	BEFORE	After	Normal Range
Blood test	Treatment may 2016	Treatment October 2016	
WBC	5.81×109/L	4.5×109/L	4.0—12.0
RBC	5.51×1012/L	5.9×1012/L	3.85.80
HGB	16.9 g/dL	16.4 g/dL	11.0—16.5
HCT	35%	51.6%	30.0—50.0
PLT	216×109/L	133×109/L	100—300
MCV	68.5 fL	87 fL	80.0—99.0
MCH	30.7 pg	27.6 pg	26.5—33.5
MCHC	483 g/L	31.7 g/L	320—360
GLYCEMIA	72 mg/dL	79 mg/dL	70—110
UREA	40 mg/dL	30 mg/dL	15—40
CREATINEMIA	1.1 mg/dL	1.0 mg/dL	0.7—1.2
URIC ACID	4.1 mg/dL	5.8 mg/dl	4.0—8.5
CHOLESTEROL	170 mg/dL	147 mg/dl	150—200
TRIGLYCERIDES	108 mg/dL	64 mg/dl	50—150
TOTAL PROTEIN	6.4 g/dL	6.8 g/dL	6.4—8.3
Na+	127 mmol/l	134 mmol/l	135.37—145.00
Cl-	102 mmol/l	114 mmol/l	96.00—106.00
Ca++	1.8 mmol/l	2.3 mmol/l	2.2—2.7
K+	8.3 mmol/l	5.1 mmol/l	3.48—5.50
Albumin	3.6 g/dL	3.8 g/dL	3.5—5.5
CORTISOLI 8.00 A.M	0.34μg/dL	2.41 μg/dL	5—25
ACTH 8.00 A.M	712.9	522.3	6—80 pg/Ml
TSH	6	3.63	0,4—4 μIU/ml
ANTI TPO	381	105	3—45 μIU/ml
ANTI 21-HYDROXILAZA	48.5	40.1	<0.1

used was 15—25 mg per day given as $^{2}/_{3}$ in the morning (20 mg) and $^{1}/_{3}$ in the evening (10 mg). Usual mineralocorticoid regimen is fludorcortisone 0.1 mg/day with monitoring of blood pressure, volume status, weight, plasma rein activity, sodium and potassium. **Discussion.** Type 2 Polyglandulare Syndrome typically occurs in early adulthood with a peak during the third or fourth decades and is three times more common in females than in males. This patient with, autoimmune thyroid disease and Addison disease has two major components of Schmidt's Syndrome. 10% of Schimidt's Syndrome patients have all three major DM, Addison and Hashimoto. Patients with autoimmune thyroiditis disease are prone to develope other autoimmune disease .In this case the patient with Addison is associated with Hashimoto thyroiditis.

KEYWORDS: polyglandular autoimmune syndrome, Schmidt syndrome, case report.

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A CASE OF HYPOPITUITARISM AND A SPONTANEOUS REGRESSION OF MASSIVE LESION OF HYPOTHALAMIC AREA

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Introduction. Hypopituitarism is a complex of one or more pituitary hormone deficiency due to diversity of underlying etiology, including rare poor studied forms. Clinical case presentation: we present a case of 57 years old woman with intracranial and extracranial mass lesions localized in medial part of the middle cranial fossa, skull base, third ventricle walls, chiasma opticum, sphenoid sinus and panhypopituitarism. The disease debut was associated with reactivation of chronic polypous sinusitis and left side otitis. It was suggested as a neoplasm due to aggressive progress of neurologic signs such as diplopia, vertigo, facial numbness and typical visualization feature with high contrast accumulation by MRI and positron emission tomography. But histological examination of sphenoid sinus mass, cytological liquor assessment did not reveal any tumor cells and alpha fetoprotein level was normal in contradiction to malignant lesion. Compensation of vital functions by substitution therapy by hydrocortisone 10—15 mg per day, L-thyroxine 75 mkg and desmopressin 0.1 mg twice a day improved overall health of the patient. Spontaneous regression of the vast majority of mass lesion within 6 months confirmed inflammatory process as a probable cause of this accident. **Conclusions.** Infection process could mimic tumor. Clear understanding of etiology of pathologic process in each case is necessary for accurate prognosis and treatment individualization.

KEYWORDS: hypopituitarism, case report.

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