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Diagnostic Dilemma: Incidental Diagnosis of Decompensated Liver Cirrhosis during Evaluation of Unexplained Neurological Decline in an Elderly Patient: A Case Report

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Abstract

Background: Neurological deterioration in older adults is frequently correlated to cerebrovascular disease, spinal pathology, or age-related neurodegenerative processes. However, such presentations are multifactorial, and systemic illnesses should be considered part of the differential diagnosis that contribute significantly to cognitive and functional decline. **Case Presentation:** A 69-year-old man who presented with progressive confusion, gait instability, recurrent falls, and functional deterioration following a fall, neurological examination was done a distinct sensory deficit below the T5 - T6 dermatomal level was incidentally noted during an abdominal examination, Initial neurological evaluation was initiated, including brain and spinal imaging, which revealed chronic microangiopathic changes, cervical spinal stenosis, a T12 compression fracture, and a possible lacunar infarct. However, these findings were insufficient to explain the severity of his cognitive impairment, raising concern for a clinicoradiological mismatch. Further evaluation included abdominal CT, which revealed a large-volume ascites and features of chronic liver disease. Diagnostic paracentesis was performed and demonstrated portal hypertensive ascites with a serum-ascites albumin gradient (SAAG) greater than 1.1 g/dL. Laboratory investigations showed hypoalbuminemia, prolonged coagulation, elevated liver enzymes, and increased serum ammonia levels. The patient was diagnosed with decompensated alcohol-related liver cirrhosis (Child-Pugh Class C) complicated by hepatic encephalopathy. Treatment with lactulose, rifaximin, spironolactone, and carvedilol was initiated, and clinical improvement was noted; the patient was subsequently transferred back to the rehabil-

itation department for ongoing care. **Conclusion:** This case demonstrates the importance of maintaining a broad differential diagnosis when imaging findings do not fully explain neurological symptom, that unexplained neurological deterioration in older adult may be the first manifestation of an underlying systemic illness. Recognition of clinicoradiological discordance may uncover systemic diseases with major implications for management, rehabilitation, and prognosis.

Keywords

Hepatic Encephalopathy, Liver Cirrhosis, Ascites, Diagnostic Dilemma, Neurological Decline, Multimorbidity, Older Adults

1. Introduction

Neurological complaints are a leading cause of hospital admission among older adults and are commonly attributed to cerebrovascular disease or degenerative spinal disorders [1]. However, these presentations may reflect underlying systemic disease or multimorbidity, particularly in older populations. Decompensated liver cirrhosis is a chronic, progressive condition frequently associated with ascites, portal hypertension, and metabolic derangements [2] [3]. Although ascites is typically identified through abdominal symptoms or physical examination, it may occasionally be detected incidentally during evaluation for unrelated complaints [3] [4]. We present a case in which neurological symptoms initially led diagnostic reasoning toward central and spinal pathology; however, further investigation revealed previously unrecognized decompensated liver cirrhosis.

2. Case Presentation: Initial Presentation and Neurological Workup

A 69-year-old male with a medical history of benign prostatic hyperplasia, psoriasis, chronic alcohol use, and active smoking was admitted to the neurological rehabilitation department following a fall at home that initially suspected to be related to a transient ischemic attack (TIA). He presented with a several-week history of progressive mobility decline and sensory loss. On admission, the patient exhibited mild cognitive impairment and reduced functional capacity. A neurological examination revealed left upper-limb weakness (Medical Research Council grade 4/5). Additionally, a distinct sensory deficit below the T5-T6 dermatomal level was incidentally noted during an abdominal examination. Given these focal neurological findings, a primary central nervous system etiology was suspected. An emergent non-contrast computed tomography (CT) scan of the head (**Figure 1**) was performed to rule out acute intracranial pathology; the scan demonstrated no evidence of acute hemorrhage, space-occupying mass lesions, or acute territorial infarction.

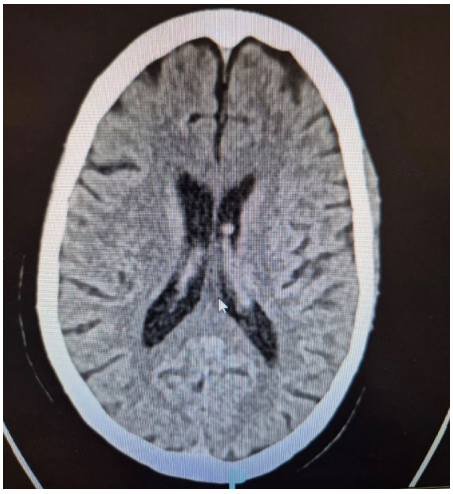

Figure 1. Non-contrast CT scan of the brain demonstrating no acute intracranial hemorrhage, mass lesion, or territorial infarction.

Magnetic Resonance Imaging (MRI) of the spine identified an acute T12 compression fracture without spinal canal stenosis or cord compression. A brain MRI showed non-specific periventricular white matter changes consistent with small vessel disease and a suspected minor subacute lacunar infarct in the left thalamus. However, these findings did not fully explain the patient's global disorientation and severe cognitive deficits, highlighting a profound clinicoradiological mismatch. Electromyography (EMG) findings demonstrated right peroneal neuropathy and acute L3 - L4 radiculopathy, consistent with background nutritional or alcoholic polyneuropathy. Neuroimaging did not demonstrate an acute cerebrovascular event sufficient to explain the severity of cognitive dysfunction. No laboratory evidence of infection or electrolyte disturbance explained his neurological symptoms. Then, alternative causes of altered mental status were systematically evaluated.

3. Incidental Abdominal Findings and Targeted Workup

As a part of an internal medicine evaluation, secondary to a mild abdominal distension observed on physical examination and distinct sensory deficit below the T5 - T6 dermatomal level was incidentally noted during an abdominal examination expanded imaging was performed. An abdominal X-ray and subsequent abdominal computed tomography (CT) scan (Figure 2) revealed significant, large-volume ascites and morphological features of chronic liver disease then a diagnostic paracentesis was performed.

4. Laboratory and Paracentesis Analysis

Laboratory investigations showed preserved renal function and a normal complete blood count with hemoglobin 14 g/dl, WBC 5.8×10^9/L and normal platelet count 191×10^9/L. In contrast, serum albumin was reduced (3.1 - 3.2 g/dL), coagulation studies were prolonged (a prolonged prothrombin time of 19.4 seconds

and an elevated international normalized ratio (INR) of 1.50), and cholestatic liver enzymes were elevated (elevated gamma-glutamyl transferase (365 - 492 U/L) and alkaline phosphatase (225 - 275 U/L), with mildly elevated aspartate aminotransferase (39 - 45 U/L)). Serum ammonia measured 113.3 μg/dL, supporting a metabolic cause for the patient's encephalopathy.

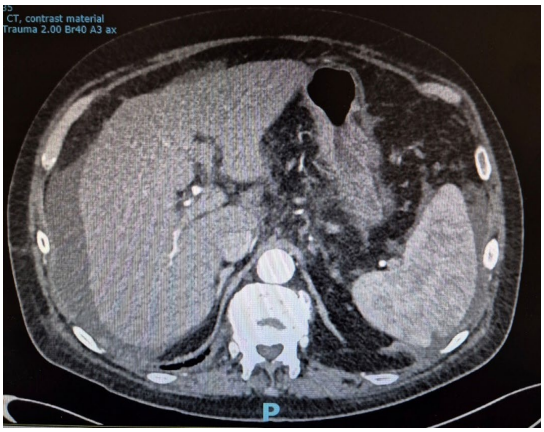

Figure 2. Contrast-enhanced abdominal CT demonstrating large-volume of abdominal free fluid, cirrhotic liver morphology.

Analysis of the ascitic fluid demonstrated an albumin concentration of 0.65 g/dL and a total protein concentration of 1.2 g/dL. The calculated serum-ascites albumin gradient (SAAG) exceeded 1.1 g/dL, indicating portal hypertensive ascitesThe calculated Serum-Ascites Albumin Gradient (SAAG) was greater than 1.1 g/dL, confirming the presence of sinusoidal portal hypertension. Ascitic fluid leukocyte count was 656 cells/μL, with 24.5% polymorphonuclear (PMN) cells (absolute PMN count <250 cells/μL), effectively ruling out spontaneous bacterial peritonitis (SBP).

Further evaluation revealed a documented history of chronic alcohol consumption. Viral hepatitis screening—including hepatitis B surface antigen (HBsAg), hepatitis B core antibody (anti-HBc), and hepatitis C antibody (anti-HCV) and Extended immunological profiles, including anti-smooth muscle antibody (ASMA), antinuclear antibody (ANA), and anti-neutrophil cytoplasmic antibodies (ANCA), were negative, ruling out autoimmune etiologies. Based on the simultaneous presence of macro-ascites, overt hepatic encephalopathy, hypoalbuminemia, and hypercoagulability, the patient fulfilled the definitive clinical and laboratory criteria for Child-Pugh Class C alcohol-related liver cirrhosis.

5. Therapeutic Intervention

The patient was started on a targeted medical regimen consisting of lactulose (titrated to 2-3 soft stools per day) and rifaximin (550 mg twice daily) for the specific management of hepatic encephalopathy. For portal hypertension and ascites management, spironolactone (100 mg daily) and carvedilol (6.25 mg daily) were initiated under close hemodynamic and renal function monitoring. High-dose intra-

venous thiamine supplementation (500 mg three times daily) was administered empirically for three days to address potential alcohol-related nutritional deficiencies and Wernicke's encephalopathy, followed by oral maintenance.

Following this targeted intervention, a significant and rapid enhancement of the patient's cognitive and functional status was observed. Following stabilization of his metabolic state and complete resolution of his acute confusion, the patient was successfully re-enrolled in an intensive, multidisciplinary rehabilitation program involving focused physical and occupational therapy to manage his orthopedic recovery and deconditioning safely.

6. Outcome and Follow-Up

Following initiation of lactulose, rifaximin, spironolactone, carvedilol, and supportive nutritional therapy, the patient mental status improved gradually his confusion resolved, cognitive function returned close to baseline, his Abdominal distension decreased following medical management, and no cirrhosis-related complications was developed during hospitalization. After stabilization, he was transferred back to the rehabilitation department to continue multidisciplinary care, in physical and occupational therapy. Meanwhile in the rehabilitation department, functional mobility gradually improved, although mild residual gait instability persisted, likely related to deconditioning, peripheral neuropathy, and the T12 compression fracture. At discharge, the patient was medically stable and was transferred back to his home and continued his community-based rehabilitation. Outpatient hepatology follow-up was arranged for ongoing surveillance and management of cirrhosis-related complications, including ascites, hepatic encephalopathy, and portal hypertension.

The patient's clinical course, key diagnostic investigations and therapeutic interventions are summarized in Table 1.

Table 1. Timeline of clinical events and interventions.

	Clinical Status and Interventions
March 23	Onset of progressive confusion, gait instability, recurrent falls, and functional decline
April 6	Hospital admission for evaluation of worsening confusion and neurological findings in physical examination acute cerebrovascular event ruled out.
April 7	Secondary to a mild abdominal distention and sensory deficit below the T5 - T6 dermatomal level was incidentally noted during an abdominal examination. Abdominal CT incidentally revealed large-volume ascites and features of liver cirrhosis, Diagnostic paracentesis performed; fluid analysis confirmed portal hypertension.
Inpatient hospitalization	Diagnosed with Child-Pugh Class C alcohol-related cirrhosis and hepatic encephalopathy; medical therapies initiated.
April 10 hospital Discharge	neurological and functional improvement achieved, transferred to rehabilitation.
MAY 10 home Discharge	The patient was medically stable and was transferred back to his home and continued his community-based rehabilitation and hepatology follow-up.

7. Discussion

The main diagnostic challenge in this case was determining whether the patient's neurological deterioration was caused by a primary neurological disorder or by a systemic metabolic condition. Initial neuroimaging showed chronic microangiopathic changes and a possible subacute lacunar infarct. Although these findings could explain some of the patient's symptoms, they did not fully account for his severe confusion, global disorientation, and rapid functional decline. This discrepancy prompted further investigation for an alternative diagnosis.

In older adults, cognitive decline and recurrent falls are commonly attributed to age-related neurodegenerative disease or chronic cerebrovascular changes, especially when neuroimaging demonstrates small-vessel ischemic disease [1]. In our patient, the acute T12 compression fracture and chronic lumbar radiculopathy explained his pain and impaired mobility but were insufficient to explain the severity of his altered mental status and sensory deficit below the T5 - T6 dermatomal level was incidentally noted during an abdominal examination, Hepatic encephalopathy should be considered in the differential diagnosis of elderly patients presenting with unexplained cognitive impairment, particularly when neurological findings appear disproportionate to imaging results. Its presentation may be subtle and can include cognitive dysfunction, sleep disturbance, gait impairment, and asterixis [5] [6].

The incidental finding of large-volume ascites on the abdominal CT images prompted further evaluation for a systemic cause. Diagnostic paracentesis revealed a serum-ascites albumin gradient (SAAG) greater than 1.1 g/dL, indicating portal hypertension as the underlying cause of the ascites [3]. Combined with the elevated serum ammonia level and a Child-Pugh score of 11 (Class C), these findings were consistent with previously unrecognized decompensated liver cirrhosis complicated by overt Type C hepatic encephalopathy. Chronic alcohol-related polyneuropathy likely contributed to his gait impairment but did not fully explain the acute cognitive decline.

This case illustrates the importance of avoiding diagnostic anchoring. The structural neurological abnormalities identified on imaging initially appeared to explain the patient's presentation, but a broader diagnostic approach led to the recognition of advanced liver disease. Following treatment with lactulose and rifaximin, the patient's mental status improved significantly, supporting the diagnosis of a reversible metabolic encephalopathy. This improvement also allowed him to participate in the rehabilitation program required for recovery from his spinal and orthopedic injuries.

8. Conclusion

This case demonstrates that decompensated liver cirrhosis can develop insidiously in older adults, presenting as an unexplained neurological decline without any other outstanding symptoms. When a clear clinicoradiological mismatch exists between neuroimaging and a patient's functional status, physicians should broaden the diagnostic evaluation to include systemic and metabolic disorders. An expanded differ-

ential diagnosis should be considered, including abdominal assessment and metabolic screening, can reveal treatable etiologies such as hepatic encephalopathy, facilitates rehabilitation, and may substantially improve patient outcomes.

9. Limitations

As a single-patient case report, causality cannot be established with certainty. The patient's presentation was likely multifactorial, involving chronic cerebrovascular disease, peripheral neuropathy, and hepatic encephalopathy. Nevertheless, the marked clinicoradiological mismatch and subsequent clinical improvement following targeted therapy strongly support the contribution of decompensated liver disease to the overall presentation.

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The author would like to thank the medical staff involved in the patient's care and management.

Ethics Approval and Consent to Participate

According to institutional policy, ethical approval was not required for this case report.

Consent to Publish

Written informed consent for publication of this case report and accompanying clinical details were obtained from the patient and/or the patient's legal representative.

Data Availability

No datasets were generated or analyzed during the current study. All relevant clinical information is included within the manuscript.

Author Contributions

An Internal Medicine and Geriatrics resident with a particular interest in geriatric medicine, diagnostic complexity, and interdisciplinary clinical research conceptualized the report, collected and interpreted the clinical data, prepared the manuscript, and approved the final version for publication.

Conflicts of Interest

The author declares that there are no competing interests related to this publication.

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