PROPHYLAXIS KILLS

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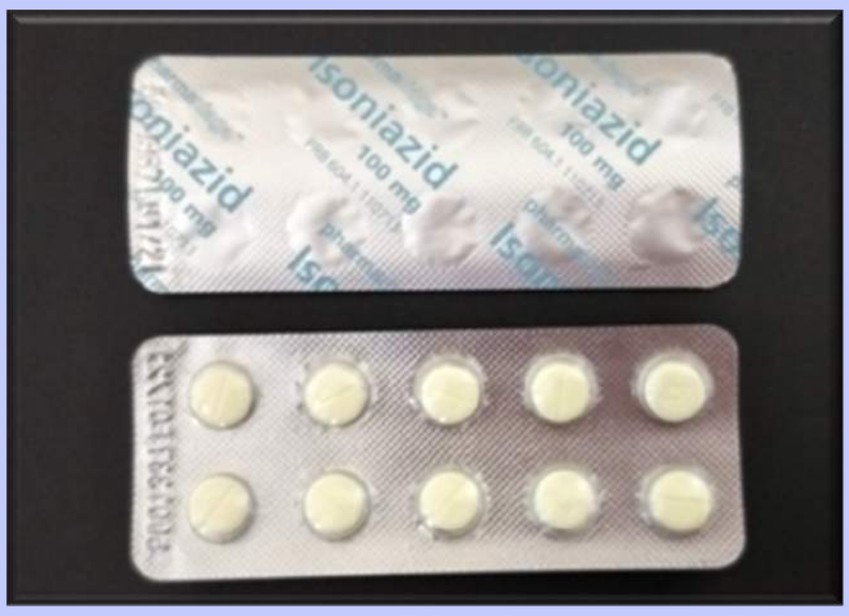
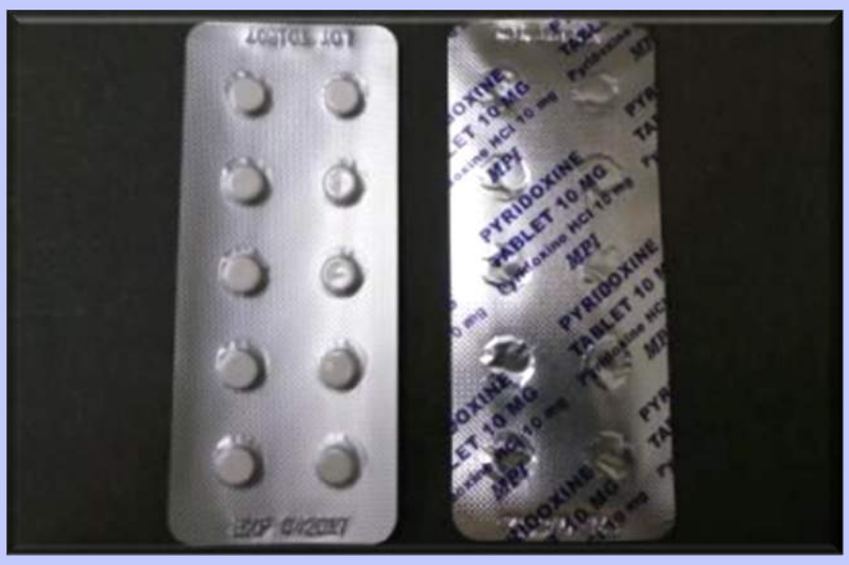
INTRODUCTION

Isoniazid (INH) toxicity is a rare presentation in emergency department. It has high mortality rate hence prompt treatment with appropriate antidote is vital.





CASE DESCRIPTION

A 28-year-old Indonesian lady was brought to our casualty by prehospital team who witnessed an ongoing seizure at her resident. At scene, they found empty strips of medication next to the patient. The medication was prescribed to her prophylactically as her child was recently diagnosed with pulmonary tuberculosis. Upon arrival to ED, she was stuporous, tachypneic and had another episode of generalized tonic seizure. As she was not responding to boluses of benzodiazepines, we intubated her for airway protection. Initial blood parameters showed severe metabolic acidosis with normal capillary blood sugar level. There were no evidence of leukocytosis or any electrolytes abnormalities. We identified the empty strips of medication as isoniazid with a total of three grams assumed to be ingested. She was started on three grams of intravenous pyridoxine due to limited availability in our center with remaining doses given orally from crushed tablet preparation. Police report was made and further history from the husband revealed that she ingested total of five grams of isoniazid. She was admitted to medical ward and required repeated bolus of intravenous pyridoxine. Her condition continued to deteriorate, she remained comatose, and succumbed to death after seven days.

DISCUSSION

INH toxicity is commonly intentional with triad of refractory seizures, high anion gap metabolic acidosis and coma.[1] Its incidence in our country is unknown and associated with high mortality up to 20%.[1] Human ingestion of greater than 80mg/kg to 150mg/kg may results in seizures which usually refractory to conventional therapy.[2]

Diagnosis of INH toxicity is made on clinical ground.[1] Serum INH levels are not available widely in our country, hence physician should not wait for INH levels before initiating antidote therapy. Furthermore, serum INH is poorly correlated with clinical severity of INH toxicity.[3] Detail history taking with high index of suspicion are important to avoid misdiagnosis.

The antidote of INH toxicity is pyridoxine which rapidly terminates seizures, corrects metabolic acidosis and reverse coma. The proposed dosage of pyridoxine should equal the amount of INH ingested, or up to five grams intravenously if the ingested amount is unknown.[4] Refractory seizures and coma despite pyridoxine initiation should receive an additional dose of pyridoxine. In the setting of limited availability of intravenous preparation of pyridoxine, tablet pyridoxine may be crushed and administered via nasogastric tube.[5]

Our country shows increase trend of tuberculosis prevalence, indicating more antituberculosis drugs were prescribed. Effort should be made to stock appropriate amount of antidotal therapy to antituberculosis in the event of toxicity. Prompt therapy with high dose of pyridoxine has been shown to result in good recovery.

CONCLUSION

INH toxicity is fatal if not treated properly. It requires high degree of suspicion and prompt initiation of antidote with high dose pyridoxine and general support measures.

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