagnostic evaluations, such as electroencephalography and magnetic resonance imaging, reduced the ability to exclude alternative neurological etiologies, including occipital infarction and encephalitis. In addition, the lack of long-term follow-up precluded assessment of the reversibility and prognosis of the visual impairment.

Conclusion

This case report demonstrates that visual disturbance may occur in patients with hepatic encephalopathy even in the absence of identifiable ocular abnormalities. Cortical blindness should therefore be recognized as a possible neurological manifestation of hepatic metabolic

dysfunction. In such patients, preserved pupillary reflexes, a normal anterior segment examination, and the absence of focal neurological deficits may indicate that the visual disturbance originates from cortical rather than ocular pathology. The present findings may contribute to greater clinical awareness of this uncommon presentation, support more appropriate diagnostic consideration, and encourage further investigation into prognostic factors and the reversibility of cortical visual dysfunction associated with hepatic encephalopathy and cirrhosis.

Ethics approval

Ethics approval was not required for this case report. Written informed consent for publication of the clinical details was provided by the patient's son.

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Competing interests

All authors declared that there were no competing interests.

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Underlying data

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Declaration of artificial intelligence use

Artificial intelligence tool, ChatGPT, was used for language refinement. All AI-assisted outputs were critically reviewed by the authors to ensure the integrity and reliability of the manuscript. All final interpretations and decisions presented in this article were made solely by the authors.

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