Subacute or chronic neurological toxicity following acute diquat poisoning: a case report

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Case presentation

In January 2025, a 40-year-old male with no significant medical history ingested 200 mL of diquat (DQ) and required urgent medical intervention. He presented with dizziness, fatigue, and throat discomfort. Treatment at a local centre in China included gastric lavage, cathartics, fluid resuscitation, and alternating haemoperfusion (4-hour sessions) and continuous renal replacement therapy (20-hour cycles). Laboratory tests revealed a serum creatinine level of 187 μmol/L and elevated DQ concentrations in both blood (122 ng/mL) and urine (2535 ng/mL).

Three days post-ingestion, the patient was transferred to our emergency department with ongoing throat discomfort. Subsequent laboratory tests revealed a further increase in DQ levels (205.66 ng/mL in blood and 4278.52 ng/mL in urine), while paraquat (PQ) levels were undetectable. A computed tomography scan of the brain (Fig 1a) showed no abnormalities. Due to the lack of a specific antidote for DQ toxicity, management prioritised enhanced elimination through sustained haemoperfusion-continuous renal replacement therapy cycling (a 7-day course until blood DQ

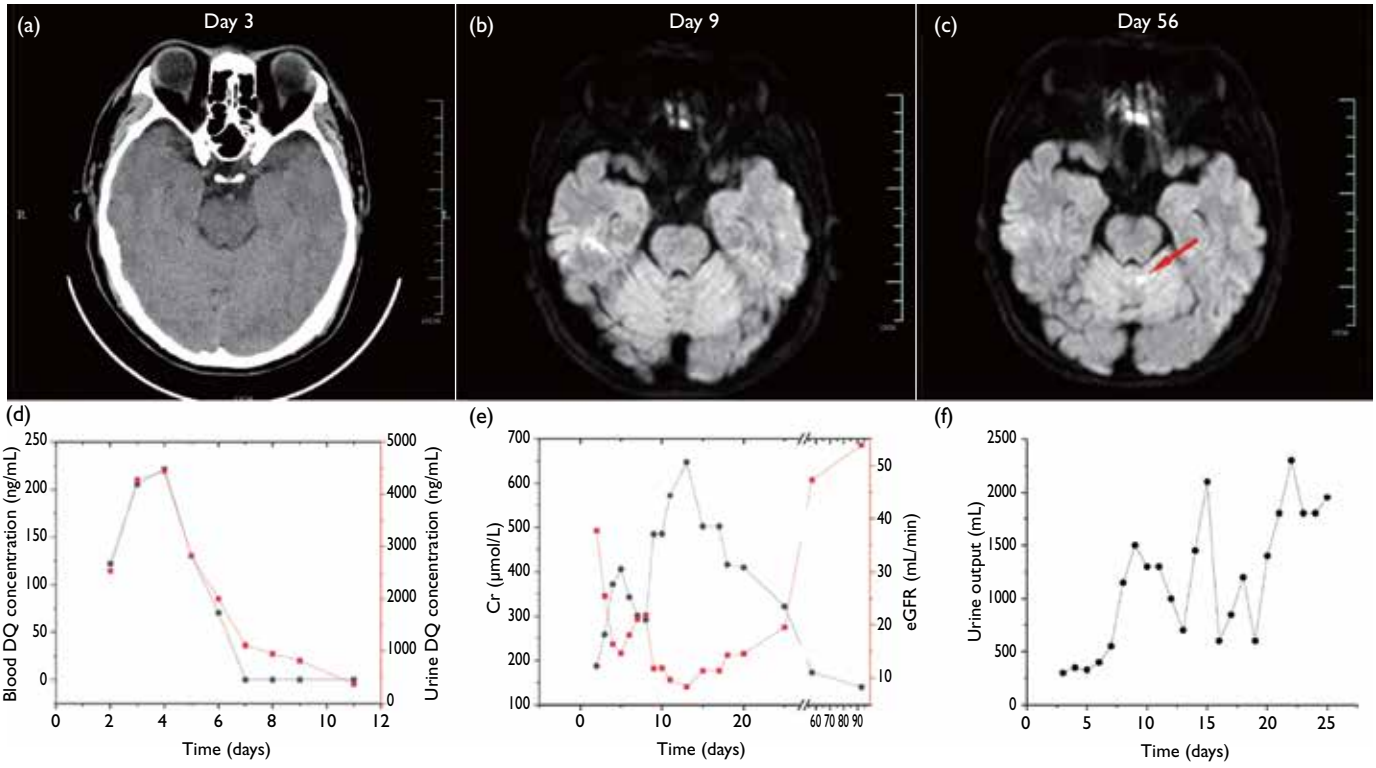

FIG 1. (a) Brain computed tomography showing no abnormalities upon initial assessment. (b) Magnetic resonance imaging (MRI) of the brain during hospitalisation revealing no pathological changes. (c) Brain MRI on readmission demonstrating punctate hyperintense foci in the cerebellar region on diffusion-weighted imaging (red arrow). (d) Variations in diquat (DQ) concentrations in blood and urine, (e) changes in renal function, and (f) alterations in urine output over the course of treatment

Abbreviations: Cr = creatinine; eGFR = estimated glomerular filtration rate

was undetectable), along with corticosteroids (methylprednisolone 80 mg intravenously every 8 hours, tapered gradually over a 4-week period) and antioxidant therapy (intravenous reduced glutathione 1.2 g once daily until discharge). Lumbar puncture on days 4 and 16 indicated elevated intracranial pressure (230-250 mm H_2O), with an initial cerebrospinal fluid (CSF) DQ level of 111.06 ng/mL, which later became undetectable. Mannitol and a glycerol-fructose compound were administered as dehydrating agents to manage intracranial pressure, although follow-up magnetic resonance imaging (MRI) of the brain showed no pathological changes (Fig 1b). Following comprehensive treatment, the patient was discharged in a stable condition.

On day 57, the patient returned with exacerbated headaches and intermittent blurred vision that had developed over the preceding week. Brain MRI revealed cerebellar hyperintensities (Fig 1c). Neurophysiological assessment demonstrated a marked reduction in the right-sided P40-N50 amplitude of somatosensory evoked potentials (0.86 μV vs 2.35 μV contralaterally, 63.4% interhemispheric asymmetry), bilateral prolongation of visual evoked potentials latencies (122 ms right vs 126 ms left, 3.3% latency disparity), and left-lateralised attenuation of wave I/III/V complex amplitudes in auditory evoked potentials, indicative of multisensory pathway dysfunction. The patient commenced a 1-month course of oral methylprednisolone (4 mg daily) combined with long-term neurotrophic support, including vitamin B complex (1 tablet 3 times daily) and mecobalamin (500 μg 3 times daily). By day 93 following exposure, he reported less intense headaches, although intermittent blurred vision persisted, alongside new symptoms of anosmia and ageusia. Follow-up assessments demonstrated partial improvement, including somatosensory evoked potential right-sided N50 amplitude recovery (right: 2.24 μV; left: 3.29 μV) and reduced right-sided visual evoked potential latency (right: 114 ms; left: 128 ms). The patient was advised to continue oral neurotrophic therapy (vitamin B complex and mecobalamin), with ongoing follow-up to monitor clinical recovery. Changes in DQ levels in blood and urine are illustrated in Figure 1d, while alterations in renal function and urine output are depicted in Figure 1e and f, respectively. The patient's clinical progression is summarised in Fig 2.

Discussion

Diquat is an organic herbicide characterised by a heterocyclic structure and has increasingly replaced PQ following the latter's ban in China in 2016.[1] This regulatory shift has correlated with a rise in DQ poisoning incidents. Although the toxicological mechanisms of DQ are still being elucidated, current hypotheses suggest that its harmful effects may mimic those of PQ, while also producing distinct organ-specific consequences. Although DQ is systemically distributed, it primarily targets

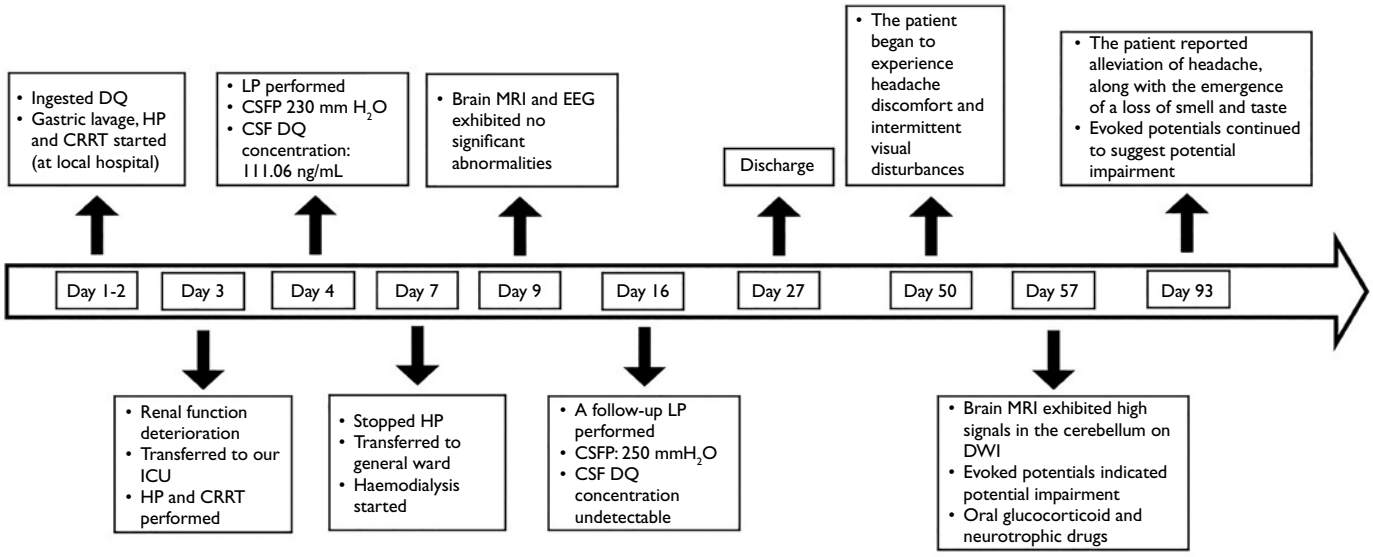

FIG 2. Clinical progression of the patient

Abbreviations: Cr = creatinine; CRRT = continuous renal replacement therapy; CSF = cerebrospinal fluid; CSFP = cerebrospinal fluid pressure; DQ = diquat; DWI = diffusion-weighted imaging; EEG = electroencephalography; eGFR = estimated glomerular filtration rate; HP = haemoperfusion; ICU = intensive care unit; LP = lumbar puncture; MRI = magnetic resonance imaging

the kidneys, with markedly lower concentrations detected in the brain.[2] In previous clinical studies, Yu et al[3] and Zhou and Lu[4] observed that exposure to DQ can adversely affect the nervous system, often presenting as acute toxic encephalopathy. The onset of acute encephalopathy typically occurs 24 to 72 hours post-exposure. Research indicates that the neuroinflammatory response triggered by DQ involves multiple mechanisms, including oxidative stress, mitochondrial dysfunction, and neuronal degeneration.[5] An experimental study[6] demonstrated that neuroinflammation plays a critical role in DQ-induced toxic encephalopathy and is exacerbated by disrupted autophagic processes in microglial cells. While earlier investigations have focused on the immediate and severe clinical manifestations observed in hospital settings, the potential for delayed neurological deficits during long-term follow-up remains underexplored.

In the current case, the patient initially displayed no significant neurological symptoms, and both computed tomography and MRI scans of the brain were unremarkable. Routine biochemical tests revealed an isolated elevation of serum creatinine, with all other parameters within normal ranges. To further assess potential neurotoxicity, CSF analysis from lumbar puncture was performed. Cerebrospinal fluid profiles showed normal routine and biochemical parameters, while serial measurements indicated a rapid decline in DQ concentration. Nevertheless, approximately 50 days post-exposure, the patient began experiencing headaches, intermittent blurred vision, and loss of smell and taste. This case offers important insights into the neurological complications following DQ exposure, highlighting the potential for delayed deficits even when CSF DQ levels fall below detectable thresholds. A limitation of this report is its focus on a single case, which limits the ability to draw broader conclusions or conduct systematic investigations involving larger patient cohorts.

Previous studies[7,8] provides limited guidance on the management of post-acute DQ toxicity, which may contribute to misdiagnosis in patients presenting with neurological or ophthalmic symptoms post-discharge. This report highlights the urgent need for healthcare professionals to recognise that DQ poisoning can result in delayed subacute or chronic neurological complications, emphasising the critical importance of early detection, timely intervention, and longitudinal follow-up to optimise long-term outcomes.

Author contributions

Concept or design: Y Lu.
Acquisition of data: X Jin.
Analysis or interpretation of data: Both authors.
Drafting of the manuscript: X Jin.
Critical revision of the manuscript for important intellectual content: Both authors.

Both authors had full access to the data, contributed to the study, approved the final version for publication, and take responsibility for its accuracy and integrity.

Conflicts of interest

Both authors have disclosed no conflicts of interest.

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Ethics approval

This study was approved by the Clinical Research Ethics Committee of the First Affiliated Hospital, Zhejiang University School of Medicine (Ref No.: 2025B-0359). Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

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