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REVIEW ARTICLE

SHEEHANS SYNDROME: POST PARTUMADE NOHYPOPHYSEAL INSUFFICIENCY: ACASE REPORT

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ABSTRACT

Sheehans syndrome can present with complete or partial pituitary insufficiency resulting from severe post partum hemorrhage. The pituitary gland, owing to its enlargement during pregnancy secondary to high estrogen levels and hypervascularity, remains vulnerable to arterial prressure changes and prone to emorrhage/infarction following hypovolemic shock encountered during post partum hemorrhage. The presentation ranges from sudden catastrophic event of hypovolemic shock followed by pituitary necrosis to gradual onset of partial to complete pituitary insufficiency over months to years. Here is the case of a patient presenting with central hypopituitarism 9 yrs after the acute episode of post partum hemorrhage.

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INTRODUCTION

Sheehans syndrome, first described by harold sheehan in 1937, is characterised by pituitary necrosis characteristically after post partum hemorrhage. It includes features of central hypothyroidism including peri-orbital puffiness, hoarseness of voice, dry coarse yellowed skin, sparse hair and features like dizziness, hypotension, hypoglycemia, vomitting, asthenia, weight loss signifying low cortisol production with ovarian failure characterised by lactational failure, ammenorrhea, regression of secondary sexual characters. For the signs and symptoms of central hypopituitarism to appear, atleast seventy five percent of the gland has undergone destruction/necrosis.

Case/Observation

35 year female presented to the casualty with drowsiness and inability to move limbs/inability to speak immediately after a seizure episode that had occurred 5 mins prior to her presentation, on the way from the ambulance to the casualty. patients relatives gave history of the patient having fever associated with chills, headache, drowsiness. Vomitting episodes and had not taken any fliud or solid food since 2 days. on examination the patient was drowsy, hemodynamically stable, bp 106/70 mm hg, taken over right arm supine position, pulse rate of 72 beats/min.

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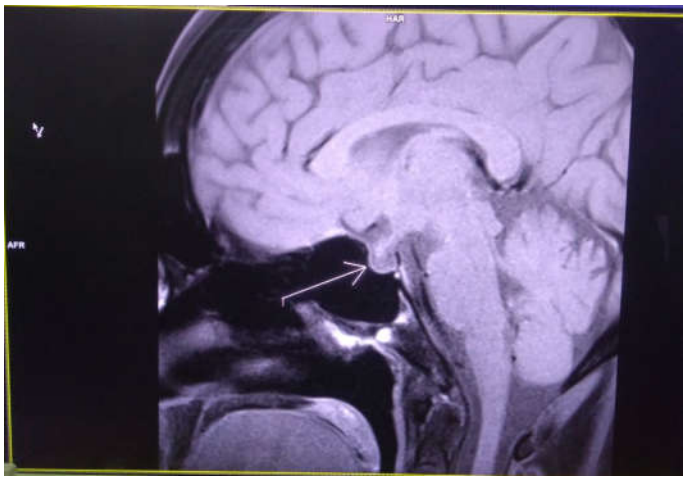
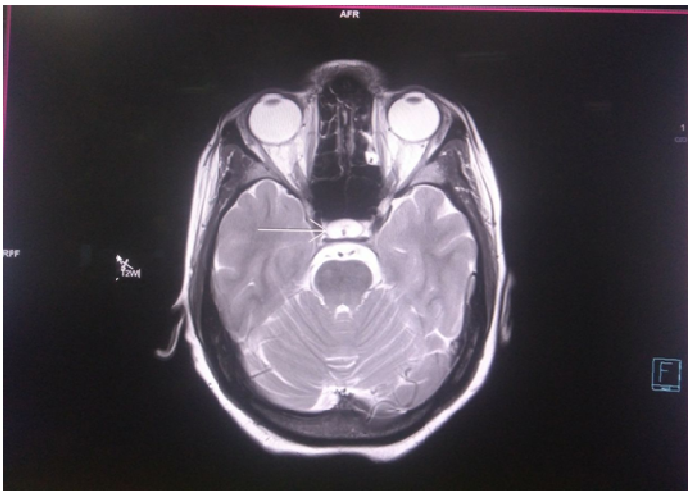
cns examination revealed drowsiness/confusion with inability to follow simple verbal commands. b/l pupils normal sized, reactive to light. upper and lower limb dtr present. the cardiovascular, respiratory and abdominal examination did not reveal any significant abnormality. patients blood sugar level was checked on the spot using a glucometer which revealed very low/nonrecordable blood sugar level, intravenous access was immediately established and patient was administered 25% dextrose infusion with improvement in her sensorium 2-3 mins after administration.Other possible causes of seizure were worked up. Serum electrolytes, calcium, magnesium were within normal limits. fundoscopy did not reveal any evidence of papilledema.cect brain did not reveal any significant abnormality. ecg revealed sinus rhythm with no st-t wave changes thus ruling out syncope due to arrythmias or brugada/stoke adams/inferior wall myocardial infarction given the clinical scenario of sudden collapse. Csf examination was within normal limits except for mildly elevated proteins. Patient gave history of ammenorrhea since 9 years immediately after her fourth and last child was born,with inability to nurse her last child after delivery. Previous three pregnancies remained absolutely normal with no evidence of lactational failure. Also history of extensive blood loss was elicited immediately after her last delivery followed by 3-4 blood transfusions suggesting catastrophic post partum hemorrhage. Patients skin was dry, coarse with facial and periorbital puffiness, hoarseness of voice. in view of these findings and history of ammenorrhea patients thyroid function was evaluated which revealed low t3 and t4 with lower normal levels of tsh.(t3-35.69 ng/dl,t4 0.85 ug/dl, tsh 1.39uiu/ml) suggestive of central hypothyroidism. patients

hormonal panel revealed low cortisol (1.35ug/dl),low prolactin (3.91 ng/ml) low fsh (2.8miu/ml),low lh (0.71 miu/ml), low gh(0.05ng/ml),low acth () suggesting hypopituirism. In the light of history of post partum hemorrhage followed by lactational failure and ammenorrhea with features of hypothyroidism and hypoglycemic crisis, central hypopituitarism secondary to pituitary necrosis in sheehans syndrome was kept as the provisional diagnosis. Mri brain confirmed pituitary atrophy with partial empty sella with secondary herniation of optic chiasm through diaphragm sellae thus confirming sheehans as the diagnosis. The diagnosis is confirmed by mri brain imaging on t1 and t2 weighted images showing empty sella with herniation of csf.





DISCUSSION

The Pituitary Glandis A Highly Vascular Structure With The Peculiarty Being The Hypothalamohypophyseal Portal System. It Is Hence Prone To Necrosis From Vascular Injury Following Sudden Hypotension/Sudden Pressure Changes As May Occur In Case Of Sheehans Syndrome After A Massive Post Partum Hemorrhage. Other causes of hypopituitarism include piyuitary and parapituitary tumors-craniopharyngioma, meningioma, irradiation, infiltration -sarcoidosis, lymphocytic hypohysitis, tuberculosis, hemochomatosis, histiocytosis, trauma. The clinical presentation depends on the type and degree of hormonal deficit. Empty Sella Syndrome Refers To An Enlarged Pituitary Fossa Due To Arachnoid Herniation Through Congenital Diaphragmatic Defect Or Secondary To Surgery, Radiotherapy Or Pituitary Infarction

Conclusion

Features of hypothyroidism and adrenal insufficiency with ovarian failure in a female with history of post partum blood loss should be worked up for central hypopituitarism keeping sheehans syndrome as one of the most likely diagnosis confirmed by an mri scan of the brain. Following are the mri images of the above patient. Saggital view sshows a partially atrophic pituitary gland with thin rim of tissue with herniation of csf into the sella. The transverse view shows csf as bright/white.it has occupied the empty sella just above the optic chiasm.

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