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Acute toxic encephalopathy induced by organic solvent exposure: a case report of diagnostic challenges and occupational health implications

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Abstract

Background Toxic encephalopathy, which results from exposure to neurotoxic substances, poses a considerable clinical challenge, especially for occupational groups like painters and construction workers.

Case presentation This case report describes a 35-year-old female painter who experienced acute toxic encephalopathy, presenting with severe headache, nausea, and vomiting. Initially, her condition was misdiagnosed as cervical spondylosis. However, recognizing her occupational exposure to organic solvents was essential for arriving at the correct diagnosis. This case highlights the importance of thorough evaluations, particularly detailed occupational histories, to avoid misdiagnosis and ensure timely management of such conditions. Neuroimaging results showed bilateral symmetric white matter changes, which supported the diagnosis of organic solvent toxicity. This finding aligns with existing literature that discusses how lipid solubility can disrupt neuronal function. The patient's positive outcome following prompt treatment underscores the critical need for early recognition of toxic encephalopathy, as it can significantly improve recovery chances.

Conclusion This case enhances our understanding of the complexities associated with diagnosing acute encephalopathy caused by organic solvents and underscores the urgent need for healthcare providers to be more aware of occupational hazards.

Keywords Acute toxic encephalopathy, Neurotoxic substance exposure, Organic solvent exposure

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Background

Toxic encephalopathy is a syndrome that occurs due to exposure to neurotoxic substances and presents a range of neurological symptoms such as headaches, cognitive impairments, and changes in consciousness. This condition can develop from both acute and chronic exposures, especially in work environments where organic solvents are commonly found [1].

Acute toxic encephalopathy typically presents with a sudden onset of symptoms, including intense headache, nausea, vomiting, and dizziness. If these symptoms are not quickly identified and treated, they can escalate into more serious neurological impairments [2]. However, diagnosing this condition presents considerable challenges because its symptoms often overlap with those of other neurological disorders, including infections and tumors. This overlap can result in misdiagnosis and consequently delay appropriate treatment [3]. Initial diagnostic evaluations, such as neuroimaging and laboratory tests, may not always provide clear results, which makes it essential to conduct a comprehensive occupational history to identify any possible toxic exposures [4].

This case report presents a distinctive instance of toxic encephalopathy in a female painter who suddenly developed severe symptoms. Her significant exposure to organic solvents in her occupation played a crucial role in pinpointing the root cause of her condition. This case underscores the essential importance for healthcare providers to take occupational factors into account when assessing unexplained neurological symptoms, highlighting the need for a thorough evaluation of a patient's work environment and potential exposures [5].

Case presentation

Initial presentation

The patient was a 35-year-old female who presented with a primary complaint of a headache accompanied by vomiting that had lasted for 10 days. She reported a sudden onset of headache, mainly located in the occipital region, along with symptoms of dizziness, generalized weakness, nausea, and projectile vomiting of gastric contents. Importantly, she denied any presence of blood in her vomit, visual disturbances, loss of consciousness, or seizures; however, she did mention experiencing chest tightness and difficulty breathing. There were no indications of fever, cough, sputum production, palpitations, chest pain, abdominal pain, distension, or diarrhea.

Initial evaluation

Initially, she sought medical attention at one hospital, where a cervical MRI revealed "cervical degenerative changes." Unfortunately, fluid therapy did not relieve her symptoms, prompting a transfer to another hospital. A cranial CT scan conducted there showed "cerebral edema," and a lumbar puncture indicated elevated cerebrospinal fluid (CSF) pressure of 250 mmH_2O, with the CSF appearing clear and colorless. The Pandy's test returned negative, and the white blood cell count was low at $1 \times 10\text{^}6$/L. CSF cultures were negative, and hematological tests showed no autoimmune antibodies. The patient was treated with mannitol and dexamethasone to address dehydration and reduce intracranial pressure; however, she continued to suffer from headaches, nausea, and vomiting.

Emergency department findings

Concerned about her deteriorating condition, her family brought her to our emergency department, where she received treatment aimed at reducing intracranial pressure, preventing thrombosis, and supplementing electrolytes. Despite these interventions, her headache persisted.

Upon physical examination, the patient exhibited a temperature of 36.7 °C, a pulse rate of 58 beats per minute, a respiration rate of 18 breaths per minute, and blood pressure measured at 126/88 mmHg. Although she was alert, she displayed signs of lethargy, as evidenced by her spontaneous eye opening, appropriate responses to questions, and ability to follow commands. Her pupils were equal and reactive to light, measuring approximately 2.0 mm in diameter, and there was no icterus noted in the sclerae. The examination of her spine and limbs revealed no deformities, and her lower extremities were free of edema. Muscle strength was assessed at a rating of 4, with normal muscle tone observed. A positive Babinski sign was noted, while other pathological reflexes were absent, and there were no signs of meningeal irritation.

Laboratory tests showed a white blood cell count of $10.08 \times 10\text{^}9$/L, with elevated monocytes at $0.85 \times 10\text{^}9$/L and increased neutrophils at $7.56 \times 10\text{^}9$/L. The hemoglobin level was recorded at 124 g/L, and the platelet count was $209 \times 10\text{^}9$/L. C-reactive protein levels were low, measuring less than 0.2 mg/L. Biochemical tests revealed elevated glucose levels at 6.0 mmol/L, low sodium levels at 133 mmol/L, and increased liver enzymes, with alanine aminotransferase at 175 U/L, aspartate aminotransferase at 106 U/L, and gamma-glutamyl transferase at 76 U/L. Imaging studies indicated bilateral symmetric abnormal signals in the white matter of the cerebral hemispheres and the dentate nucleus, which suggest a possible diagnosis of solvent-related toxic encephalopathy (Fig. 1).

Management

The treatment protocol included thorough diagnostic evaluations and management of symptoms, focusing on dehydration and reducing intracranial pressure. Key interventions involved providing analgesia, nutritional support, hepatoprotective measures, preventing stress

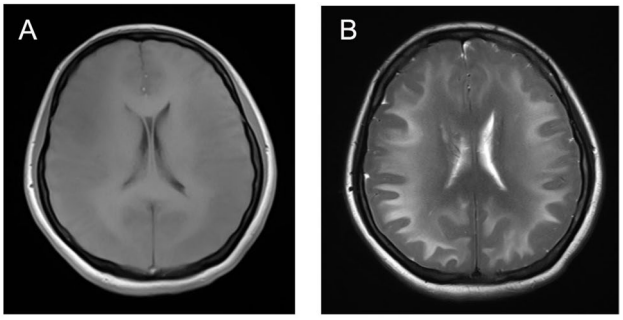

Fig. 1 Cranial MRI examination results. Bilateral white matter abnormalities with symmetrical signal changes: low signal intensity on T1-weighted images (**A**), high signal intensity on T2-weighted images (**B**), presenting as "flame-like" lesions

ulcers, and administering vitamin supplements such as vitamin C and B complex, while also ensuring electrolyte balance. The patient received consultations from both neurology and neurosurgery specialists. Treatment strategies included the use of mannitol to lower intracranial pressure, pain management with fentanyl, nutritional support through nerve growth factor, and corticosteroids to reduce neuroinflammation. Due to her poor appetite and nausea, famotidine was given to prevent stress-related mucosal disease. Considering her liver dysfunction, polyene phosphatidylcholine was prescribed for liver protection. Additionally, the patient was assessed for thyroid function, underwent EEG and evoked potentials, and had toxicology tests performed to exclude vascular conditions. After aggressive treatment, the patient's headaches showed gradual improvement. A lumbar puncture during her hospital stay revealed CSF pressure ranging from 280 to 370 mmH_2O. Hyperbaric oxygen therapy was then initiated, resulting in significant symptom relief. Following this, the patient was transferred to a rehabilitation facility to continue with hyperbaric oxygen therapy.

Outcome

The patient was monitored for six months after discharge and remained free of any headache symptoms during this period. Additionally, liver function tests showed a return to normal levels, and there were no reported neurological complications.

Discussion

The case presented illustrates the challenges involved in diagnosing acute organic solvent-induced encephalopathy, especially in relation to occupational exposure. There have been similar instances where misdiagnosis happened initially due to the overlapping symptoms with other neurological disorders. Infectious encephalitis typically presents with symptoms including fever, headache, altered consciousness, and seizures, with rapid disease progression that may lead to serious consequences [6], and clinical manifestations may vary depending on different pathogens [7].Metabolic encephalopathy is a cerebral dysfunction caused by various metabolic disorders, commonly seen in patients with liver disease, kidney disease, diabetes, and other conditions. Its clinical manifestations include disorders of consciousness, behavioral changes, motor disorders, and other symptoms that may rapidly worsen [8]. The diagnosis of metabolic encephalopathy relies on laboratory test results, such as blood glucose levels, hepatic and renal function indicators, and electrolyte levels. Timely metabolic correction can significantly improve patient prognosis [9].Demyelinating diseases are a group of neurological disorders characterized by myelin sheath damage, including multiple sclerosis and acute disseminated encephalomyelitis. Their clinical manifestations vary widely, with common symptoms such as blurred vision, limb weakness, and sensory abnormalities, and the disease course may exhibit fluctuations. The differential diagnosis of demyelinating diseases requires integration of the patient's medical history, clinical presentation, and imaging findings, particularly MRI which can reveal characteristic demyelinating lesions [10].

The injury mechanism linked to organic solvents is primarily due to their lipid solubility, which interferes with neuronal function and results in widespread damage to white matter [11].Organic solvents cross the blood-brain barrier and enter the central nervous system. The accumulation of lipophilic substances not only damages neurons but also affects the function of glial cells, which in turn leads to neuronal death and dysfunction [12]. Furthermore, oxidative stress and the release of inflammatory factors are considered important factors leading to white matter damage. These factors promote apoptosis and neuroinflammation, causing further neuronal injury [13]. Other studies have also found that white matter damage is closely related to iron deposition, decreased antioxidant capacity, and lipid peroxidation [14]. Since white matter plays a crucial role in neural signal transmission, its damage may trigger a series of neurological disorders that affect patients' cognitive and motor abilities.

MRI, as a key imaging modality, demonstrates significant efficacy in the diagnosis of organic solvent encephalopathy, enabling physicians to rapidly identify brain injury in emergency settings. Neuroimaging findings, particularly bilateral symmetric white matter changes, play a crucial role in confirming the diagnosis of organic solvent toxicity, as highlighted in comparable studies [5]. Research has shown that inhalation or skin contact with 1,2-DCE can cause neurotoxic damage to the nervous system. This solvent crosses the blood-brain barrier and enters the central nervous system, leading to edema of white matter and symmetric abnormalities in signal

intensity, particularly in bilateral cerebral white matter, basal ganglia, and cerebellar dentate nucleus [15]. MRI can clearly display features associated with infections, tumors, or other neurological diseases, providing clinicians with strong diagnostic evidence [16, 17]. By comparing imaging characteristics, doctors can more accurately diagnose the patient's underlying cause and formulate appropriate treatment strategies.

The diagnosis of organic solvent poisoning can also be confirmed through the detection of specific biomarkers and solvent metabolites. Research indicates that certain metabolites such as hippuric acid can serve as biomarkers for organic solvent poisoning, assisting physicians in rapid identification of intoxication during emergency care [18]. Additionally, abnormal liver function can also serve as a critical early warning sign of neurological damage. Research indicates a direct correlation between hepatic insufficiency and encephalopathy, particularly when liver enzymes are elevated. Under such conditions, the metabolic functions of brain cells and neural conduction are seriously compromised [19].

The diagnostic challenges associated with acute organic solvent-induced encephalopathy are significant, primarily because its clinical manifestations are often nonspecific. Initial misdiagnosis frequently occurs when symptoms like headache and vomiting are mistakenly attributed to more prevalent neurological conditions. This misattribution can result in delayed treatment and poorer outcomes for patients. Diagnosing this condition presents considerable challenges because its symptoms often overlap with those of other neurological disorders, including infections and tumors. This is a common issue with rare neurological conditions, as illustrated by cases like hemangioblastoma, where nonspecific symptoms like chronic headache and diplopia can lead to significant diagnostic delays and adverse outcomes [20].Therefore, it is crucial to obtain a thorough occupational history during the diagnostic process, particularly for patients presenting with acute neurological symptoms. The symptoms observed in this case correspond with existing literature that highlights the nonspecific characteristics of acute toxic encephalopathy. This alignment underscores the importance of increased clinical awareness among healthcare providers [21]. Moreover, the positive outcome seen in the patient following treatment reinforces the idea that early detection and intervention can greatly enhance results, a perspective that is supported by current literature on the management of toxic encephalopathy [22].

This case enhances our understanding of the health risks associated with various occupations and underscores the urgent need for preventive measures to reduce exposure to harmful substances in industrial environments. The implementation of Personal Protective Equipment (PPE) is a crucial measure in preventing organic solvent poisoning. Effective personal protection includes wearing appropriate protective gear such as masks, gloves, and goggles to reduce inhalation and skin contact with harmful substances [23].Additionally, regular neurocognitive monitoring of workers, industrial ventilation, and exposure limit controls can also help minimize the occurrence of poisoning incidents [24, 25].

Compared with existing reports of organic solvent-induced encephalopathy, the unique aspect of this case lies in the diagnostic challenge. In this case, the patient's severe headache and vomiting were initially misattributed to cervical spondylosis, highlighting the critical need to consider a wide range of differential diagnoses in young patients presenting with acute neurological symptoms [26]. After 10 days of ineffective treatment, the patient was transferred to our hospital. Upon further inquiry into the patient's occupational history, it was discovered that the patient had worked in a spray paint factory for two and a half months prior to disease onset, with inadequate protective measures. Intoxication could not be ruled out. Subsequent cranial MRI showed extensive white matter damage. Combined with the patient's occupational history, medical history, and examination results, this case was considered "acute organic solvent-induced encephalopathy." The lesson learned is that cranial MRI was not completed in a timely manner in this patient; moreover, the emergency physician failed to inquire about the patient's occupational history and medication use, and did not consider the possibility of intoxication. This reminds us that when emergency physicians encounter patients with similar symptoms, they must inquire in detail about occupational history, history of toxic exposure, and clinical manifestations to facilitate differential diagnosis [27].

Conclusion

This case underscores the critical need for timely MRI evaluations in patients experiencing acute onset headaches and highlights the importance of investigating occupational histories and potential toxic exposures. In this instance, the patient's employment at a paint factory played a pivotal role in diagnosing acute organic solvent-induced toxic encephalopathy. Managing such cases requires a multidisciplinary approach and an awareness of possible diagnostic challenges, such as overlooking occupational exposure history and delaying necessary imaging studies.

In conclusion, this case report emphasizes the importance of being vigilant in identifying toxic encephalopathy, especially in younger patients with no significant medical histories. It serves as a reminder for healthcare providers to conduct thorough evaluations, including detailed inquiries into occupational backgrounds, to

ensure prompt diagnosis and effective management of toxic exposures. Such awareness can ultimately improve outcomes and inform prevention strategies within occupational health settings.

Author contributions
H.and Y.: Reviewed the literature and drafted the case report; R.and L.:Responsible for the patient overall supervision; W.: Prepared figures 1. All authors reviewed the manuscript.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

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Not applicable.

Consent for publication
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Competing interests
The authors declare no competing interests.

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