An unusual complication of snake bite

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Anterior pituitary hypofunction is a well-known complication following snake bite. However, central diabetes insipidus as a complication of snake bite is only rarely reported in the literature. We are reporting a case of central diabetes insipidus, which developed as sequelae to viper bite.

Keywords:
arginine vasopressin, central diabetes insipidus, hypopituitarism, polyuria, snake bite, viper

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Introduction

This case is unique because of its rarity. There are only four case reports of central diabetes insipidus following snake envenomation.

Case history

A 49-year-old man was admitted with complaints of diarrhea, vomiting, and altered level of consciousness. He gave a history of viper bite followed by acute kidney injury about 4 months back. He had features of dehydration. He was treated with antibiotics and IV fluids. Diarrhea stopped and he became fully conscious and oriented. Even after diarrhea subsided, he complained of severe fatigue and was noticed to have urine output of around 6 l/day. His blood sugar was 146 mg%, serum creatinine was 0.7 mg%, urine osmolality was 174 mosmol/kg, plasma osmolality was 332 mmosmol/kg, and serum sodium was 147 mmol/l. He was given injection desmopressin. His urine osmolality increased to 515 mosmol/kg. His urine output decreased and his serum sodium also decreased to 138 mmol/l. MRI of brain plain and contrast showed absence of bright signal of the posterior lobe of pituitary, thinned out lower infundibular stalk, and normal anterior lobe of pituitary. Free T3 was 4.5 pg/ml (1.2–4.2 pg/ml), free T4 was 47 pg/ml (9–22 pg/ml), thyroid-stimulating hormone was 0.01 μIU/ml (0.4–4.2 μIU/ml), and serum cortisol was 2.6 μg/ml (4.3–22.4 μg/ml).

Discussion

There are only four case reports of central diabetes insipidus following snake envenomation [1]. All of these reports are from India, with three being from South India. In one study, which included 1000 patients with snake bite, there was only one case of diabetes insipidus [2]. Viper venom contains procoagulant enzymes that can lead to consumption coagulopathy, metalloproteinase called hemorhagin that damages the vascular endothelium, and toxins that impair platelet function. Viper venom can cause spontaneous hemorrhage and microvascular thrombin deposition. These phenomena occurring in the pituitary gland are responsible for the development of pituitary dysfunction. These changes are very much similar to that seen in Sheehan's syndrome. Another mechanism that has been postulated is antigen–antibody reaction. The pathology for central diabetes insipidus following snake bite may lie either in the hypothalamus or in the posterior pituitary. The blood supply to the anterior pituitary is by the hypothalamo-pituitary portal system from the superior hypophyseal artery. The posterior pituitary receives blood supply directly from the inferior hypophyseal artery [3]. Newer dynamic MRI techniques have shown that occlusion of inferior hypophyseal arteries can be the cause of central diabetes insipidus [4]. Anterior pituitary dysfunction can occur without any structural changes obtained on imaging. This can be due to microvascular injury. Arginine vasopressin (AVP) and oxytocin are produced in supraoptic and paraventricular nuclei of the hypothalamus and are carried by axons to the posterior pituitary where they are stored, which is enough for a basal release for up to 30–50 days. As the posterior pituitary only serves as storage place for AVP, it is unlikely that diabetes insipidus develops unless the hypothalamus is also damaged. The same may be the reason why the clinical features of diabetes insipidus may regress after a short interval of time. It has been shown that, in situations where there is posterior pituitary damage, the site of release of AVP shifts to the median eminence, which can be demonstrated by the bright spot in MRI. AVP binds to V2 receptors on the basolateral membrane of renal collecting tubules, ultimately leading to insertion of aquaporin 2 channels on the apical membrane and helping to produce a concentrated urine.

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Polyuria occurs when more than 80% of neurons secreting AVP are damaged. Polyuria is characterized by urine output more than 40–50 ml/kg/day. This patient had a urine output of nearly 6 l/day. The causes for polyuria that were thought of were osmotic diuresis, recovery from acute tubular necrosis, or diabetes insipidus. There was no evidence for solute diuresis, and his renal function was normal. His urine osmolality was less than plasma osmolality, and his serum sodium was 147 mmol/l. This picture was consistent with diabetes insipidus. After desmopressin injection, there was more than 50% increase in urine osmolality and his polyuria also decreased, thereby confirming a diagnosis of central diabetes insipidus. MRI brain showed absence of posterior pituitary bright spot. His thyroid function test was interpreted to be due to sick euthyroid status. His serum cortisol was inappropriately low. In this patient, vascular occlusion produced as a consequence of viper bite could be the cause for the central diabetes insipidus as well as hypocortisolism. Acute kidney injury following snake bite may be an indicator for the later development of hypopituitarism. ACTH deficiency may mask the features of diabetes insipidus. Hence, polyuria may manifest after initiating steroid replacement for anterior pituitary dysfunction.

The treatment of central diabetes insipidus is with desmopressin, which is available as nasal spray, oral, or parenteral preparations. The adverse effects of desmopressin spray include rhinitis, epistaxis, eye irritation, headache, chest pain, palpitation, nausea, and vomiting. This patient was treated with desmopressin spray. The urine output decreased within 1–2 h of use of nasal spray. He was also given replacement dose of steroid. He showed remarkable improvement, and the desmopressin nasal spray was successfully tapered after 3 months.

Conclusion

Snake bite is an important cause of mortality in certain parts of the world. A high index of suspicion is to be maintained to diagnose hypopituitarism following snake bite. This complication can occur both acutely as well as in the later stages. Hence, the people should be on follow-up for at least 6 months after snake bite. Chronic fatigue and malaise could be a harbinger of pituitary dysfunction. Posterior pituitary dysfunction is far less common than anterior pituitary dysfunction, but still it is an important cause of morbidity. If polyuria develops, the diagnosis of diabetes insipidus should be strongly considered.

Acknowledgements

Conflicts of interest

There are no conflicts of interest.

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