CASE REPORT

# SUICIDAL ISONIAZID POISONING

**Haji Khan Khoharo, Shuaib Ansari*, Allauddin Abro**, Fatima Qureshi**

**Department of Medicine. Muhammad Medical College Hospital, Mirpurkhas, *Liaquat University Hospital Hydyderabad/Jamshoro, **Department of Biochemistry, Ghulam Muhammad Medical College, Sukkur, Pakistan**

Accidental or intentional Isoniazid poisoning may manifest within half to three hours as intractable seizure, acidosis, and coma. Single high dose of pyridoxine was used as an antidote with good response as reported earlier. Ingestion of more than 80 mg/kg body weight produces severe central nervous system symptoms and a dose of 125 mg/kg is potentially lethal if not promptly treated. We report a case of suicidal attempt with use of Isoniazid, who developed grand mal seizures and was controlled with diazepam and symptomatic treatment.

**Keywords:** Isoniazid, INH, grand mal, suicidal attempt, poisoning

## INTRODUCTION

Isoniazid (INH) is anti-tuberculous drug used mainly in the treatment and prevention of tuberculosis. It may cause hepatitis with long term use, especially in alcoholic and elderly persons. It produces acute toxic effects by competing with pyridoxal 5-phosphate, resulting in lowered brain ϒ-aminobutyric acid (GABA) levels. Acute ingestion of as little as 1.5–2.0 g can cause toxicity, severe poisoning is likely to occur after ingestion of more than 80–100 mg/kg. Confusion, slurred speech[1], toxic psychosis[2] and seizures may occur abruptly after acute overdose[1]. Severe lactic acidosis out of proportion to the severity of seizures is probably due to inhibited metabolism of lactate. Peripheral neuropathy and acute hepatitis may occur with long term use.[1] Its overdose taken either accidental or suicidal has been reported with wide clinical manifestations.[2] We report a case of one lady, who took overdose of INH for suicidal purpose immediately following family dispute.

## CASE REPORT

A 26-year-old lady was admitted through emergency room (ER) in November 2008 following intentional ingestion of more than 50 INH tablets (100 mg each) immediately following family dispute. Her husband was on anti-tuberculosis drug therapy, and family was facing financial problem. She was brought immediately to hospital, developed grand mal seizure 30–40 minutes after ingestion of INH and was give diazepam 10 mg intravenous as diluted bolus. She again developed another 2 tiers of generalised seizures in the ER. Both fits were aborted with diazepam 10 mg infusion. A thorough clinical examination revealed restless and drowsy patient with grade III coma. The vitals were stable with increased respiratory rate (acidotic breathing) without cyanosis and no neurological deficits. The patient was having mid-dilated pupil with sluggish light reflex. Deep tendon reflexes were also diminished. Bedside routine laboratory tests were done. The LFT's revealed mild increases in SGPT (thrice normal) with normal PT and APTT. Serum electrolytes, blood sugar, urea and creatinine were within normal limits except reduced bicarbonate (12 mmol). Serum *p*H and INH level could not be done due to lack of facility. Gastric lavage was performed after endotracheal intubation to protect the airway. Activated charcoal was given. In the absence of injectable preparation of pyridoxine, she was treated with oral pyridoxine. Intravenous fluids and symptomatic treatment was given, intravenous diazepam, phenytoin, and sodabicarb as intensive treatment were administered. Recovery of patient was uneventful, she made full recovery within 48 hours and was discharged on 3rd day on anxiolytics, antidepressants and pyridoxine.

## DISCUSSION

Accidental or intentional INH poisoning may manifest within half to three hours as intractable seizure, acidosis, and coma. Single high dose of pyridoxine was used as an antidote with good response as reported earlier.[2] Ingestion of more than 80 mg/kg body weight produces severe central nervous system symptoms[3] and a dose of 125 mg/kg is potentially lethal if not promptly treated[4]. In literature, one paper had documented INH dose as high as 12 and 15 gm and all these patients survived with effective treatment[5]. Tai *et al*[6] had reported one case in which the recovery was uneventful after INH toxicity. Sood *et al*[7] reported INH toxicity in a young girl in which the generalized tonic clonic seizures were successfully treated with high intravenous doses of pyridoxine. Intravenous pyridoxine should be administered in amounts equal to the estimated quantity of INH ingested, even in asymptomatic patients[6]. Large doses of pyridoxine have shown to prevent the seizures and metabolic acidosis caused by ingestion of more than 2–3 gm of INH. The earlier the

pyridoxine is given the fewer the complications.[6] Wason *et al*[8] reported five cases of their own with review of 41 cases from the literature. All their reported patients had seizures, coma and acidosis. Among this group of patients, all had vertigo and different stages of coma I to III. Cent percent seizure activity and acidosis is reported in cases of Wason *et al*. Prabhakaran[9] reported death with 0.5 gm of INH. Serious ill effects were also observed by Ansari *et al*[10] after 0.6 gm of INH. Eight cases of accidental INH toxicity were reported by Agarwal *et al*[2] in whom the intractable seizure and acidosis were observed in two cases only and the recovery was uneventful in all these cases. No residual toxicity and neurological effects were recorded in our patient on follow up.

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## Address for Correspondence:

**Dr. Haji Khan Khoharo,** Bungalow No. C-17/II, Phase I Anwer Villas, Behind Govt: Technical College New Wahdat Colony, Qasimabad, Hyderabad, Pakistan. **Tel:** +92-321-3010577
**Email:** drhajikhan786@gmail.com