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(CASE REPORT)

BEYOND COMPUTED TOMOGRAPHY: NON-INVASIVE MULTIMODAL NEUROMONITORING IN AN ADOLESCENT PATIENT WITH SEVERE TRAUMATIC BRAIN INJURY AND A RIGHT TEMPOROPARIETAL EPIDURAL HEMATOMA – A CASE REPORT

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ABSTRACT

Severe cranial trauma (TCE) constitutes an important cause of morbidity and mortality and represents a disruption in the neurocritical care system, especially when associated with expansive intracraneal lesions and intracraneal hypertension. We present the case of a 16-year-old male patient who suffered a severe TBI secondary to a fall from a height of approximately 10 meters, with loss of consciousness and a Glasgow Coma Scale score of 7. Computed tomography of the head revealed a right temporoparietal epidural hematoma of approximately 200 mL, cerebral edema, ventricular compression, a 6 mm midline shift, and a small contralateral intraparenchymal hemorrhage. A right temporoparietal craniotomy was performed with evacuation of approximately 180 mL of bloody contents, followed by neurocritical treatment with 3% hypertonic saline solution, mechanical ventilation, and neuroprotective measures. As a complementary brain surveillance strategy, non-invasive multimodal neuromonitoring was employed using optic nerve sheath diameter (OND) ultrasound, transcranial Doppler (TCD), and follow-up computed tomography (CT). The OND decreased from 6.2 to 5.5 mm during the postoperative period, while normalization of the pulsatility index was reported. Initial hypocapnia was also corrected through ventilatory adjustments. At 48 hours, the patient showed favorable clinical progress, no new bleeding on follow-up CT, weaning from mechanical ventilation, and transfer to the

surgical ward. This case illustrates the potential of non-invasive multimodal neuromonitoring as a complement to clinical and radiological evaluation in neurocritically ill patients, particularly in resource-limited settings. However, these parameters do not replace invasive intracranial pressure measurement when indicated.

Keywords: Severe head trauma; Epidural hematoma; Multimodal neuromonitoring; Optic nerve sheath diameter; Transcranial doppler; Intracranial pressure.

1 INTRODUCTION

Traumatic brain injury (TBI) is one of the leading causes of morbidity and mortality worldwide and represents a significant challenge for emergency, neurosurgical, and intensive care services. Among intracranial injuries of traumatic origin, epidural hematoma is characterized by the accumulation of blood between the inner table of the skull and the dura mater, generally as a consequence of vascular injury associated with the trauma. Its clinical course can be variable, ranging from initially stable patients to rapidly progressive neurological deterioration, conditioned by the mass effect, displacement of intracranial structures, and increased intracranial pressure (ICP). Temporoparietal epidural hematoma is of particular relevance due to its potential for expansion and the possibility of causing cerebral compression, alterations in cerebral perfusion, and herniation; therefore, timely recognition and neurosurgical intervention, when indicated, are essential to improve the prognosis. (1,2)

Non-contrast computed tomography (CT) of the head is the first-line imaging study for diagnosing epidural hematoma, identifying associated intracranial lesions, and assessing their main radiological characteristics, such as hematoma volume, midline shift, ventricular compression, and signs of cerebral edema. It also allows for the evaluation of postoperative progress and the identification of complications, such as rebleeding, persistent blood collections, and the progression of parenchymal changes. However, although it provides essential anatomical information, CT represents a single point in time and does not allow for continuous direct measurement of intracranial pressure or, on its own, the determination of the dynamic state of cerebral hemodynamics. Furthermore, transferring critically ill patients to the imaging department can involve logistical and clinical risks, especially in those with hemodynamic or neurological instability. (1,2, 3, 4)

In this context, non-invasive multimodal neuromonitoring has gained relevance as a complementary strategy for the evaluation of patients with acute brain injury. The integration of clinical findings, tomographic parameters, and bedside ultrasound techniques allows for obtaining complementary information on anatomical changes, the possible elevation of intracranial pressure (ICP), and the condition of cerebral circulation. Although these tools do not replace invasive intracranial pressure monitoring when indicated, they can contribute to evolutionary surveillance, early identification of pathophysiological changes, and individualized decision-making, particularly when used serially and interpreted in conjunction with the neurological examination and radiological findings. (2, 3, 5, 7)

Among the available techniques, optic nerve sheath diameter (OND) ultrasound is based on the anatomical continuity of the subarachnoid space surrounding the optic nerve with the intracranial compartment. Changes in cerebrospinal fluid pressure can be transmitted to this sheath and produce modifications in its diameter, allowing its ultrasound measurement to be used as an indirect marker of possible intracranial pressure (ICP) elevation. It is a rapid, reproducible, and bedside technique that allows for serial assessments without exposure to ionizing radiation. However, its accuracy depends on the measurement technique, the operator's experience, the cutoff points used, and the patient's clinical characteristics; therefore, its results should be interpreted as part of a multimodal evaluation and not as a direct measurement of intracranial pressure. (4, 5, 6, 8)

Furthermore, transcranial Doppler (TCD) allows for the non-invasive assessment of blood flow velocities in the main intracranial arteries and the analysis of hemodynamic parameters that can be altered by changes in vascular resistance, cerebral autoregulation, and perfusion pressure. Serial assessment of systolic, diastolic, and mean velocities, along with the pulsatility index, can provide complementary information on the evolution of cerebral circulation in patients with traumatic brain injury (TBI). However, these parameters are influenced by factors such as blood pressure, arterial partial pressure of carbon dioxide, blood volume, and the characteristics of the brain injury, and therefore should not be interpreted in isolation as definitive estimates of intracranial pressure (ICP). (3, 6, 9)

The combined use of head CT, ultrasound measurement of the anterior chamber depth (ACD), and transcranial Doppler (TCD) offers a complementary approach to monitoring neurocritically ill patients by integrating structural information, an indirect marker of possible intracranial hypertension, and cerebral hemodynamic parameters. This strategy is particularly relevant during the postoperative period following epidural hematomas, when it is necessary to monitor the evolution of cerebral edema, recognize possible signs of deterioration, and guide therapeutic decisions. Serial evaluation of these variables can facilitate the identification of changes over time and contribute to individualized management; however, the evidence supporting each technique is heterogeneous, and its usefulness depends on the

quality of the measurements, the combined interpretation of the results, and the correlation with clinical and radiological evolution. (2–6)

This article presents the case of a patient with a right temporoparietal epidural hematoma who required neurosurgical intervention for hematoma evacuation. The patient underwent follow-up monitoring of cerebral edema using a non-invasive neuromonitoring strategy based on CT findings, ultrasound measurement of the anterior superior ventricular distance (ASD), and transcranial Doppler (TCD). The clinical application of these complementary tools during the postoperative period and their usefulness in neurological surveillance and monitoring of cerebral evolution are described, within the context of successful clinical management. The aim is to highlight the potential of non-invasive multimodal neuromonitoring as a complement to clinical and radiological evaluation in neurocritically ill patients, particularly in settings where the availability of invasive intracranial pressure (ICP) monitoring may be limited.

1.1 Clinical case presentation

A 16-year-old male patient, identified by the initials JJHC, from an urban area, with no relevant past medical or non-medical history, was admitted with a history of severe traumatic brain injury secondary to a fall from a height of approximately 10 meters while working in a construction area, impacting a hard surface. Following the injury, he experienced a sudden loss of consciousness, associated with otorrhagia. He was initially taken by his family to a primary care unit, where significant neurological deterioration was documented, with no response to stimuli and a Glasgow Coma Scale (GCS) score of 7. Due to his altered mental status, endotracheal intubation was performed and invasive mechanical ventilation was initiated. Subsequently, he was transferred to a more specialized hospital unit with a computed tomography scanner, neurosurgery services, and an intensive care unit.

Upon arrival at the secondary care unit, a non-contrast computed tomography scan of the head was performed, which revealed a right temporoparietal epidural hematoma with an estimated volume of 200 mL, associated with vasogenic cerebral edema, compression of the lateral ventricles, and a 6 mm midline shift. A small left intraparenchymal hemorrhagic lesion was also identified. These radiological findings, along with the initial neurological deterioration, indicated a traumatic intracranial injury with a significant mass effect and a risk of progression of the secondary brain injury.

neuromonitoring was implemented using optic nerve sheath diameter ultrasound and transcranial Doppler. The initial optic nerve sheath diameter measurement was 0.62 cm bilaterally, equivalent to 6.2 mm in each eye. From this finding, an intracranial pressure of approximately 26 mmHg was estimated; it should be noted that this figure corresponds to an indirect estimate and not a direct invasive measurement. The initial transcranial Doppler showed a peak systolic velocity of 160 cm/s, a pulsatility index of 1.1, and a resistance index of 0.6. These parameters were recorded as baseline values for comparison with post-intervention assessments.

Initial blood gas analysis revealed hypocapnia, a relevant finding in the context of severe traumatic brain injury due to its potential impact on cerebral blood flow. Ventilatory parameters were adjusted to correct the abnormality and prevent an excessive reduction in arterial partial pressure of carbon dioxide ($PaCO_2$), with the aim of maintaining physiological conditions compatible with neuroprotection.

1.2 Neurosurgical intervention and intensive care treatment

Given the presence of a large epidural hematoma, mass effect, midline shift, and neurological deterioration, urgent surgical evacuation was decided upon. A right temporoparietal craniotomy was performed, with drainage of approximately 180 mL of bloody fluid, without reported intraoperative complications. The difference between the volume estimated by CT scan and the evacuated volume should be interpreted considering that radiological estimation and intraoperative quantification are based on different methods.

In the postoperative period, the patient remained in the intensive care unit under invasive mechanical ventilation, sedation, and analgesia, with measures aimed at controlling cerebral edema, including the administration of 3% hypertonic saline solution. Clinical and hemodynamic monitoring was maintained, along with blood gas analysis and ventilatory adjustments as needed. The therapeutic objective was to prevent secondary brain injury, avoid hypoxemia and significant alterations in $PaCO_2$, and maintain adequate conditions for cerebral perfusion.

Follow-up continued with computed tomography, serial ultrasound of the optic nerve sheath diameter, and transcranial Doppler. The integration of these tools allowed for comparison of anatomical findings with ultrasonographic and hemodynamic parameters during the postoperative period. According to the clinical records provided, transcranial Doppler parameters and measurements related to cerebral edema showed improvement after hematoma evacuation and the implementation of anti-edema measures. Likewise, the initial hypocapnia improved after adjustment of the mechanical ventilator settings.

1.3 Clinical evolution and outcome

Forty-eight hours after surgery, sedation and analgesia were tapered to facilitate neurological evaluation. During this period, favorable clinical progress and improvement in brain monitoring parameters were documented. A follow-up computed tomography scan showed no new signs of intracranial hemorrhage, according to available information. These findings, along with the clinical and respiratory progress, allowed for the weaning of the patient from mechanical ventilation.

The patient was successfully extubated and subsequently transferred to the operating room for continued clinical monitoring, rehabilitation, and multidisciplinary follow-up, including assessment by physiotherapists and other specialists as needed. A detailed neurological score after extubation and a functional assessment at hospital discharge are not available in the provided data.

This case describes the application of a non-invasive, multimodal neuromonitoring strategy in an adolescent patient with severe traumatic brain injury, a large-volume right temporoparietal epidural hematoma, and mass effect. The integration of computed tomography, ultrasound measurement of the optic nerve sheath diameter, and transcranial Doppler allowed for complementary follow-up during the perioperative period, in conjunction with the observed clinical improvement and the discontinuation of mechanical ventilation. Although the temporal evolution is consistent with a favorable response to surgical and medical treatment, the independent contribution of each neuromonitoring technique cannot be established from a single case.

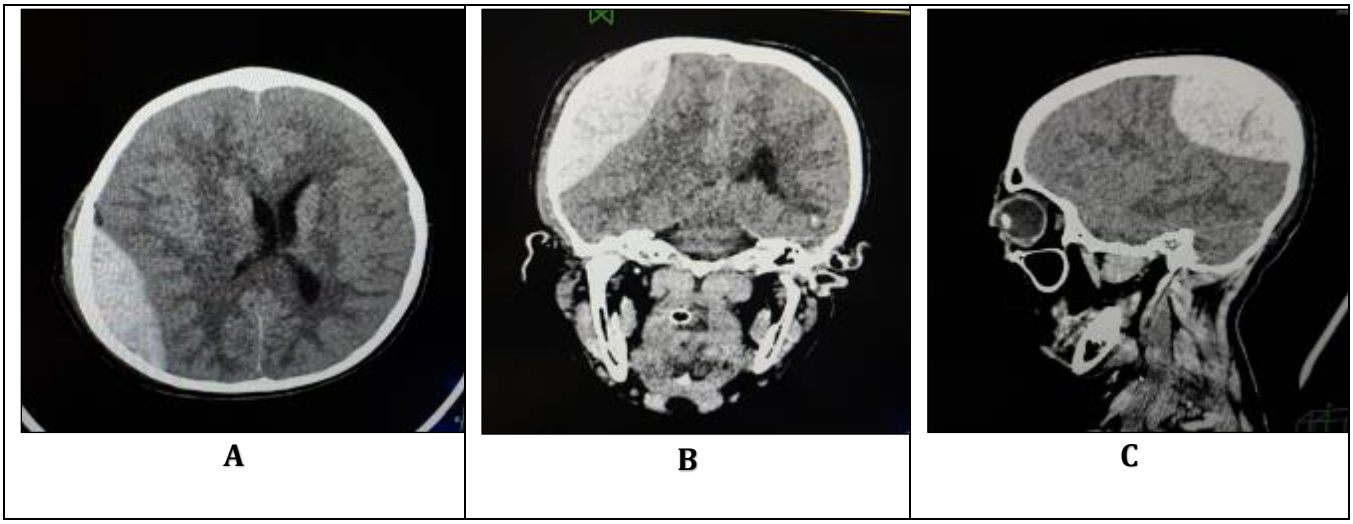

Figure 1: A, B and C; Severe traumatic brain injury with a right temporoparietal epidural hematoma, shown across different tomographic planes

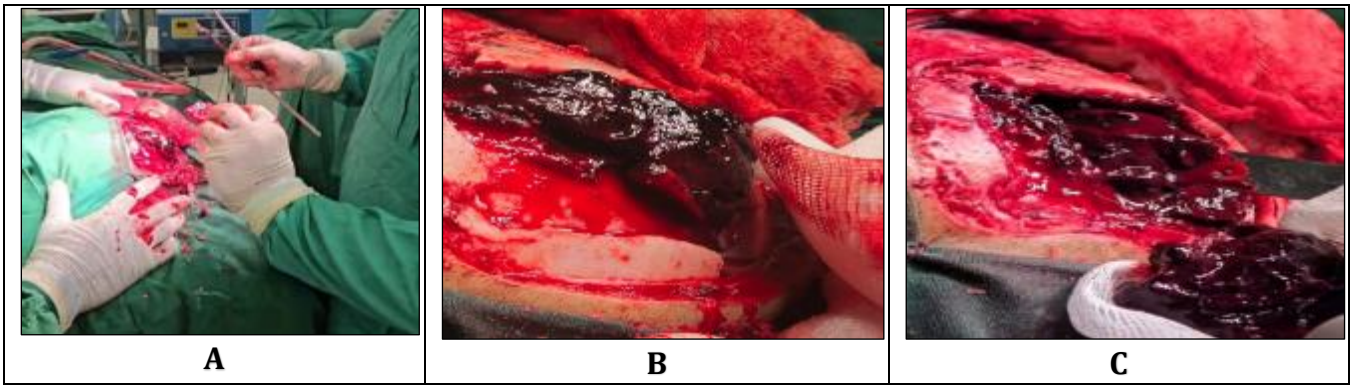

Figure 2: A, B and C; Right temporoparietal craniotomy plus drainage of an epidural hematoma

Figure 3: A, B and C; Non-invasive monitoring of intracranial pressure: Figure A, middle cerebral artery isualized via transcranial Doppler; Figure B, elevated pulsatility index of the middle cerebral artery; Figure C, enlarged optic nerve sheath, suggesting increased intracranial pressure.

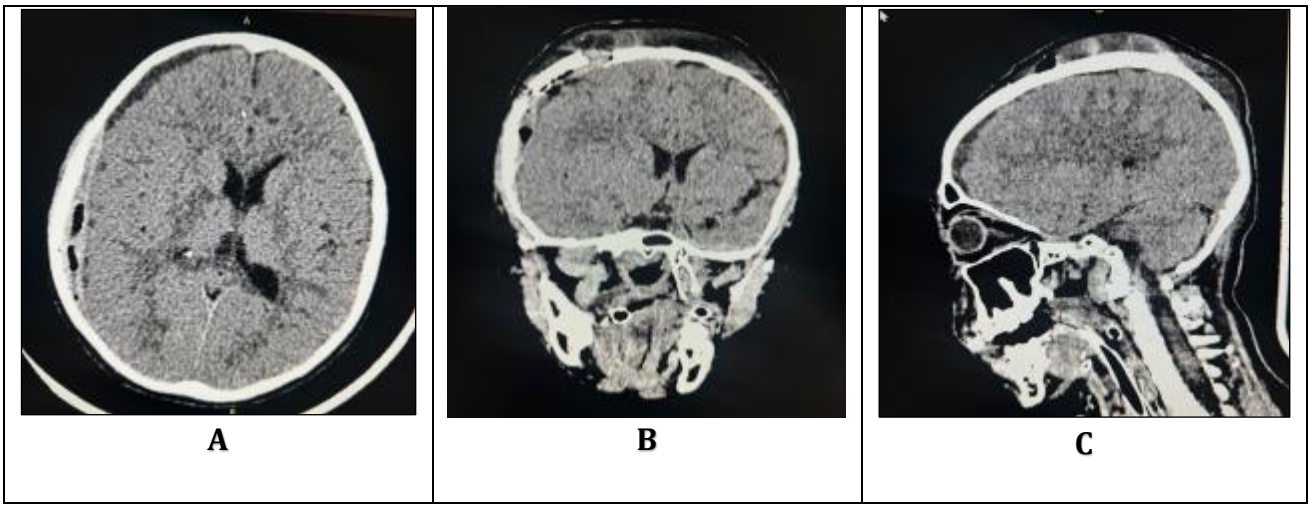

Figure 4: A, B and C; Brain CT scan in different planes, 48 hours after drainage of an epidural hematoma

Figure 5: A, B and C; Non-invasive monitoring using transcranial Doppler 48 hours after epidural hematoma drainage, showing improvement in the optic nerve sheath and the pulsatility index of the middle cerebral artery.

2 DISCUSSION

Severe traumatic brain injury (TBI) is a major cause of morbidity and mortality in adolescents, especially when accompanied by expansive intracranial lesions that produce a mass effect and a risk of intracranial hypertension. In this

case, a 16-year-old male patient suffered a fall from a height of approximately 10 meters, with loss of consciousness and otorrhagia. The initial assessment showed a Glasgow Coma Scale score of 7, requiring orotracheal intubation and mechanical ventilation. Computed tomography (CT) revealed a right temporoparietal epidural hematoma, cerebral edema, ventricular compression, a 6 mm midline shift, and a small left intraparenchymal hemorrhage.

Acute epidural hematomas with mass effect constitute a neurosurgical emergency due to the risk of neurological deterioration and brain herniation. In this patient, a right temporoparietal craniotomy was performed, with evacuation of approximately 180 mL of hematoma, supplemented with 3% hypertonic saline solution, neuroprotective measures, and multimodal monitoring, in accordance with the recommendations for the surgical management of epidural hematomas and severe traumatic brain injury (1,2).

The interest in this case lies in the serial use of optic nerve sheath diameter (OND) and transcranial Doppler (TCD) as complementary tools for monitoring intracranial dynamics. Initially, the OND was 6.2 mm bilaterally, a finding consistent with the possibility of intracranial hypertension in the clinical and radiological context. The initial intracranial pressure estimate of 26 mmHg should be interpreted with caution, since, if derived from the OND, it constitutes an indirect estimate and not an invasive measurement.

During postoperative follow-up, the optic nerve sheath diameter (OND) decreased from 6.2 to 5.5 mm. This reduction represents a favorable trend, consistent with less distension of the optic nerve sheath after hematoma evacuation and treatment of cerebral edema. However, the final value alone does not confirm normalization of intracranial pressure, due to the variability of the cut-off points and the technical limitations of ultrasound measurement (3,4).

The initial CTN showed a peak systolic velocity of 160 cm/s, a pulsatility index (PI) of 1.1, and a resistance index of 0.6. These parameters provide information on cerebral hemodynamics, although they are influenced by factors such as blood pressure, $PaCO_2$, and vascular resistance. During follow-up, normalization of the PI was reported, although the exact postoperative value is not available. The combination of NVA and CTN allows for complementary clinical and radiological evaluation, but it does not replace invasive intracranial pressure monitoring when indicated and available (4, 15).

The initial hypocapnia improved after adjustment of mechanical ventilation. Since $PaCO_2$ modifies cerebral vascular tone and blood flow, its correction was part of the physiological management and facilitated a more accurate interpretation of hemodynamic parameters. Hyperosmolar therapy and ventilatory control were used as adjuncts to surgical evacuation, aimed at controlling edema and preventing secondary brain injury (9, 10, 13).

The patient's progress during the first 48 hours was favorable: the follow-up CT scan showed no new bleeding, a reduction in the DVNO was observed, and the PI was reported to normalize. Sedation and analgesia were also reduced, allowing for extubation and transfer to the surgical ward for follow-up and physiotherapy. The temporal concordance of these findings suggests that non-invasive multimodal neuromonitoring can help identify trends during clinical evolution, although it does not demonstrate causality nor does it, on its own, confirm the resolution of intracranial hypertension. (11, 12, 14)

Limitations include the lack of invasive intracranial pressure monitoring to validate non-invasive parameters, the absence of complete serial TCD values, and the lack of long-term functional neurological follow-up. In conclusion, this case illustrates the potential utility of combining anterior descending ventricle ultrasound and TCD with CT and clinical assessment in the follow-up of severe traumatic brain injury. These tools can complement neurological monitoring, especially in resource-limited settings, provided their results are interpreted in an integrated manner.

3 CONCLUSION

This case highlights the importance of a comprehensive and timely approach to severe traumatic brain injury associated with epidural hematoma and mass effect in an adolescent patient. Surgical evacuation of the hematoma, along with neuroprotective measures, hyperosmolar therapy, and adjustment of mechanical ventilation, constituted a strategy aimed at controlling the primary injury and preventing secondary brain damage.

Serial monitoring of the optic nerve sheath diameter documented a decrease from 6.2 to 5.5 mm during the postoperative period, while transcranial Doppler showed a reported normalization of the pulsatility index. These changes, observed along with a follow-up CT scan showing no new bleeding and extubation at 48 hours, were consistent with a favorable clinical outcome. However, they do not, on their own, confirm the normalization of intracranial pressure.

Correction of hypocapnia through ventilatory adjustment was another relevant component of management, due to the influence of $PaCO_2$ on cerebral blood flow and the interpretation of hemodynamic parameters obtained using transcranial Doppler. This finding underscores the need to integrate blood gas analysis, ventilatory parameters, neuroimaging, and neuromonitoring tools to accurately interpret physiological changes in patients with severe traumatic brain injury.

Finally, this case illustrates the potential of non-invasive multimodal neuromonitoring as a complement to clinical and radiological assessment, particularly in settings with limited access to invasive technologies. However, the lack of direct intracranial pressure measurement and long-term neurological follow-up limits conclusions regarding diagnostic accuracy and functional prognosis. Prospective studies comparing optic nerve sheath diameter trends and transcranial Doppler parameters with invasive measurements and clinical outcomes are needed to better define its utility in neurocritical care practice.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

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Disclosure of conflict of interest

The authors declare that they have no financial, professional, personal, or institutional conflicts of interest that could have influenced the development, interpretation, or publication of this manuscript. No relationships, affiliations, or activities are considered to have compromised the objectivity, integrity, or scientific independence of the work. All authors have reviewed and approved the final version of the manuscript and agree with its submission for publication.

Statement of ethical approval

This case report was prepared in accordance with internationally recognized ethical principles for medical research and scientific publication, including the principles established in the Declaration of Helsinki. Particular attention was given to the protection of the patient's privacy, dignity, and confidentiality throughout the preparation of the manuscript.

All clinical information was handled responsibly, and identifying personal data were omitted or anonymized to prevent the identification of the patient. The clinical information presented is limited to the details considered necessary for understanding the case, its diagnostic approach, clinical evolution, and relevance to medical practice.

Statement of informed consent

The preparation and reporting of this manuscript followed the CARE (CAse REport) guidelines, which promote transparency, accuracy, and completeness in the publication of clinical case reports. The authors acknowledge the importance of respecting patient autonomy and confidentiality and recognize that institutional requirements regarding informed consent and ethical review must be followed according to the applicable regulations. Where required, informed consent for publication was obtained from the patient or the legally authorized representative.

Authors' Contributions

All authors made substantial contributions to the development of this manuscript. Their participation included involvement in the patient's clinical assessment and management, collection and organization of relevant clinical data,

review of diagnostic findings, and analysis of the patient's clinical course. The authors also contributed to the interpretation of the findings in the context of the available scientific literature.

In addition, the authors participated in the literature search, critical review of relevant publications, development of the discussion, and formulation of the manuscript's conclusions. The writing process involved collaboration among the authors to ensure that the clinical information was presented accurately, coherently, and in accordance with the standards of scientific reporting.

All authors critically reviewed the manuscript for important intellectual content, contributed to its revision, and approved the final version before submission. Each author accepts responsibility for their respective contributions and agrees to be accountable for the accuracy and integrity of the work, including the appropriate investigation and resolution of any questions related to its scientific content.

Data Availability Statement

The clinical information supporting this case report is included in the manuscript to the extent necessary to understand the clinical presentation, diagnostic process, management, and outcome. Additional information is not publicly available because of patient confidentiality and privacy considerations. Any request for further clinical information should be evaluated by the corresponding author, subject to applicable ethical requirements and the protection of the patient's identity.

Publication Responsibility

The authors confirm that the manuscript represents an honest and accurate account of the clinical case and that the information has been presented without intentional fabrication, falsification, or inappropriate alteration of clinical findings. The manuscript has been prepared with the objective of contributing to medical knowledge and promoting discussion of the diagnostic and clinical implications of the case.

All authors have approved the submitted version and accept responsibility for the integrity of the work. The authors further confirm that the manuscript will be submitted in accordance with the target.

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