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OBJECTIVE

Sheehan's syndrome or postpartum pituitary necrosis, is an important but rare cause of hypopituitarism, caused due to severe postpartum hemorrhage. Seen more commonly in the developing world, it is less common in developed countries due to advanced obstetric practices. It can present acutely but more frequently has an insidious course (onset 10-20 years later) with variable hormonal deficiencies. Here, we report a late-onset case of Sheehan's syndrome, 24 years after the incident event, presenting as life threatening adrenal failure.

CASE PRESENTATION

A 48-year-old female with no significant past medical history was admitted to the hospital after being found unresponsive at home. She had not seen a physician for many years. She complained of weakness and lethargy for a week and recently established care with a primary care physician. The patient was severely hypotensive in the emergency department and had an elevated temperature of 101°F. Physical examination showed no significant abnormalities. CBC and metabolic panel were not significantly altered. CSF analysis and CSF/blood cultures were negative for any infection. TSH was 4.29 mIU/mL (0.27-4.20) but the total and free T4 (fT4) were severely low at 1.1 mcg/mL (4.6-12) and 0.24 ng/dL (0.93-1.70) respectively. On further questioning, patient reported severe postpartum hemorrhage 24 years ago, needing multiple units of blood transfusion. This was followed by inability to lactate and menstruate but was never worked up as she had not seen any physician all these years. Pituitary hormonal panel was obtained, demonstrating multiple hormonal deficiencies with fT4 severely low at 0.24 ng/dL, ACTH of 2.6 pg/mL (7.2-63.3), prolactin (PRL) 1 ng/mL (4.8-23.3) and insulin like growth factor-1 (IGF-1) low at 10 ng/mL (56-194). Cortisol level was elevated in the hospital due to administration of high dose IV steroids but a morning cortisol level obtained 1 week prior by her primary was 1.5 mcg/dL (10-20). Estradiol levels were low with FSH and LH levels inappropriately normal. MRI of the pituitary was obtained which showed an empty sella turcica. Patient was diagnosed as late-onset Sheehan's syndrome. She was started on hormone replacement with hydrocortisone followed by levothyroxine and had marked improvement in her symptoms. She continues to do well.

CONCLUSION

Our patient presented late due to lack of medical care and awareness. A great number of patients with Sheehan's diseasae go undiagnosed due to subtle clinical presentations, thus delaying treatment. It is imperative to diagnose this condition timely with appropriate obstetric/gynecological history and clinical suspicion to avoid late manifestations of the disease, especially adrenal crisis. Patients at risk need long term follow-up. Early treatment is necessary to improve quality of life and reduce morbidity and mortality associated with this condition.

Cardiovascular Endocrinology

ENDOCRINE HYPERTENSION AND ALDOSTERONE EXCESS

The Unsuppressed Plasma Renin Activity May Not Enough for Management of Non-Surgically Treated Primary Aldosteronism

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It is well known the primary aldosteronism (PA) is most common endocrinological hypertension and accounted for 10% among all hypertension population, and it develops cardiovascular disease more frequently than blood pressure matched essential hypertension. Those patients with bilateral hyperaldosteronism, called idiopathic hyperaldosteronism (IHA), or unwilling for surgical treatment are treated by mineralcorticoid receptor antagonists (MRAs). Although it had been unclear how titrate MRAs to prevent atherosclerotic cardiovascular events, a managemental target for those patients was recently reported as plasma renin activity (PRA) ≥ 1.0 ng/ml/hr to prevent cardiovascular events (Hundemer GL, et. al. Lancet Diabetes Endocrinol. 2018 Jan;6(1):51-59).

Thus, we investigated 77 cases of adrenal venous sampling performed patients with PA and followed up for 3 years in our hospital since 2007, including 24 males and 53 females, and their mean age was 56.3 ± 12.5 years old. All patients underwent AVS and showed bilateral hyperaldosteronism and treated with MRAs and followed up more than 3 years. We collected blood pressure, serum sodium and potassium concentration, estimated glomerular filtration ratio (eGFR), PRA, plasma aldosterone concentration (PAC), atherosclerotic parameter, such as mean intima media thickness (IMT), brachial-ankle pulse wave velocity (baPWV) and ankle-brachial index (ABI). We evaluated the relationship of those patients' PRA and aldosterone to renin ratio (ARR) with eGFR, IMT, baPWV, and ABI. The change of mean IMT after 3 year-follow up were 0.03 ± 0.11 mm vs. 0.06 ± 0.09 mm for well controlled (PRA ≥ 1.0 ng/ml/hr) and poorly controlled (PRA < 1.0 ng/ml/hr), respectively, and no significant difference between them. In the other hand, the change of mean IMT after 3 year-follow up showed 0.03 ± 0.10 mm vs. 0.08 ± 0.10 mm for well controlled (PRA ≥ 1.0 ng/ml/hr and ARR <20) and poorly controlled (PRA < 1.0 ng/ml/hr or ARR ≥ 20), respectively, and the mean IMT increase was significantly lower in this group.

The mean IMT increase showed significantly lower only with PRA ≥ 1.0 ng/ml/hr and ARR <20 rather than PRA ≥ 1.0 ng/ml/hr alone.

In our results, both PRA ≥ 1.0 ng/ml/hr and ARR<20 are important to prevent or improve atherosclerosis, rather than only PRA ≥ 1.0 ng/ml/hr and should be titrated MRAs to achieve this target.

In conclusion, our result revealed the titration of MRAs is important to prevent atherosclerotic cardiovascular event and not only PRA ≥ 1.0 ng/ml/hr, but both PRA and ARR <20 should be achieved.