Plasmapheresis: A Therapeutic option for Ethylene Di Bromide poisoning.

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Abstact:

Eleven Ethylene Di Bromide poisoning cases were treated by plasma pheresis. The results are encouraging. This can be a life-saving therapeutic option.

Introduction:

Ethylene-Di Bromide (popularly Known as EDB) is highly toxic pesticide & results in 90% mortality, if not intervened in time. Ethylene Di Bromide is rapidly absorbed from gastro intestinal tract, skin and lungs & produces fulminant hepatic failure & renal failure. Plasma pheresis is used as an early treatment modality, so that liver & real failure could be prevented. We did plasma pheresis in 11 patients of EDB poisoning out of which 6 could be salvaged with early intervention, which otherwise has significantly high mortality.

Case Descriptions

Case I:

Twenty-six yrs female reported within 2 hours of ingestion of 2 ampoules of EDB. Her initial SGPT (6hrs after admission) was 450U/L, immediately she under went plasmapheresis 2 cycles, each 12 hrs apart, SGPT during course rose up to 1800U/L & when started declining 3 days later, it was stopped. The patient gradually recovered over 7 days & was discharged.

Case II:

Forty yrs male presented with ingestion of 2-3 ampoules of EDB with alcohol in a state of shock. After initial management (fluid & vasopressor) when SGPT was found to be rising from 550-1100U/L, patient was taken for plasma pheresis 12 hrs later. He required 3cycles of plasmapheresis & later when SGPT levels started declining to 200U/L, he was managed conservatively & discharged.

Case III:

Twenty-two yrs female reported within half an hour of ingestion of EDB, 2 ampoules. After gastric lavage & fluid management, she underwent only single plasmapheresis within 4hrs of admission. Her SGPT rose up to 110U/L initially & when her SGPT started declining, she was discharged.

Case IV:

Twenty-five yrs female who had consumed 3 ampoules of EDB & one tablet of Aluminum Phosphide, reported to us after 8-10 hrs of ingestion. She was in cardiogenic shock, with ECG showing features of carditis & Echo showing LVEF 25%. She could undergo only one cycle of plasmapheresis & succumbed within 48 hrs of admission to multi-organ failure.

Case V:

Twenty-eight yrs male presented after 12 hrs of ingestion of 3 ampoules of EDB. On admission his SGPT was 2000U/L, which gradually rose to 4128 U/L in spite of 2 cycles of plasmapheresis. He underwent 5 cycles of plasmapheresis, each 12-24hrs apart and when SGPT started declining, the patient was discharged 12 days later.

Case VI:

Thirty-five yrs male was shifted from local hospital after 72 yrs of EDB ingestion. He was in hepatorenal failure with upper gastrointestinal bleed. Patient during plasmapheresis developed ARDS & was put on ventilator. He succumbed after 24 hrs.

Case VII:

Thirty-one years male presented with ingestion of 2 ampoules of EDB. He was taken for plasmapheresis. His SGPT levels gradually rose up to 3590 U/L. He underwent 3 cycles of plasmapheresis and was discharged 10 days.

Case VIII:

Thirty-five yrs male presented 8-10 hrs after ingestion of 10 ampoules of EDB. He was taken for plasmapheresis but succumbed to multiorgan failure within 6 hrs.

Case IX:

Twenty-three yrs old male had ingested 14 ampoules of EDB. The liquid had been kept in a bowl for some days and then was ingested. His SGPT levels rose up to 2495 U/L and after one cycle of plasmapheresis he succumbed.

Case X:

Twenty yrs male had ingested 1 ampoule of EDB and presented within 3-4 hrs. His SGPT levels gradually rose up to 160 U/L. He was taken for 1 cycle of plasmapheresis and when his SGPT levels started declining, he was discharged.

Case XI:

Thirty-five yrs old female presented 2-3 days after ingestion of 2 ampoules of EDB. Her SGPT on admission was 1420 U/L. After one cycle of plasmapheresis, she succumbed to multi-organ failure.

Discussion:

EDB is a toxic, fumigant pesticide & a severe eye, mucous membrane, skin irritant & kidney, liver & lung toxin. [(1)] Consumption of EDB as suicidal agent is very high. EDB is 1, 2, Di Bromoethane, a colorless, liquid which is readily absorbed from G.I. Tract when ingested orally & from lungs when inhaled. [(2)] Highest concentration is seen in liver, kidney & adrenals. 65% of metabolites are excreted in urine, 3% in faces. Acute liver and kidney damage have caused human fatalities from EDB exposure. [(3)] In vitro studies of EDB metabolism in human liver show that EDB is metabolized by cytosolic glutathione-S transferases, microsomal GST & microsomal mixed function oxidases. EDB is metabolized by two pathways:

1. Oxidative pathway yields metabolite – 2 - Bromo Acetaldehyde which is associated with cell macromolecule binding and liver damage.
2. Conjugative pathway principally yields glutathione products such as S – 2 – Bromoethyl Glutathione mainly responsible for DNA binding & mutagenesis. These products irreversibly damage proteins and DNA.

Apheresis means filtration and plasmapheresis means filtration of plasma to remove the circulating toxin or auto antibody. It is frequently used for various poisoning like organo-phosphorus [(4)], carbamazepine [(5)], and mushroom (Amantia phylloides) [(6)]. It hastens the removal of the toxic agent. 2-3 cycles of plasmapheresis are usually required and removed plasma is replenished with fresh frozen plasma. Our study showed that the patients who under went plasmapheresis early, showed reduced mortality and morbidity. Since the biological degradation products of EDB could no be measured in serum, therefore SGPT was taken as marker of recovery and deciding of further cycles of plasmapheresis.

Conclusion:

Plasmapheresis is a good therapeutic option for removal of toxic products of EDB if carried out as early as possible, before irreversible hepatic and renal damage sets in.

References:

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