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Case Report

Snake bite induced delayed hypopituitarism: a rare case report

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ABSTRACT

Hypopituitarism following snake bite induced AKI and dialysis is an uncommon complication. Often the presentation is delayed and Can present with a myriad of features. We present a case of a 27 year old male patient, with past history of snake bite and acute kidney injury (AKI) requiring dialysis and which subsequently resolved, presenting to us with fatigability and weakness, absence of secondary sexual character and poor academic performance. On examination, the patient had pale white and coarse skin. Hoarseness of voice was present. There was proximal upper and lower limb muscle weakness present with pseudo myotonia on examination. There was loss of axillary and pubic hair with low testicular volume. Routine reports suggested mildly elevated creatinine with severely elevated Triglyceride levels. Creatine phosphokinase (CPK) was raised. Hormonal profile revealed low free thyroxine (FT4) and serum 8 a.m. Cortisol but an inadequate increase in thyroid stimulating hormone (TSH) and adrenocorticotropic hormone (ACTH) levels were suggestive of central hypopituitarism. Luteinizing hormone (LH), follicle-stimulating hormone (FSH), Insulin-like growth factor 1 (IGF-1) levels were reduced. Neuroimaging revealed empty sella suggestive of pituitary apoplexy. The above were suggestive of delayed hypopituitarism following post snake bite dialysis mediated pituitary apoplexy. In all cases of snake bite, a common occurrence in our country the possibility of hypopituitarism should be kept in mind and the hormonal profile followed up closely along with the renal parameters.

Keywords: Acute kidney injury, Empty sella, Hypothyroidism, Hypopituitarism, Snake bite, Russel's viper

INTRODUCTION

The primary causes of Hypopituitarism can be developmental, traumatic, neoplastic, infiltrative, vascular and infectious. Among vascular, pituitary apoplexy is a leading cause[1]. Hypopituitarism can manifest as weakness, lethargy, hoarseness of voice, swelling of the body, reduced body hair, loss of libido, erectile dysfunction and menstrual disturbance Endocrinal insufficiency caused by vasculotoxic snake envenomation is an under diagnosed problem and can lead to acute and chronic hypopituitarism as well as adrenal gland failure. Linear depression surrounded by parallel transverse ridges running from the proximal to the distal end.

CASE REPORT

Our patient was a 27 year old non-alcoholic, non-smoker male who suffered from Russell Viper snake bite 11 years ago when he was 16 years old. He was admitted in hospital during that time with anuria for 23 days and received 6 episodes of dialysis during hospital stay. He was discharged with a diagnosis of snake bite induced acute kidney injury (AKI) when his creatinine values showed a downward trend and urine output was normal. Before snake bite his academic performance was normal and was apparently healthy.1 year post snake bite, patient started developing easy fatigability and lethargy and malaise which gradually progressed. The patient's academic performance progressively worsened and he had to drop out of school. He developed a hoarseness of

voice and pale white skin colour. Gradually over time there he developed coarse facies and skin. There developed regression of axillary hair which he previously developed and no appearance of any further secondary sexual characters like pubic hair and facial hair. The patient also developed shortness of breath of 2 months duration which was not associated with orthopnea, paroxysmal nocturnal dyspnea (PND) or any pedal swelling. There was no cough or any wheezing. On examination, he was conscious, alert and co-operative with mini-mental state exam (MMSE) score of 30/30. He was found to have moderate pallor, no jaundice, no cyanosis. Blood Pressure at presentation was 100/60 mmHg with pulse of 70/ min which was regular in rhythm and normal in volume. No neck swelling or thyroid swelling was observed. On abdominal examination, there was no tenderness. There was palpable hepatomegaly (liver span 17cm) with no splenomegaly. No other abdominal masses were palpable. Chest examination revealed normal vesicular breath sounds in all regions and no evidence of pleural effusion. Cardiac examination revealed normal findings without any audible murmur. On neurological examination, there was proximal muscle weakness in both upper and lower limbs with power of (4-)/5 in all 4 limbs. The knee and ankle jerks had delayed relaxation and upper limb biceps, triceps and supinator jerks were normal. There was no atrophy or hypertrophy of any muscles seen. No fasciculation was observed. Pseudo myotonia was seen in thenar muscles and in the proximal thigh muscles. No ocular myotonia was observed. Cranial nerve examination was normal. No cerebellar signs were seen. Sensory examination was normal. Testicular volume was found to be 15 ml which is low for his age. Routine blood reports revealed mildly macrocytic anemia (Hb- 7.2 gm/dl PCV-23.5% MCV 97.2fl corrected reticulocyte count-1.1%). Total WBC count (6800/cumm) and platelet count (1.7 lac/cumm) was normal. Renal parameters revealed Urea-11 mg/dl and creatinine -1.4 mg/dl with eGFR - 68.4 suggestive of stage 2 CKD as per KDIGO guidelines. . Routine urine report was normal with no evidence of pus cell, cast or proteinuria .Urinary albumin creatinine ratio was found to be 14.2 mcg/mg Creatinine. Sodium and potassium levels were in normal range. His liver profile was unremarkable with normal liver enzymes (ALT-10IU/dl AST-28IU/dl), and normal albumin globulin levels (albumin-3.7gm/dl globulin 3.5 gm/dl). Lipid profile revealed elevated cholesterol (340mg/dl) and triglyceride (1338 mg/dl) levels with reduced HDL levels. Serum calcium (10.2mg/dl) and FBS (102mg/dl), PPBS (131mg/dl) were normal. Triple serology was negative. In lieu of muscle weakness, CPK levels were done which revealed a very high value of more than 1000 IU. Ferritin level was mildly elevated (367 mg/lit).T4 level was low (0.21 ng/dl). TSH was in normal range (1.29 microIU/ml). Serum cortisol levels were very low (<1 micro gram/ dl) with ACTH within normal levels (45pg/ml normal- 0-46). IGF-1 was low (<25 ng/ml) LH and FSH values were low or the concerned age and sex (0.172mIU/ml and 0.156 mIU/ml) as illustrated in (Table 1).

Table 1. Table showing the hormonal profile of patient

Hormone (unit)	Measured value	Normal range
FT4 (ng/dl)	0.21	0.800-1.90
TSH (uIU/ml)	1.29	0.400-4.00
ACTH (pg/ml)	45.6	0.000-46.0
Cortisol (ug/dl)	<1.00	5.000-25.0
IGF-1 (ng/dl)	<25	
LH (mIU/ml)	0.172	0.800-7.60
FSH (mIU/ml)	0.156	0.700-11.1

Anti TPO antibody was found negative. EMG study of proximal upper and lower limb muscle revealed no spontaneous activity. USG whole abdomen revealed hepatomegaly and no other abnormalities. 2D echocardiography revealed chink of pericardial effusion. In Neuroimaging, MRI HPA axis was done which revealed normal sized but empty sella which was a sign of pituitary apoplexy. After diagnosis patient was started on oral hydrocortisone supplementation followed by oral L-thyroxin supplementation and intra muscular injection of testosterone supplements. After initiation of medication, the patient is doing clinically much better and his symptoms have reduced.

DISCUSSION

Snake bite is a common occupational hazard in India especially rural parts of the country. India is home to about 60 venomous snakes. Spectacled cobra (*Naja naja*), common krait *(Bungarus caeruleus)*, saw-scaled viper *(Echis carinatus)* and Russell's viper (*Daboia russelii*) are the most commonly encountered venomous variants. [2] Acute kidney injury is a common complication seen in snake bite patients. Kidney injury is more common after bites from hemato-toxic and myo-toxic snakes. The culprit snakes mainly include Russell's viper, saw-scaled viper, hump-nosed pit viper, green pit viper, and sea-snake. Its incidence varies from 8-30% in snake bite patients.[3] The primary mechanisms of renal injury are acute tubular damage, patchy or diffuse cortical necrosis and rarely glomerulonephritis, interstitial nephritis and cortical necrosis of the kidney. The time of onset of hypopituitarism in patients of snake bite can vary from a few weeks to more than 20 years. It can be the sequelae of an acute event or can be the manifestation of progressive pituitary damage over the years.[4] In most studies, it has been seen that hypopituitarism in snake bite patients is seen among younger patients. In the study by Bhat et al most common axis affected was of the corticotrophs which was deficient in 36.4%, followed by lactotrophs in 27.3% patients, gonadotrophs in 27.3% patients, somatotrophs in 15.2% and thyrotrophs in 12.1% patients. MRI pituitary axis in patients with

hypopituitarism following snake bite may reveal empty sella, partially empty sella, hemorrhage in pituitary or normal findings as seen in most studies. In the study by Bhat et al, the two primary predictors of development of hypopituitarism in patients were the number of hemodialysis required and the initial whole blood clotting time.[5] Local vascular occlusion by fibrin could be a major cause of the pituitary lesion but venom haemorrhagins may also contribute by damaging pituitary capillary endothelium.[4]

CONCLUSION

The symptoms from history and the subsequent investigations reveal a hypopituitarism. The stage 2 CKD can be a result of post snake bite AKI sequelae. Hypopituitarism is a neglected aspect in snake bite management and its incidence is not uncommon. An index of suspicion and follow up of the endocrinological parameters are necessary in early detection and management.

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