CLINICAL IMAGES

Unusual manifestations of emesis

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Key words: emesis, Trousseau's sign, Chvostek's sign

Case Description

A 30-year-old woman, with repeated emesis, more than 20 times in the last 4 hours, which was triggered by motion sickness. The emesis was then followed by complaints of tingling and cramps in both hands. The patient had no previous history of disease or medication. On physical examination, there were signs of dehydration. Interestingly, signs of carpopedal spasm were accidentally discovered when measuring blood pressure. Then a further examination was carried out to look for Trousseau's sign, which was later found to be positive (Figure 1A). There were also signs of twitching of the lateral facial muscles when tapped (Chvostek's sign) (Figure 1B). An ECG evaluation was performed, and the QT interval was prolonged (480 millisecond) which is associated with ST segment prolongation (corrected with the Fridericia formula, obtained QTc= 488 millisecond) (Figure 1C). Electrolytes (Potassium, Sodium, Chloride), blood glucose, and routine blood tests are all within normal levels. Treatment options include intravenous fluid rehydration, antiemetics, and antihistamines to relieve the symptoms. Supplementation of Calcium lactate 1000 mg and vitamin D 1000 IU were also administered. Interestingly, during

observation, the patient's clinical symptoms improved significantly, and ECG revealed a normal QT interval.

Discussion

Emesis can cause metabolic alkalosis as a consequence of the loss of gastric acid and fluid through the acid-producing activity of parietal cells. In contrast, HCO3- ions are released into the bloodstream via the anion exchange protein, creating an alkaline condition known as the "alkaline tide" (1). Severe emesis will cause significant loss of gastric fluid, thus triggering volume depletion which triggers the Renin-Angiotensin-Aldosterone system (RAAS), thereby preventing sodium loss by increasing Na+ absorption and secreting K⁺ and H⁺ ions. Both increased secretion of H⁺ which triggers increased reabsorption of HCO³⁻ through the α-intercalated cells and K⁺ secretion through exchange with Na+ absorption via ENaC can cause metabolic alkalosis (1). In metabolic alkalosis, hydrogen ions bound to albumin undergo dissociation, thereby increasing the fraction of albumin that can bind to ionized Ca²⁺. Therefore, metabolic alkalosis causes hypocalcemia (2). In this case, severe emesis

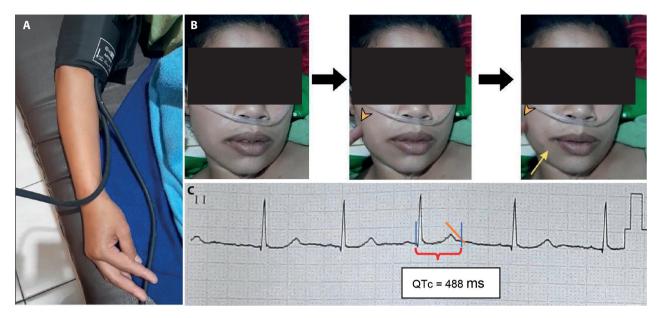


Figure 1. (A) Trousseau's sign. (B) Chvostek's sign is depicted with image sequences. The yellow arrowheads point at the finger tapping on the facial surface. The yellow arrow points at the twitched lip commissure. (C) QTc interval prolongation.

can be explained as the trigger of clinical manifestation of hypocalcemia such as Trousseau's sign, Chvostek's sign and QT interval prolongation (3).

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