

From coma to consciousness: Effective extracorporeal clearance in severe phenobarbital overdose

Bhayolina Bora¹, Moinul Islam¹, Amrit Jyoti Sonowal², Ananta Kumar Bora²

From ¹Consultant Nephrologist, Department of Nephrology, ²Consultant Critical Care, Department of Critical Care, GNRC Hospital, North Guwahati, Assam, India

ABSTRACT

Phenobarbital remains widely used for epilepsy management in resource-limited settings, but severe overdose can lead to prolonged coma and life-threatening complications. We report a 22-year-old female who presented with deep coma and respiratory failure following intentional ingestion of approximately 100 phenobarbital tablets. Despite optimal supportive care, neurological recovery was delayed with persistently elevated serum drug levels. Two consecutive sessions of intermittent hemodialysis were performed, resulting in rapid clinical improvement and a significant reduction in serum phenobarbital concentration. The patient achieved complete neurological recovery without sequelae. This case highlights the effectiveness of hemodialysis as an accessible extracorporeal modality for enhanced drug elimination in severe phenobarbital poisoning and emphasizes the importance of early identification of candidates for such therapy.

Key words: Barbiturate poisoning, Drug overdose, Extracorporeal treatment, Hemodialysis, Phenobarbital toxicity

Phenobarbital is a long-acting barbiturate that exerts its pharmacologic effect through potentiation of gamma-aminobutyric acid (GABA)-mediated inhibitory neurotransmission. Despite declining use in developed nations, it remains an essential antiepileptic drug in many low- and middle-income countries due to its cost-effectiveness and availability [1]. Toxic ingestion results in dose-dependent depression of the central nervous system, ranging from drowsiness to deep coma. Severe poisoning may be complicated by respiratory failure, hypotension, hypothermia, and aspiration pneumonia [2]. Phenobarbital possesses pharmacokinetic properties that make extracorporeal removal feasible: low-molecular weight (232 Da), moderate protein binding (20–45%), limited volume of distribution, and prolonged elimination half-life in overdose settings [3]. Current toxicology recommendations support extracorporeal treatment in severe cases characterized by prolonged coma, need for mechanical ventilation, or markedly elevated serum concentrations [4].

We describe a case of severe phenobarbital poisoning successfully managed with two sessions of intermittent hemodialysis. Despite existing recommendations for extracorporeal therapy in severe barbiturate poisoning, its timely utilization remains inconsistent in clinical practice,

particularly in resource-limited settings. Reporting such cases is important to reinforce awareness among clinicians regarding early recognition and appropriate use of hemodialysis in severe phenobarbital toxicity, which can significantly improve patient outcomes.

CASE PRESENTATION

A 22-year-old female who was a known case of seizure disorder controlled on phenobarbital at therapeutic doses got admitted to the emergency department with a history of intentional ingestion of approximately 100 phenobarbital tablets (estimated total dose ~10 g, 100 mg/tablet). The estimated dose exceeded therapeutic limits substantially.

On admission, the patient presented with deep coma (markedly reduced Glasgow Coma scale = 3), depressed respiratory effort, hypotension requiring fluid resuscitation, reactive pupils, and no focal neurological deficit.

The patient required airway protection and was transferred to intensive care for ventilatory support. Laboratory Investigations showed markedly elevated serum phenobarbital concentration (131 µg/mL, normal level 10–30 µg/mL), mild metabolic acidosis, normal renal function, and electrolytes within acceptable limits (Table 1). Supportive care was

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Correspondence to: Moinul Islam, Vill & P.O- Japia, P.S- Hajo, Dist-Kamrup (R), North Guwahati - 781382, Assam, India.
E-mail: moinul.jnv cotton@gmail.com

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Table 1: Laboratory parameters of the patient

Laboratory parameters	February 2, 2026	February 26, 2026	February 27, 2026	February 28, 2026
Total leukocyte count (/uL)	6700	7200	-	6480
Differential leukocyte count	N62, L27, E1, M10	N78, L15, E1, M6	-	N82, L6, E1, M11
Hemoglobin (g/dL)	10.6	10.4	-	10.2
Platelet count (/uL)	1.7 lac	1.6 lac	-	1.84 lac
Creatinine (mg/dL)	0.6	0.5	0.4	0.5
Urea (mg/dL)	17	15	6	12
Na (mmol/L)	142	141	145	141
K (meq/L)	3.6	3.0	3.2	3.6
Ca (mg/dL)	7.6	-	-	-
Mg (mg/dL)	2.0	-	-	-
Aspartate aminotransferase/alanine aminotransferase/alkaline phosphatase (U/L)	28/20/90	-	-	-
C-reactive protein (mg/dL)	0.47	-	-	1.2
Serum phenobarbital level (µg/mL)	131	40	24	-
Computed tomography scan of the brain	No significant abnormality seen			

instituted promptly, including endotracheal intubation and mechanical ventilation, intravenous crystalloids, continuous hemodynamic monitoring, activated charcoal (administered early), and consideration of urinary alkalinization.

Despite optimal supportive therapy, the patient remained deeply comatose with persistently elevated serum phenobarbital levels after 24 h. Given the severity of neurological depression and sustained high drug levels, extracorporeal elimination was initiated. Two sessions of intermittent hemodialysis were performed on consecutive days using a low-flux dialyzer, blood flow rate: approximately 250–300 mL/min, dialysate flow rate: 500 mL/min, duration: 2.5 h for the first session and 4 h for the second session. After the first hemodialysis session, there was a noticeable improvement in the consciousness level, improved spontaneous respiratory effort, and a significant decline in serum phenobarbital concentration (40 µg/mL). Following the second session of hemodialysis, she gained complete restoration of consciousness, hemodynamic stabilization, and successful weaning from mechanical ventilation and normalization of serum phenobarbital level (24 µg/mL).

The patient demonstrated complete neurological recovery and was discharged in stable condition after psychiatric consultation. Her anticonvulsant has been changed to Lacosamide. She got discharged on the 5th day of admission in a stable condition, and on follow-up, she is doing well.

DISCUSSION

Phenobarbital enhances GABA-mediated chloride channel opening, producing central nervous system depression. In overdose, suppression of brainstem

respiratory centers leads to hypoventilation and hypercapnia [2]. Hypotension may occur due to peripheral vasodilation and myocardial depression. The effectiveness of extracorporeal therapy depends on drug characteristics.

Phenobarbital's relatively low molecular weight and moderate protein binding allow significant dialytic clearance [3]. In overdose, protein binding may decrease, further enhancing dialyzability. Several case reports and series have demonstrated the effectiveness of extracorporeal therapies in severe phenobarbital poisoning. For instance, Ghannoum *et al.* (extracorporeal treatments in poisoning workgroup) systematically reviewed available evidence and strongly recommended hemodialysis in cases of prolonged coma and high serum drug levels [4,5]. Similarly, Palmer *et al.* reported rapid neurological recovery following hemodialysis in severe barbiturate toxicity [6]. Another case by Hoyland *et al.* highlighted a significant reduction in phenobarbital half-life with intermittent hemodialysis compared to conservative management alone. The half-life reduction was from >80 h to <20 h during dialysis in their study [7]. These findings are consistent with our case, where early initiation of dialysis resulted in rapid clinical improvement.

Intermittent hemodialysis provides high clearance rates and substantially reduces the elimination half-life compared to endogenous metabolism alone and shortens the duration of coma [5]. Early consideration of extracorporeal therapy reduces intensive care unit stay, shortens duration of mechanical ventilation, minimizes complications of prolonged coma, and improves resource utilization [8]. In resource-limited settings where phenobarbital use remains common, nephrologists and intensivists must maintain awareness of dialysis indications in severe overdose.

CONCLUSION

This case highlights the critical role of intermittent hemodialysis in severe phenobarbital poisoning. In patients presenting with deep coma and elevated serum concentrations, extracorporeal therapy can dramatically accelerate neurological recovery. Timely intervention requires multidisciplinary coordination and awareness of current toxicology guidelines.

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