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Acute Peritoneal Dialysis in Phenobarbitone Toxicity

Case Report

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Abstract

Barbiturates are rare cause of poisoning and they are said to be cause of 15 per cent of all poisoning cases. It can be intentional or unintentional. Most patients improve by conservative management. But few patients develop progressive deterioration requiring ventilator support. Drug elimination in these patients will save patients life and shorten recovery period. We report a case of 26 yrs. old female with severe phenobarbital poisoning complicated with hypotension, respiratory depression requiring ventilator support and very high inotropic support. She was initiated on alkaline diuresis but had poor response. In view of hemodynamic instability she was initiated on peritoneal dialysis. Patient underwent 50 cycles of peritoneal dialysis over 4 days. Her general condition improved with inotropic support stopped on day 3 and ventilators were removed on day 4. Very few patients of barbiturate poisoning treated with peritoneal dialysis are reported till date.

Keywords: Peritoneal Dialysis; Barbiturate Poisoning; Phenobarbitone

Introduction

Most common cause of poisoning in India is usually caused by household agents (44%), which is followed by drugs (19%) [1]. Barbiturates are considered as important cause of drug related poisoning. Barbiturates class of drug are mostly used for seizure disorder, most cases of poisoning are mildly symptomatic and can be dealt with conservative management but in some cases severity is more with progressive deterioration. Patient may require ventilator and inotropic support. Management of deeply comatose patients is difficult, patient may take prolong time for recovery. Morbidity if not treated is very high and a significant number of deaths are reported despite intensive therapy [2]. Here we report a case in which severe phenobarbital poisoning which was successfully treated by peritoneal dialysis. It is suggested that this technique has a place in the management of severe phenobarbital poisoning.

Case

26yr old female patient known case of seizure disorder was initiated on tab phenobarbitone 60 mg twice a day since last 6 month. On 13/09/2022 patient consumed 15-20 tablets of phenobarbitone (900 – 1200 mg) in morning hours at 8 am to 9am. She was fasting for 2 days before the event. She presented to Government medical college Akola in drowsy state at 5 pm. No history of fever, vomiting, headache, trauma, active convulsions. . No history of DM, HTN and cardiac illness.On examination heart rate 60 beats per min, Blood pressure was not recordable; pupils were dilated and sluggishly reacting to light. Patient was intubated started on ventilator support, iv fluids and noradrenaline infusion up to 0.4 mg / hr. The diagnosis of phenobarbital poisoning was made based on clinical history and patient's presentation. Dose and fasting attributed to toxicity of phenobarbitone. Ryles tube was placed and gastric lavage was done

with normal saline, forced alkaline diuresis with furosemide and iv sodabicarb was given.

Despite all supportive care and alkaline diuresis her condition worsened with increase in requirement of inotropic support noradrenaline 8mg in 50 ml ns @ 15 ml per hour), pupils sluggishly reacting to light. In view of hemodynamic instability she was initiated on peritoneal dialysis for removal of toxins. Under all aseptic precaution straight acute peritoneal dialysis catheter was placed below umbilicus. 3 sessions of initial cycles were done without dwell time, further cycles with 2 lit each was instilled and dwell time of 30 minutes was kept. DAY 1 –Completed 12 cycles of peritoneal dialysis, at the end of day 1 patient had very minor improvement in blood pressure, pupils were still dilated sluggishly reacting to light, no improvent in respiratory support or other neurological status. Day 2 – Completed 32 cycles of peritoneal dialysis, she had significant improvement in blood pressure with noradrenaline support 8mg in 50 ml ns @ 5ml/hr, GCS was same with no improvement in ventilator support. Pupils were mildly dilated reacting to light. DAY 3 – Patient on peritoneal dialysis completed 50 cycles, blood pressure was 100/60 on noradrenaline 8mg in 50 ml ns @ 2ml/hr, Glass glow Coma Scale started improving to E3VETM4. Pupils normal reacting to light. Patient was moving her limbs and opening her eyes on deep painful stimulation. DAY 5 – Patient on peritoneal dialysis completed 70 cycles, ionotropic support was tapered and stopped BP was maintaining 100/60 throughout the day and patient was conscious oriented, obeying commands, neck holding present. GCS improved to E4VETM6. DAY 5 - Ventilator was removed, peritoneal dialysis was stopped, patient was observed in ICU for next 24 hrs peritoneal dialysis catheter was removed then she was shifted to ward. She was observed for another 2 days and then discharged.

Discussion

Barbiturates are a class of sedative-hypnotic drugs. They are commonly used as antiepileptic's (phenobarbital) and for the induction of general anesthesia (thiopental) [3]. Barbiturates act on GABA-A receptors by increasing the amount of time the chloride ion channel is opened, which increases the affinity of the receptor for GABA. They are classified as long acting, intermediate acting, short acting and ultra-short acting [4]. Phenobarbital is long acting barbiturates. The toxic dose varies, an oral dose of one gram for most barbiturates can cause significant poisoning in an adult. Fatal cases have occurred after ingestion of 2 to 10 gms of barbiturates [2]. Therapeutic serum drug level varies from 10-40 mcg/ml, lethal effect reported at serum level of 80 mcg/ml [5]. In our case ingestion was around one gram, probably fasting for 2 days before ingestion lead to overt manifestation. We were unable to check blood levels due to unavaibility of resources.

Symptoms of barbiturate toxicity vary from case to case but commonly include difficulty thinking, decreased level of consciousness, bradycardia, poor coordination, vertigo, nausea, muscle weakness, thirst, oliguria, decreased temperature, and dilated or contracted pupils. Fatal cases are marked by coma, hypotension, and respiratory depression [2]. Our patient presented with severe toxicity of barbiturates, she was drowsy not responding to painful stimulus, pupils were dilated and sluggishly reacting to light, respiratory depression and severe hypotension requiring intubation with inotropic support. Initial management of severe barbiturate poisoning include supportive care with intravenous fluids, mechanical ventilation and inotropic support, the administration of activated charcoal (a single 1g/kg dose) to limit the enterohepatic recirculation of barbiturates is useful very early of patient presentation. That can be administered maximum 6 hours of toxin ingestion. Our patient presented 9 to 10 hours of drug consumption. Short acting barbiturates are metabolized in liver while long-acting barbiturates like phebarbital are excreted renally thus urinary alkalinization to increase their urinary excretion can be useful treatment option in this case [6]. This was attempted in our patient also but in view of severe hypotension she started having low urinary output which lead to decrease urinary excretion of phenobarbital. She persists to have severe symptoms and poor response to above treatment.

There is no specific antidote for overdose of barbiturates. Removal of toxin from body remains further option if there is no improvement of symptoms. Only 20 to 40 per cent of phenobarbital in the circulating blood is bound to plasma protein[6]. Because of these pharmacologic characteristics the phenobarbital should be more easily removed from the plasma than short-acting barbiturates. By lowering plasma level. tissue concentration is concomitantly lowered and mortality can be prevented [6].

Schreiner listed the indications for hemodialysis in barbiturate poisoning. These includes:

(1) Progressive deepening of anesthesia or progressive deterioration of the patient's condition

(2) Ingestion of a potentially fatal dose

(3) Blood level in the potentially fatal range

(4) development of any severe complication.

These indications are also applicable for peritoneal dialysis [7] The 2015 recommendations of the EXTRIP (Extracorporeal Treatments in Poisoning) Workgroup suggest using intermittent hemodialysis to treat long-acting barbiturate poisoning in case of prolonged coma, shock (after initial fluid resuscitation) [8]. The successful use of hemodialysis in lowering blood barbiturate levels and decreasing the expected period of coma has been reported [9].

Peritoneal dialysis though less efficient than hemodialysis is an effective technique for removing many toxic substances from the blood. Procedure of peritoneal dialysis is simple does not require very trained personnel as in hemodialysis. Despite its potential value, there are few cases reported about use of this procedure in barbiturate intoxication. Muirhead in 1951 suggested that peritoneal lavage might be an effective means of therapy for barbiturate poisoning in humans [10]. Very early in 1954 Lackey used peritoneal dialysis in experimentally produced barbiturate intoxication in dogs [11]. He found that recovery of the drug was small and that treatment had no effect on the course of recovery. Maxwell in 1959 first reported the use of peritoneal dialysis in the treatment of severe barbiturate poisoning [12]. Cohen et al. reported use of peritoneal dialysis in a comatose patient with amobarbital overdosage [13].

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In the current case, peritoneal dialysis dramatically improved the clearance of phenobarbital and, hence, neurological status improved concomitantly. Patient underwent slow but steady removal of barbitone toxin over 4 days. To conclude acute peritoneal dialysis is very effective in preventing dreaded complications of long-acting barbiturate toxicity.

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