A case of bismuth intoxication with irreversible renal damage

Abstract: Bismuth is a chemical element symbolized as Bi and is a trivalent poor metal, which chemically resembles arsenic and antimony. Colloidal bismuth subcitrate (CBS) and bismuth subsalicylate are the bismuth salts widely used in the treatment of peptic ulcers, functional dyspepsia, and chronic gastritis. Intoxications with CBS are rare. In a few case reports, acute renal failure was described, but the literature review revealed no chronic renal failure related to CBS intoxication. In this case report we present a 21-year old female with chronic renal failure after a one year follow-up of CBS intoxication.

Keywords: acute renal failure, bismuth, intoxication, chronic renal failure

Introduction

Bismuth is a chemical element with the symbol Bi and atomic number 83. Bismuth, a trivalent poor metal, chemically resembles arsenic and antimony. Bismuth salts, especially colloidal bismuth subcitrate (CBS) and bismuth subsalicylate, are widely used to treat peptic ulcers, functional dyspepsia, and chronic gastritis. Other names for CBS include colloidal bismuth subcitrate and tripotassium dicitratobismuthate.

In general, the mechanisms of action of CBS are still not fully understood, although prostaglandin E2-mediated mechanisms, antimicrobial activity, and alteration of the viscosity of the gastric mucus with decreased H+ diffusion are discussed.1,2 Intoxications with CBS are rare. In a few case reports, acute renal failure was described.3

The reported toxic effects caused by overdose of bismuth compounds include encephalopathy, nephropathy, osteoarthropathy, gingivostomatitis, and colitis.3 Bismuth poisoning mostly affects the kidney, liver, and bladder. Chronic exposure to high levels of bismuth salts result in encephalopathy, whereas acute toxicity manifests as nephrotoxicity.2 In this case report, we present a 21-year-old woman with chronic renal failure 1 year after CBS intoxication.

Case report

A 21-year-old woman was admitted to the emergency department 4 hours after taking 20 tablets of CBS in a suicide attempt. Each tablet included 300 mg of CBS, which is equivalent to 120 mg of Bi₂O₃. There was no alcohol or another intravenous drug usage. In the emergency service, the patient underwent gastric lavage and received intravenous fluid therapy, after which she was admitted to the internal medicine intensive care unit.
Eight weeks after discharge, the patient's renal function discharge were 5.2 mg/dL and 20.0 mg/dL, respectively. Her serum creatinine and BUN levels upon recovered clinically and was discharged after 15 days.

Levels of LDH, ALT, and AST also returned to normal. The patient's urine output progressively increased. Arterial blood gases returned to normal. Afterwards, conduct daily for the first 4 days and then three times 8 hours for 14 days. Hemodialysis was performed because of the tubular necrosis of proximal tubule, defective reabsorption occurs. Although reversible Fanconi's syndrome has been described in heavy metal intoxication, our patient developed irreversible Fanconi's syndrome that resulted in chronic kidney failure. The patient continues to require hemodialysis.

Türkez et al provided biochemical evidence of CBS-induced hepatic injury. As a matter of fact, the elevation in
transaminase is encountered in conditions causing hepato-cellular damage, loss of functional integrity of the cell membrane, and necrosis such as in chemically induced liver injury and elevation in enzymes.11 Türkez et al10 showed that the lowest doses of CBS caused the greatest increases of ALT and LDH after 24 hours. ALT and LDH were increased in our patient as well. In case reports published thus far, ALT and LDH were normal and no one had chronic renal failure. The increased ALT and LDH in our patient may be a poor prognostic factor.

Clinicians should be aware that chronic renal failure could occur after bismuth intoxication. Therefore patients with bismuth intoxication should be monitored with renal function tests, liver function tests, and monitoring of arterial blood gases.

Disclosure
The authors report no conflicts of interest in this work.

References