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Review Article Open Acces

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[1]

ndings showed Fasting blood glucose in normal range, [FBS=87 mg/dL [74 – 110]], Serum electrolytes within normal range [sodium= 143 mmol/L [136 – 146], potassium= 4.3mmol/L [3.4 - 4.5]].

Serum creatinine was normal with [0.56 mg/dL [0.51 - 0.95]] and complete blood count within normal limit with hemoglobin of [12.3 g/dl [11.8 - 14.7]], leucocytes [6000/micro liter [4100 – 9900]] and platelets [233,000/micro liter [150,000-450,000]] and liver function test was also normal. VDRL, HBSAg, HCV Ab and HIV all were negative. e initial hormonal study done was compatible with hypopituitarism [thyrotropin = 0.09 mU/L [0.55 - 4.78], free thyroxine = 0.68 ng/dL [0.80 - 1.76], Free T3 = 3.18 pmol/l [3.5-6.5], Cortisol <2.9ng/dl [4.22 - 22.4], LH= 1.89 U/L [1.9 - 12.5], FSH = 2.62 U/L [3.9-12]; estradiol, within normal limit =353.73 pg/mL [93-575] and prolactin; in the lower half of the reference range = 8.74 ng/mL [2.8 - 29.2]]. Chest X-ray showed normal ndings.

Initial Brain MRI showed di use enlargement of the pituitary gland measuring 16x15x14mm and thickening of the pituitary stalk measuring 5.7mm which suggests lymphocytic hypophysitis (Figure 1). Patient was initiated on hormone replacement therapy with Prednisolone 7.5mg PO daily for secondary adrenal insu ciency, and followed by Levothyroxine 25 mcg PO per day for central hypothyroidism. A er four weeks of follow up treatment, she showed marked clinical improvement with no headache and started to breast feed her infant. She has also improvement in hormonal axes (Table 2). Control Brain MRI showed resolution of pituitary gland enlargement measuring 9x8x5mm and pituitary stalk thickening measuring 2.9mm (Figure 2). Progressively her prednisolone was reduced to 5mg PO daily and levothyroxine discontinued a er normalization of the free thyroxine level (Table 1 and Figure 1).

Figure 1: ,QLWLDO 05, D 7 ZHLJKWHG VDJLWWDO %UDLQ 05, E 7 ZHLJKWHG DLDO %UDLQ

Discussion

Lymphocytic hypophysitis is more common in female than male in a ratio of 6:1 according to P. caturegli [7], and tend to occur at younger age in women [35±13 years] compared to men [45 ± 14years] that is consistent with our case [7]. It is found that signi cant percentage of cases of lymphocytic hypophysitis occur during third trimester of pregnancy and the rst two months in the post-partum period [7,8] which is similar with our patient who presented one month a er delivery. One of the common presenting symptoms in the “single center case series” described by Brandon S. Imber et al. [9] was headache that was found in about 57% of cases [9] and it was the main presentation in our case. In about 80% of cases multiple hormone de ciencies are found in lymphocytic hypophysitis [10] which is a presentation in our case with cortisol, thyrotropin and gonadotropin de ciencies. Inhibition of lactation can be found in about 11% of cases [1,7].

Failed breast feeding was one of the rst presentation in our patient possibly associated with relative hypoprolactinemia. Due to pituitary stalk compressive e ect some cases may present with mild hyperprolactinemia which is not found in our case. Our patient’s initial presentation of severe headache with di culty opening the eyes and unresponsiveness to analgesics made us to consider pituitar apoplexy as a possible di erential diagnosis as it could present with similar signs and symptoms in the post-partum period. In our case the improved clinical and hormonal responses and the marked Brain MRI normalization to replacement dose of steroid avoided unnecessary neurosurgical management (Table 2 and Figure 2).

According to the description made by S. khare et al, [12] characteristically cortcotrophs are a ected in about 75% of cases lymphocytic hypophysitis but in pituitary adenoma the last hormone to be a ected is corticotroph which was the case in our patient with secondary adrenal insu ciency resulted from hypophysitis [8].

Brain MRI is the imaging procedure of choice in the diagnosis of lymphocytic hypophysitis. Currently it is not easy to distinguish speci cally hypophysitis from a pituitary adenoma based on radiologic nding and about 40% of patients are misdiagnosed as having pituitary macroadenoma and undergo unnecessary surgery [5,13]. erefore, based on the presence of clinical features and radiologic signs a scoring system by Gutenberg, A., et al [13] is proposed (Table 3). Here we applied the scoring to our patient individual items were added and a score of 12 was obtained which is suggestive of lymphocytic hypophysitis [13].

Lupi et al. [5] reviewed a total of 44 cases of autoimmune hypophysitis treated with glucocorticoids and/or azathioprine. A reduction in the size of the adenohypophysis was found in 84% of patients, the function of the adenohypophysis improved in 45% and that of neurohypophysis in 41% of these patients [5]. In our patient the majority of the hormone de ciency and all the symptoms had improved. e sizes of the pituitary and stalk were normalized following one month treatment with corticosteroid indicating the underlying autoimmune Etiology of lymphocytic hypophysitis and also showing that other inmuno supressive drugs can be used in the management of these cases. e role of corticosteroids in improving compression symptoms and as a replacement in lymphocytic hypophysitis is helpful in avoiding unnecessary transsphenoidal surgery [11]. Even though pituitary tissue biopsy was not done, on the basis of marked