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Intracerebral Bleeding and Multiple Body Cavity Effusions as Presenting Symptoms of Hypothyroidism

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Abstract: *Hypothyroidism can present with a wide spectrum of presenting symptoms. Patients can have symptoms like fatigue/ lethargy, weight gain, cold intolerance, constipation, menstrual abnormalities in women which are commonly seen and also rare modes of presentation like cerebrovascular accident, isolated effusion (ascites, pleural effusion, pericardial effusion) or in combination of multiple body cavity effusions along with tissue oedema. They can also present with no symptoms at all. According to the literature, the association of hypothyroidism with minor bleeding events (gum bleeding, bruises, and menorrhagia) is widely known, but only a few reports of hypothyroidism as a risk factor for intracerebral bleeding are published. Very few cases have been reported in the literature about the presence of ascites, pericardial effusion and pleural effusion as a separate entity in hypothyroidism or a combination of any two,but the combination of all these findingson presentation in a case of hypothyroidismis extremely rare. We present a 40 year old female patient who presented to the emergency department in a comatose state. On detailed history and examination, intracerebral haemorrhage was found on computer tomography along with pericardial effusion on 2-D Echocardiography, pleural effusion on chest X-ray and ascitis on ultrasound.Blood investigations showed Thyroid Stimulating Hormone(TSH) levels to be markedly raised (>100). She had no known co-morbidities.*

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I. Introduction

Body cavity effusion in isolation such as pericardial effusion (PE), pleural effusion or ascitis in the setting of hypothyroidism is commonly seen.Ascitis is seen < 4% of cases of hypothyroidism[1]. Incidence of Pericardial effusion is 3-6% [2]. Intracerebral hemorrhages among hypothyroid patients are also extremely rare. Multiple body effusions and intracerebral hemorrhage all occurring concurrently due to hypothyroidism is extremely rare. We present a case who developed intracerebral hemorrhage, pericardial effusion with cardiac tamponade, ascitis , pleural effusion simultaneously.

II. Case Report

A 40 year old female patient was brought to the casualty in a drowsy and disoriented state to the casualty with history of reduced appetite, one episode of generalized tonic clonic seizure and drowsiness for one day. Patient also had one episode of generalized tonic clonic seizure in the casualty on presentation. According to the history provided by the patient's relative, she had history of facial puffiness(Figure 1) for the past one month. She had no history of dyspnoea,

Chest pain, paroxysmal nocturnal dyspnoea, decreased urine output. She was not a known case of Hypertension, Diabetes Mellitus, Ischemic Heart Disease or Seizure Disorder. She had history of hysterectomy 10 years back. On examination, the patient was afebrile with a pulse rate of 90/min and a blood pressure of 180/110mmHg. There was mild periorbital edema with facial puffiness. Oxygen saturation was 78% on room air and was subsequently mechanically ventilated. On Central Nervous system examination, bilateral pupils were reactive to light. Deep tendon reflexes were brisk on the left side and normal on the right side. Left plantar reflex was absent. On Cardiovascular system examination, there were muffled heart sounds. Gastrointestinal system examination findings were suggestive of Ascitis. On admission, laboratory data showed significantly lowered levels of potassium (2.9 mmol/L) and slightly elevated total bilirubin (1.95). The coagulation panel revealed a slightly increased International normalized ratio (INR: 1.51). The complete blood count showed a microcytic hypochromic anemia. (Haemoglobin 8.3g/dl). Thyroid function tests showed markedly raised Thyroid stimulating hormone (TSH > 100) and low levels of T3 and T4 hormones. The remaining results were unremarkable including normal renal function tests and urine analysis. 12 lead electrocardiogram(ECG) showed

normal sinus rhythm with bradycardia(63 bpm)and low voltage QRS complex(Figure 2).Chest radiograph revealed massive cardiomegaly (Figure 3). A computer tomography was done, which showed a 7.4 x 3.1 x 7 cm sized hyperdensity within the dilated right lateral ventricle involving the frontal horn, body, temporal horn and occipital horn suggestive of intraventricular haemorrhage with surrounding perilesional edema in the white matter (Figure 4). 2-D Echocardiograghy revealed concentric left ventricular hypertrophy and large pericardial effusion.Ejection fraction was 60 %(Figure 5). Pericardiocentesis was done and 200ml fluid was removed (Protein 5.7 g/dl, Sugar 103.1mg/dl, ADA 4.3 U/L, LDH 283 U/L,Total count 200cells/cu.mm, RBC 100 cells/cu.mm).Ultrasonography abdomen showed moderate ascitis with bilateral renal calculi. Diagnostic paracentesis was done (Proteins 4.19 g/dl, Sugar 103.4 mg/dl, ADA 4.5 U/L).

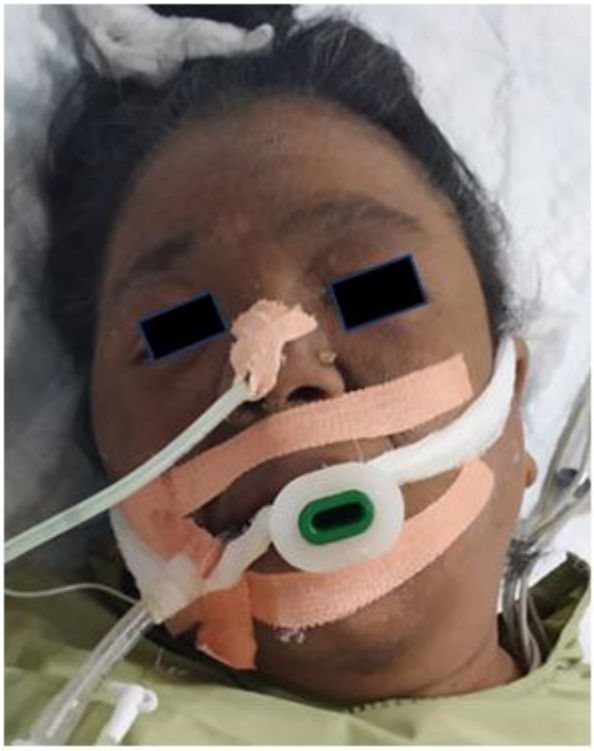

Figure 1: *Facial puffiness*

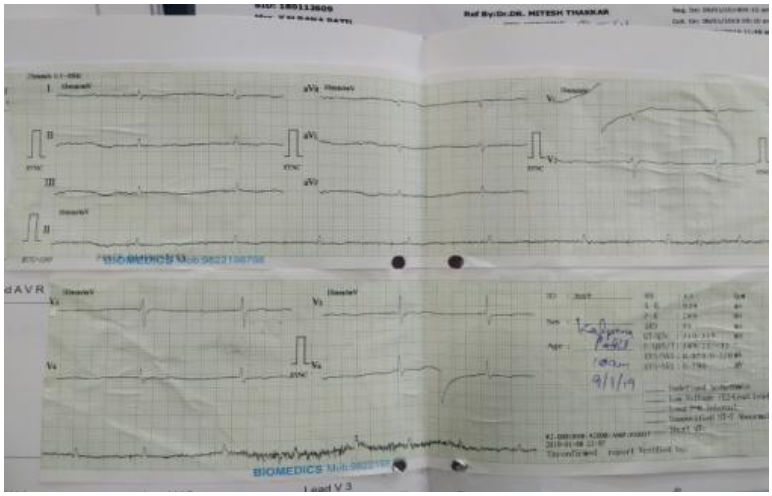

Figure 2: *ECG showing low voltage QRS with bradycardia*

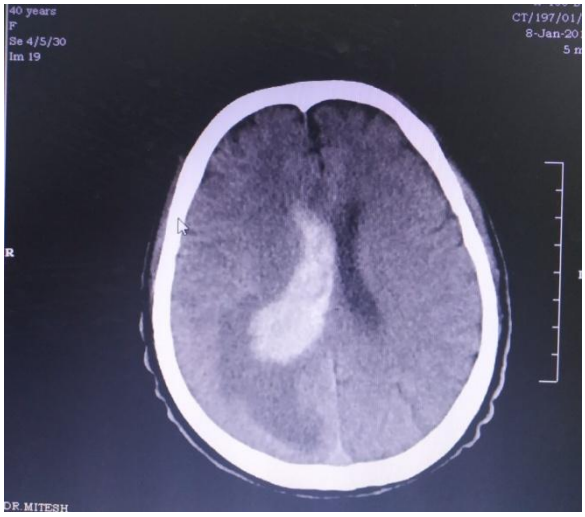

Figure 4: *Intraventricular hemorrhage involving right ventricle with perilesional white matter edema*

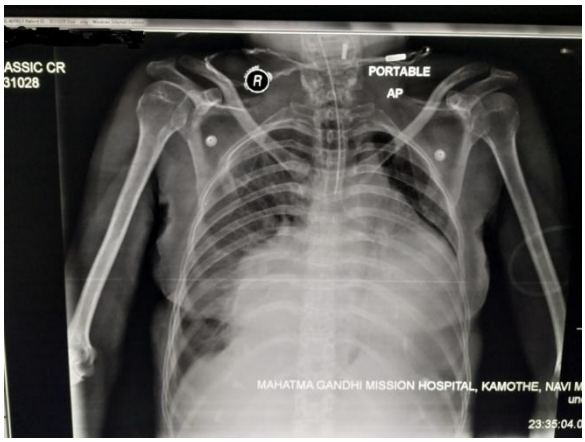

Figure 3: *Chest Radiograph(A/P)showing massive cardiomegaly*

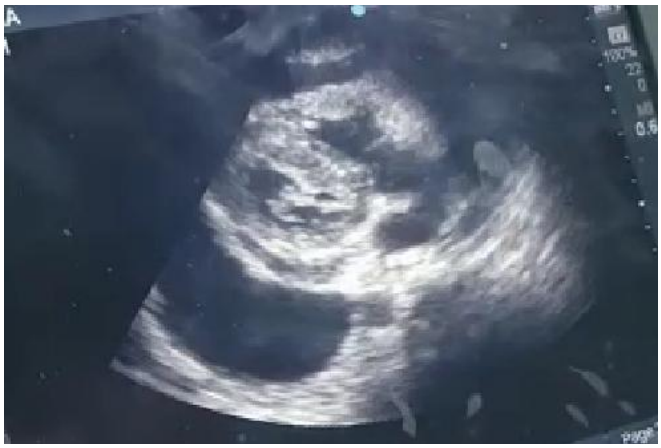

Figure 5: *2-D Echo showing massive pericardial effusion*

III. Discussion

Body cavity effusion in isolation such as pericardial effusion (PE), pleural effusion or ascitis in the setting of hypothyroidism is commonly seen. Pericardial effusion due to hypothyroidism was reported as early as 1918[3] and subsequently in 1925[4]; however, cardiac tamponade due to hypothyroidism is very uncommon. The incidence of pericardial effusion wasreported to be between 30 and 80% in the earlier studies. However, recently, this has been refuted by Kabadi *et al* [2] wherein the incidence was found to be 3% to 6 %. This is because the subjects studied in the old literature were severely hypothyroid at the time of the study, reflecting delayed diagnosis of hypothyroidism. Thus PE may be a frequent manifestation in myxoedema (advanced

severe stage) but a rare manifestation in hypothyroidism (early mild stage), as the latter condition nowadays is detected in the very early stage.

Acute hemorrhagic stroke is a leading cause of mortality and morbidity in adults. Thyroid dysfunction along with hypertension, atherosclerosis, diabetes mellitus, are identified as risk factors in the etiology of stroke. Thyroid hormones, in addition to their role in cellular metabolic activity also regulate neural development. The CNS is particularly dependent on thyroid hormone for normal maturation and function. Hypothyroidism has been shown to be neuroprotective in stroke patients. In a detailed study on 744 acute stroke patients having a median age of 70years, it was observed that hypothyroid patients had a better survival at follow-up [5]. Similarly Baek et al. [6] also found that acute ischemic patients with subclinical hypothyroidism (SCH) were more likely to show functional outcomes than those without SCH. ApurvaPande et all[7] in 2016 observed high rates of mortality involving patients of acute hemorrhagic stroke with low free iodothyronine (FT3) and free thyroxine (FT4).However, multiple body cavity effusions along with intracranial hemorrhage in hypothyroidism are extremely uncommon, which was seen in our patient.

The accepted pathogenesis of multiple effusions in hypothyroidism is [8]i) Generalized polyserositis. ii) Leak of plasma proteins because of abnormal capillary permeability and the lack of a compensatory increase in lymph flow and protein return rate. iii) Alteration in albumin metabolism. iv) Mucopolysaccharide and protein deposition. v) Cardiac tamponade causing congestive heart failure.

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