Brief Communication

Subclinical hypothyroidism unmasked by preeclampsia and ascites

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ABSTRACT

Subclinical hypothyroidism is an asymptomatic endocrine disorder while hypothyroidism, on the other hand, can present with a wide range of clinical features which may be nonspecific. We describe a case of subclinical hypothyroidism in a 39-year-old pregnant woman presenting with preeclampsia and ascites. Ascitic fluid was found to be exudative as typically found in persons with hypothyroidism presenting with ascites. Treatment with levothyroxine resulted in complete resolution of ascites. The possibility of subclinical and clinical hypothyroidism should be borne in mind when persons with refractory exudative ascites of unknown origin are being investigated. Also, pregnant women with severe preeclampsia will benefit from screening for subclinical and clinical hypothyroidism.

Key words: Ascites, hypothyroidism, preeclampsia, subclinical hypothyroidism

Introduction

Hypothyroidism is an endocrine disorder with varied but often subtle clinical manifestations. Some of these include cold intolerance, weight gain, sluggishness, and slow mentation. Presentation with ascites is uncommon occurring in only 4% of patients with hypothyroidism.[1] Subclinical hypothyroidism (SCH) is defined as a serum thyroid stimulating hormone (TSH) level above the upper limit of normal despite normal levels of serum free thyroid hormones.[2] It is usually asymptomatic but common problems may include dyslipidemia, diastolic hypertension, and cardiac dysfunction. It may progress to overt hypothyroidism in approximately 2-5% of cases annually.[2] We present a case of unexplained ascites in a patient with severe preeclampsia, discovered to be related to subclinical hypothyroidism.

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Case Report

The patient was a 39-year-old G4P2 +[1] at GA29 + [3] who was referred to our center from a private hospital. She presented on account of poorly controlled hypertension, generalized edema, and deranged thyroid function test (TFT). She was admitted to the Obstetrics Department with a blood pressure (BP) of 210/130 mmHg from the referring hospital. There was a history of headache and generalized body swelling of a month's duration but no history of proteinuria. There was no history of seizures or previous history of pregnancy induced hypertension. The diagnosis at admission was severe preeclampsia with intrauterine growth restriction and anhydraminios. She was commenced on intravenous magnesium sulfate and oral antihypertensives.

Endocrinology review was requested on the same day on account of a deranged TFT result from the referring hospital [Table 1]. Patient had a TFT done because she was noticed to have an anterior neck swelling at a gestational age of 24 weeks. There was also a history of anterior neck swelling 6 years before presentation which had resolved spontaneously. There were no symptoms suggestive of hypothyroidism or hyperthyroidism in the past or at the time of review neither was there goiter at presentation.

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Examination revealed a conscious young woman who was not pale, anciteric, and acyanosed with periorbital edema and bilateral pitting pedal edema up to the knee. There was no anterior neck swelling. Pulse rate was 84 beats per min, BP was 140/100 mmHg, jugular venous pulse was not raised, and heart sounds were 1 and 2 only. The abdomen was distended with gross ascites demonstrable and uterus was difficult to palpate.

Obstetric ultrasound scan confirmed maternal ascites and anhydramnios with the fetus compressed. There was gross ascites of 2 litres with kidneys and liver being normal in size and echogenicity. No intra-abdominal mass lesion was seen. Intrauterine fetal death was confirmed and the patient was delivered of a macerated stillbirth following induction of labor. Broad spectrum antibiotics were added while urinary protein, her weight, and vital signs were monitored closely.

Reexamination on the third postpartum day revealed generalized edema, generalized sluggishness, slurred speech, and gross ascites. Blood pressure remained elevated and was managed with oral nifedipine retard and alpha methyldopa while diuretics (frusemide and aldactone) were prescribed for generalized edema and ascites. Investigations in the course of admission are reported in [Table 2]. Ascitc fluid was exudative.

She had a repeat TFT, 6 weeks after the first, which was still in keeping with subclinical hypothyroidism [Table 1]. She was subsequently commenced on levothyroxine at 100 μg per day. Follow-up reviews showed marked reduction in abdominal girth and weight despite significant reduction in the dosage of diuretics prescribed [Table 3].

She was discharged home on the 20th day of admission with no pedal edema and minimal ascites. Medications at discharge included oral levothyroxine 100 μg daily, nifedipine 40 mg b.d, alpha-methyldopa 500 mg b.d and slow k on account of persistent hypokalemia.

She was reviewed at the medical outpatient clinic 34 days after discharge with history of poor adherence to her medications for a few days prior to that visit and poor BP control. Ascites had completely resolved clinically and radiologically, while TFT result showed reduction in TSH value. She however did not return for follow-up visits.

Discussion

It appears that recognition of subclinical hypothyroidism and hypothyroidism, when the initial presentation is ascites may be difficult. This is because hypothyroidism accounts for only 1% of patients presenting with ascites.

Table 1: Thyroid function tests

From referring hospital	Day 4 on admission	Outpatient visit
T3-2.4 (0.5-1.88) ng/ml	FT3-3.6 (3.8-6.0) pmol/l	FT3-4.2 (3.8-6.0) pmol/l
T4-112 (59-153) nmol/l	FT4-12.8 (7.2-16.4) pmol/l	FT4-9.0 (7.2-16.4) pmol/l
TSH-20.5 (0.4-4.0) IU/ml	TSH-14.35 (0.37-3.50) mIU/l	TSH-8.64 (0.37-3.50) mIU/l

T3: Triiodothronine, T4: Thyroxine, FT3: Free triiodothyronine, FT4: Free Thyroxine, TSH: Thyroid stimulating hormone

Table 2: Investigation results

Urinalysis			
	Day 0	**Day 9**	**Day 13**
Uro	Normal	Normal	Normal
Protein	++	+++	Negative
Blood	+	+	++
SG	1.015	1.020	1.015
Ketones	Negative	Negative	Negative
Bilirubin	Negative	Negative	Negative
Glucose	Negative	Negative	Negative
Leukocytes	Negative	Negative	++
Liver function test			
Conjugated bilirubin	1.0 mg/dl		
ALP	141 IU/L		
AST	47 IU/L		
ALT	42 IU/L		
Total protein	5.8 g/dl		
Albumin	3.1 g/dl		
Ascitic fluid protein	5.1 g/dl		
Coagulation profile			
	PTT	aPTT	
Test (s)	15	35	
Control (s)	14	33	
INR	1.08		
Electrolytes, urea, and creatinine	**Day 1**	**Day 2**	**Day 7**
Na	132 mmol/l	130 mmol/l	
K	2.6 mmol/l	2.5 mmol/l	2.8 mmol/l
Cl	99 mmol/l	99 mmol/l	
HCO_3	18 mmol/l	17 mmol/l	
Urea	32 mg/dl	60 mg/dl	24 mg/dl
Creatinine	1.0 mg/dl		0.7 mg/dl
Full blood count			
PCV	34%		
WBC	8000/mm^3		
Platelet	173,000		
Neutrophils	56%		
Eosiniphils	0%		
Lymphocytes	33%		
Monocytes	10%		

Uro: Urobillogen, SG: Specific gravity, ALP: Alkaline phosphatase, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, INR: International normalized ratio, PCV: Packed cell volume, WBC: White blood cell, PTT: Partial thromboplastin time, aPTT: Activated PTT

Table 3: Anthropometric measurements

Date	Day 7	Day 12	Day 15	Day 17
Weight (kg)	87	81	65	65
Abdominal girth (cm)	117	109	90	85

Subclinical hypothyroidism is often caused by chronic autoimmune thyroiditis with a prevalence of 3-8% in the general population.[2] The prevalence is said to be higher in women and increases with age.[2]

The patient presented in this case was much younger than those in earlier reports whose findings were mainly in middle aged or elderly persons with hypothyroidism. Some cases have reported a past history of thyroid ablation without thyroid hormone treatment in such persons before the onset of ascites. However, the patient discussed here had no history suggestive of a thyroid dysfunction in the past except for a transient goiter. The first case of ascites associated with hypothyroidism was reported by Paddock in a 48-year-old deaf lady in 1950.[3] Since then about 62 articles have been published as case reports with the same. Common findings in previous cases with ascites and hypothyroidism included long duration of the ascites before diagnosis, resolution of ascites with thyroid hormone replacement, high total protein concentration in ascitic fluid, and a high serum-ascites albumin gradient some of which were present in this patient.[4] To our knowledge, this report is the first of subclinical hypothyroidism in a patient with gross ascites and preeclampsia. Persons with preeclampsia have been found to have generalized edema and ascites howbeit uncommon. The ascitic fluid has been found to be usually transudative in such patients rather than exudative as found in this case. This may suggest that the sub-clinical hypothyroidism may be responsible for ascites in this case rather than preeclampsia. Following the commencement of Levothyroxine, blood pressure control improved with gradual decrease in the dose of her antihypertensives, protenuria and ascites also resolved, resulting in gradual withdrawal of diuretics. These features were also sustained during her outpatient visit. Many previous studies have also documented similar resolution of symptoms with treatment, corroborating the finding that replacement of thyroxine restores the impairment of endothelium-derived vasodilation from diminished nitric oxide.[5-7] It is recommended that all patients with overt hypothyroidism and subclinical hypothyroidism with TSH >10 mIU/l should be treated.[2] It is also very important to treat subclinical hypothyroidism in pregnant women and women contemplating pregnancy, in order to decrease the risk of pregnancy complications and impaired cognitive development of their offspring.[8] It has been postulated that hypothyroidism may cause an increase in capillary permeability, resulting in the flow of protein-rich fluid into the extravascular space, producing ascites which is exudative.[9] Some authors have also suggested that this ascites may be due to increased interstitial oncotic pressure and inappropriate secretion of antidiuretic hormone. The reasons for accumulation in the intra-abdominal space is however not clear. The mechanism of ascites in SCH may be similar to that in hypothyroidism.

Detailed clinical evaluation including TFT should be performed in all patients with exudative ascites of unknown etiology. This will aid early diagnosis and avoid invasive and expensive investigations in this group of patients. It appears that subclinical hypothyroidism can result in ascites similar to that reported in hypothyroidism and should be corrected promptly to aid prompt resolution of ascites, thus preventing complications. Pregnant women with severe preeclampsia should be screened for SCH since an association has been found between severe preeclampsia and SCH.[10]

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