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Postoperative Intracranial Hypertension Mimicking Depressive Relapse in an Elderly Patient With Long-Standing Depression After Microvascular Decompression: A Case Report

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Case Report

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Abstract

Intracranial hypertension (ICH) typically presents with headache, nausea, and focal neurological deficits. However, atypical neuropsychiatric manifestations are not uncommon in elderly patients, especially those with preexisting psychiatric disorders. We report a 70-year-old woman with a 20-year history of well-controlled depression who underwent uneventful microvascular decompression. Postoperatively, she developed persistent low mood, reduced responsiveness, and poor appetite, which were initially attributed to environmental stress and suspected depressive relapse. Her blood pressure rose to 158/102 mmHg, while cranial CT showed no acute hemorrhage or mass effect. Lumbar puncture revealed markedly elevated intracranial pressure (350 mmH_2O). Following osmotic therapy, her headache, blood pressure, and emotional state improved dramatically without any adjustment to psychiatric medications. This case highlights that ICH can manifest primarily as neuropsychiatric deterioration in elderly patients with chronic depression. Clinicians should consider organic etiologies before attributing postoperative mood changes to psychiatric relapse alone, to avoid diagnostic delay.

Introduction

Classic manifestations of intracranial hypertension include headache, nausea, vomiting, and visual disturbances. However, elderly patients frequently exhibit atypical presentations, such as lethargy, slowed responsiveness, or mood disturbance rather than overt headache or meningeal signs.

In patients with a longstanding psychiatric history, new-onset mood or behavioral changes after surgery are easily misinterpreted as relapse of the underlying mental disorder. Such attribution bias may lead to delayed recognition of life-threatening organic conditions, including raised intracranial pressure. To our knowledge, cases of postoperative ICH mimicking depressive relapse after microvascular decompression remain rarely reported. We present this case to improve clinical awareness.

Case Presentation

A 70-year-old woman with a 20-year history of depression was admitted for microvascular decompression. Her depressive symptoms had been stable for years under combination therapy with estazolam, quetiapine fumarate, mirtazapine, and pramipexole. No changes in psychotropic medications were made during the perioperative period.

She also had a 3-year history of hypertension, well controlled with telmisartan. Preoperative blood pressure was approximately 130/80 mmHg.

The operation was completed uneventfully. On postoperative day 1, the patient became lethargic, less communicative, and reported loss of appetite. She described low mood without obvious anxiety or agitation. These findings were initially considered related to hospitalization stress and lack of family company. Although family presence brought mild relief, her mental state did not return to baseline.

Her blood pressure gradually increased to 158/102 mmHg. She did not complain of severe headache at that time. Emergent cranial CT showed no intracranial hemorrhage, midline shift, or space-occupying lesion (Fig. 1). Small amounts of intracranial air were observed, consistent with recent posterior fossa surgery. No radiological explanation for her clinical deterioration was identified.

Lumbar puncture was performed, revealing an opening pressure of 350 mmH_2O. Routine cerebrospinal fluid examination was unremarkable.

She was treated with intravenous mannitol for ICH. Within a short period, her headache (VAS 8→2), blood pressure (135/85 mmHg), general responsiveness, appetite, and mood all improved significantly. No changes to antidepressant or sedative medications were needed during her hospital stay.

In retrospective inquiry, the patient acknowledged that she had experienced moderate to severe headache shortly after surgery but did not actively report it because she thought it was an expected postoperative discomfort.

Figures

Figure 1. Postoperative axial cranial computed tomography demonstrating no evidence of intracranial hemorrhage, mass effect, or midline shift. Mild postoperative intracranial air is visible. Despite normal imaging, intracranial pressure was markedly elevated (350 mmH_2O).

Table

Table 1
Changes in clinical parameters before and after treatment

Time Point	ICP	BP (mmHg)	Headache VAS	Mood Status
Preoperative	Normal	130/80	0	Stable
Post-onset	Not tested	158/102	8	Low, unresponsive
After treatment	Decreased	135/85	2	Improved

Discussion

This case demonstrates that intracranial hypertension can present predominantly with neuropsychiatric symptoms in elderly patients, particularly those with chronic depression. Several factors contributed to the initial diagnostic challenge.

First, the patient's core manifestations were low mood and reduced responsiveness rather than classic headache or vomiting. Elderly patients often have blunted pain perception and decreased cortical arousal, which may mask typical ICH signs.

Second, attribution bias strongly influenced early clinical reasoning. Given her long and stable depression history, new mood changes were intuitively linked to psychiatric relapse rather than organic

brain dysfunction. This cognitive pitfall has been widely recognized as a major cause of delayed diagnosis in medicine.

Third, the initial cranial CT was unremarkable, which may falsely reassure clinicians. It is important to emphasize that normal brain imaging does not rule out raised intracranial pressure, especially in non-traumatic or non-hemorrhagic conditions.

The pathophysiological link between ICH and mood disturbance remains incompletely understood. Animal and human studies suggest that elevated intracranial pressure may disrupt frontal-limbic networks involved in emotional regulation, thereby inducing depressive-like symptoms. In addition, sympathetic activation secondary to ICH may cause paroxysmal hypertension, as observed in this patient.

Depression itself may further alter pain processing and reduce symptom reporting. This patient underreported headache until directly questioned, which further delayed correct identification.

Notably, the patient's rapid recovery after ICP-lowering treatment—without any adjustment of psychiatric medications—supports a direct causal relationship between ICH and her neuropsychiatric presentation.

Conclusion

Elderly patients with chronic depression may develop intracranial hypertension after posterior fossa surgery and present primarily with mood deterioration, lethargy, and poor appetite, mimicking depressive relapse.

- Normal brain CT does not exclude ICH.
- Lumbar puncture should be considered early in unexplained neuropsychiatric decline.
- Attribution bias must be avoided to ensure timely diagnosis and treatment.

This case underscores the importance of comprehensive neurological evaluation in postoperative patients with preexisting psychiatric disorders.

Patient Perspective

The patient reported that she had significant headache after the operation but considered it a normal postoperative reaction and did not mention it spontaneously. She felt much better mentally and physically after the treatment for high intracranial pressure.

Declarations

Ethics approval and consent to participate

This study was performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. Ethical approval was not required for this case report because it does not involve prospective human research, controlled interventions, or collection of identifiable private data beyond routine clinical care. Written informed consent was obtained from the patient for participation in this case report.

Consent for publication

Written informed consent was obtained from the patient and her family for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal upon request.

Competing interests

The authors declare that they have no competing interests.

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Authors’ contributions

- ZHAO Xinyan: Data collection, clinical assessment, manuscript drafting and revision.
- WENG Jiancong: Study conception, clinical supervision, critical revision of the manuscript, final approval.
- FANG Jia: Literature review, case evaluation, manuscript editing.All authors have read and approved the final manuscript and agree to be accountable for all aspects of the work.

Availability of data and materials

All relevant clinical data supporting the findings of this article are included within this published article. Raw data are available from the corresponding author upon reasonable request.

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Figures

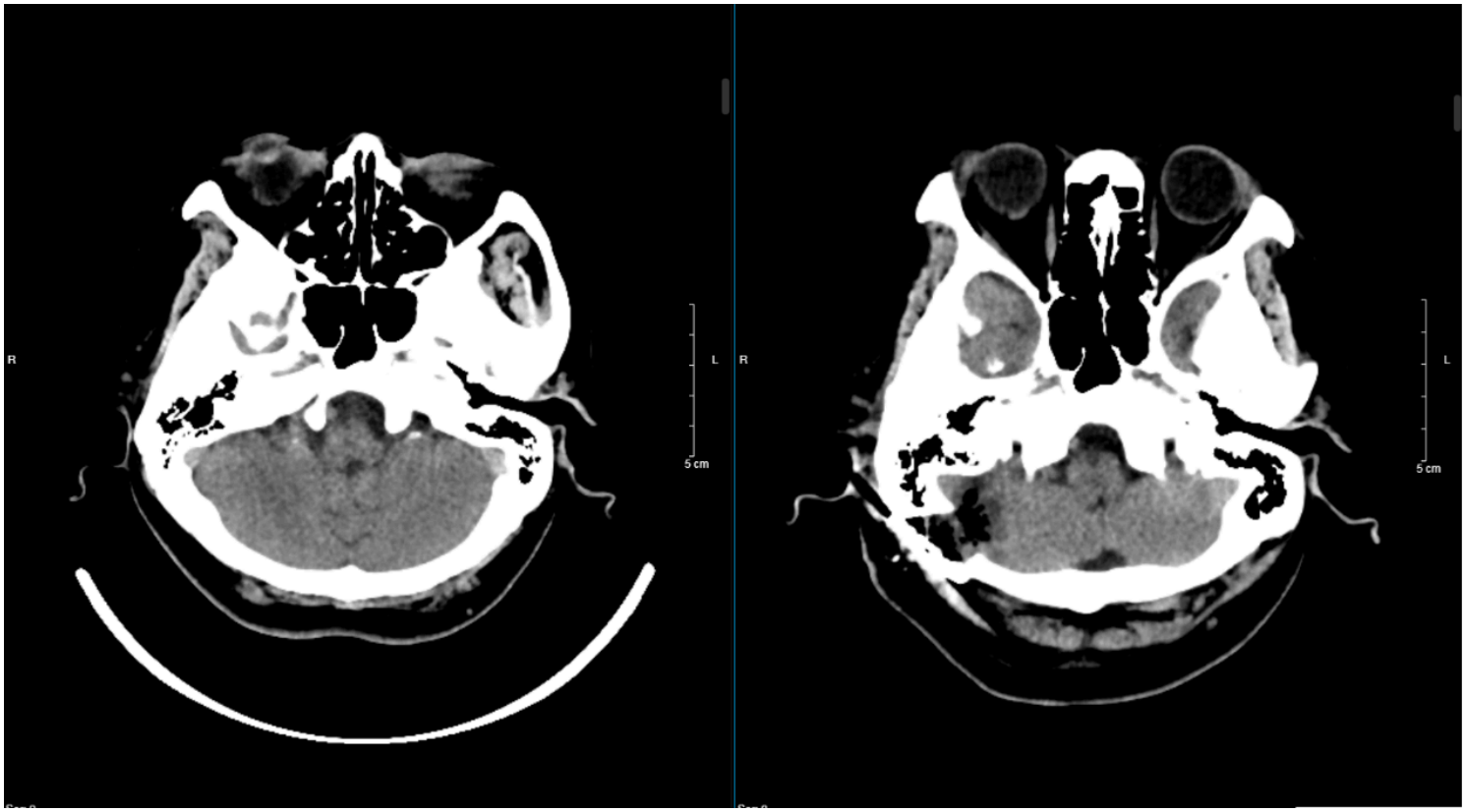


Figure 1

Postoperative axial cranial computed tomography demonstrating no evidence of intracranial hemorrhage, mass effect, or midline shift. Mild postoperative intracranial air is visible. Despite normal imaging, intracranial pressure was markedly elevated (350 mmH_2O).

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