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Case Report

Mulethi (licorice): real but unrecognised hazards of superstitions in mothers: a case report

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ABSTRACT

Licorice extract has long been known as a natural sweetener and thirst reliever. Many people who take large amounts and are prone to complications overestimate its nutritional benefits. Glycyrrhetic acid, the active metabolite in licorice, inhibits 11-ß-hydroxysteroid dehydrogenase enzyme type 2 causing increased cortisol levels and hypernatremia, hypokalemia. In this case report, we ensure a holistic study of licorice, as well as the documented side effects of excessive consumption. The study emphasises the necessity of looking into dietary habits and herbal medicines that are utilised around the world based on cultural and habitual grounds rather than scientific data. Not many case reports are available on this particular aspect of licorice consumption and its unknown adverse effects. We anticipate that our review will serve as a deterrent to parents's superstitious beliefs on the benefits of licorice consumption.

Keywords: Licorice, Sweetener, Mineralocorticoids, Glycorrhizin, Hypokalemia

INTRODUCTION

Licorice is a sweetener that can be found in a variety of soft drinks, foods and herbal medications. The traditional assumption that licorice is a safe, natural substance with no adverse effects encourages its excessive use, which can be extremely unsafe. In some climes, its value as a thirst quencher encourages excessive intake. Its primary component, glycyrrhizic acid, works in a similar way to mineralocorticoids (sodium reabsorbption and potassium secretion).[1] Here, we provide a comprehensive review of this chemical and its health risks, as well as a related case of excessive licorice intake.

Many licorice-containing commodities are readily available, unwitting large quantity consumption of which leads to various adverse effects. Licorice sticks and toffee bars, Pontefract cakes, chewing gums are all licorice-based snacks.[1] Licorice is also used as a sweet tobacco by miners/sailors while working in 'no smoking' circumstances. Licorice extracts are frequently employed as flavouring ingredients in medicinal formulations to hide the bitter taste. Herbal and licorice-flavored cough mixtures, throat pearls, licorice tea and laxatives are some of the licorice containing health products.[2] Public awareness of these compounds and their associated adverse effects is essential.

CASE REPORT

We have encountered a case of a 4 ½ year old girl, known case of developmental delay (predominantly affecting language milestones) with hearing defect with significant premature birth insult. Patient presented to our institution with recurrent episodes of vomiting since last 15 days, about 10-15 episodes per day. On admission, patient had fever of 101-degree Fahrenheit, heart rate of 108 beats/min, respiratory rate of 24/min, saturation of 99% on room air and blood pressure of 80/54 mmHg. Patient was dehydrated with a sick look. Patient was started on antiemetics. Patient had repeated episodes of vomiting even after admission. Mother also gave history of

constipation. Suspecting intestinal obstruction, X-ray erect abdomen was done suggestive of gasless abdomen (Figure 1a). Ultrasonography of abdomen and pelvis was done which was normal. Gastroenterologist opinion was sought and gastroscopy was planned. Astonishingly, patient was found to have 'mulethi sticks' in the oesophagus which was removed through endoscopy with the help of rat toothed forceps and roth net (Figure 1b and c). On further digging into the details, mother gave history of ingestion of mulethi sticks by the child as she believed that ingestion of mulethi sticks by kid would help to improve speech.

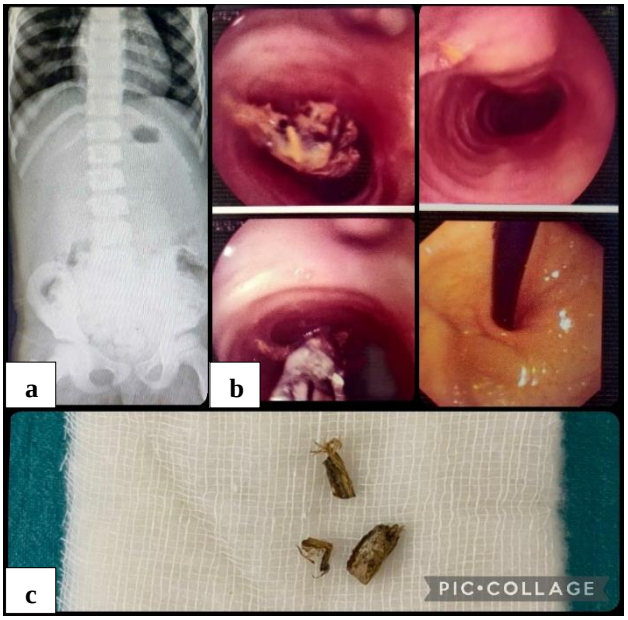

Figure 1: (a) X ray erect abdomen showing gasless abdomen, (b) mass in the oesophagus being removed by endoscopy, and (c) mulethi pieces removed from the oesophagus after endoscopy.

DISCUSSION

The general populace consumes licorice because of a long-held belief in its health benefits, being uninformed of the dangers of excessive use. Glycyrrhiza glabra, the most well-known species of licorice is made up of potassium–calcium–magnesium salts of glycyrrhizic acid. Glycyrrhizin has a 50-fold sweeter taste than sucrose.[4] It is an ancient medicine that was utilised by Egyptians and Assyrians as a sweet drink in the first millennium BC.[5] Soldiers and travellers drank licorice extract on the battlefields and in the desert to keep them from becoming thirsty during arduous marches.[5]

Glycyrrhizin has a low oral bioavailability and can only be detected at very low concentrations after a single oral intake.[6] Glycyrrhizic acid is hydrolyzed to glycyrrhetic acid by intestinal bacteria which is quickly absorbed and delivered to the liver via carrier molecules where it gets converted into glucuronide and sulphate conjugates, discharged into the bile, subsequently subjected to entero-hepatic circulation, resulting in prolonged maintenance of pharmacologically active circulating levels.[7] Glycyrrhizin is found in roughly 10–20 percent of licorice fluid extracts; typical doses of 2–4 ml yield 200–800 mg. The Scientific Committee on Food confirmed a daily limit of 100 mg in April 2003.[6]

Licoroce supresses 11-ß-HSD enzyme leading to increase in cortisol activity which binds to the mineralocorticoid receptor with the same intensity as aldosterone (MR). This explains its use in Addison's disease patients.[6] In patients with inexplicable hypokalemia and muscle weakness, licorice overconsumption should be suspected. Laboratory tests demonstrate hypokalemia and metabolic alkalosis due to its aldosterone-like activity. In cases of rhabdomyolysis (due to severe hypokalemia) creatine phosphokinase (CPK) may be high. The ratio of cortisol to cortisone in peripheral venous plasma is dramatically increased.[7]

Licorice also has a reported benefit in postural hypotension induced by diabetic autonomic neuropathy.[8] Bernardi and colleagues studied the effects of long-term licorice intake on serum potassium levels. At a daily dose of 800 mg or greater, a significant drop in plasma potassium concentration from 4.3 to 3.5 mmol/l was seen. It was used to cure gastric ulcers by Chinese when given 20-30 minutes before meals.[9] Licorice has been used in the treatment of polycystic ovarian syndrome in women in combination with spironolactone. Other favourable effects of licorice include its role in bone metabolism, as indicated by Mattarello and colleagues, evidenced by an increase in parathyroid hormone and urinary calcium levels. Due to the blockade of 17-HSD and 17–20 lyase, licorice can lower testosterone levels which aids in the treatment of hirsutism and polycystic ovary syndrome (PCOS).[9]

Licorice-induced hypertension and hypokalemic myopathy were the two most common consequences. The prognosis for licorice-induced hypertension was good, with a decent response after stopping the licorice. Hypokalemic myopathy manifests as flaccid paralysis similarly had a favourable prognosis, with full recovery following licorice discontinuation and potassium replacement. Some instances had a delayed recovery after hypokalemia was corrected, and a few others had acute renal failure due to myoglobinuria.

The arrhythmogenic impact of licorice due to hypokalemia and QT prolongation, torasades de pointes is the most common cause of cardiac arrest and death.[10] Hypersensitivity to glycyrrhizin, occupational asthma, myoclonus due to licorice-induced metabolic alkalosis, and licorice-induced contact dermatitis are among the other side effects.

CONCLUSION

This review aims to raise awareness of potential dangers of licorice. We stress the necessity of a complete and accurate dietary history, which includes licorice-

containing foodstuffs and herbal supplements, as well as the need to re-evaluate traditional patterns that encourage excessive licorice consumption due to a belief in its health benefits.

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