A Rare Case of Hepatitis E Induced Viral Thyroiditis

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**Abstract**

We report a one of its kind case of a 26-year-old Gentleman, who had initially presented to our hospital with acute symptoms of viral hepatitis. Initial laboratory studies done revealed an elevated liver function test with an elevated Anti-HEV serology and thus the patient was diagnosed and managed for Viral Hepatitis E. The patient had no prior history of any co-morbid conditions but was found to have an elevated TSH and symptomatic features that were highly suggestive of an subacute viral granulomatous thyroiditis, which is a rare and unheard presentation with acute Hep E viral symptomatology. We further concluded that the Viral Hepatitis E was the most likely culprit responsible for the Subacute Inflammatory Thyroiditis and the elevated TSH seen on the blood work up. This is a very rare and odd, unknown presentation of Hepatitis E virus.

**Introduction**

The most common cause of subacute granulomatous thyroiditis is usually a viral Infection of the thyroid gland [1]. In numerous studies various viral etiologies have been identified and implicated to cause subacute thyroiditis. Subacute Viral Thyroiditis is often preceded after an upper respiratory tract infections and is normally seen in the post convalescent phase. The most common etiological viruses to have been implicated to cause subacute granulomatous thyroiditis are the influenza, adenovirus, mumps and coxsackie virus.

We report a one of its kind case of a 26-year-old Gentleman, who had initially presented to our hospital with acute symptoms of viral hepatitis and later on went to develop subacute viral thyroiditis.

**Case Report.**

A 26 year old, non-alcoholic gentleman with no significant past medical history presented to our hospital with an acute onset jaundice that was progressive over the past ten days and was accompanied with exertional breathlessness that had begun five days back and was persistent. He also complained of other nonspecific symptoms like anorexia, mild itching and the passage of white colored stools. He denied any history of fever or abdominal discomfort or pain. He had never received any blood transfusion neither had he undergone any surgery in the past, the rest of the history was unremarkable. On physical examination there was scleral Icterus noted on the HEENT examination and mild hepatic enlargement on abdominal examination. The rest of the physical examination was unremarkable. Initial blood investigations revealed a hemoglobin of 11.5 gm% (12-16), total leucocyte count 8300 /c.mm (4000-11000) with 58% neutrophillic predominance, platelets of 3.35 lacs/c.mm (1.5-4.5).

The Liver function tests showed a total bilirubin of 19.7 mg/dl (0.2-1.2), direct bilirubin 14.2 mg/dl (0.0-0.8), indirect bilirubin 5.5mg/dl (0.3-0.8), ALT 148 u/l (0-49), AST 127 u/l (0-46), alkaline phosphatase 136 u/l (40-129), total protein 8.6 gm/dl (6.2-8.3) and albumin 4.2 gm/dl (3.5-5.1). Prothrombin time was 13.6 seconds (11-15). Imagining was done with an ultrasound abdomen which was normal. Hepatic Viral markers were sent and serology for HEV IgM was positive, HAV IgM was negative and anti HCV was negative too. HBsAg tilters were noted to be positive with negative IgM, anti Hbc, HBeAg, HBeAb and HBV DNA < 100 copies/ml which was indicative of a carrier state of hepatitis B. He was hospitalized for further management.

It was noted over the ensuing days that the patient was having a progressive increase in his breathlessness which was now associated with a non-productive cough that had become persistent at 10 day’s of hospitalization. His routine daily examination revealed a sustained tachycardia with a pulse rate overtly being around 120 beats per min and subsequently an ECG was done that revealed sinus tachycardia. In view of this, an ECHO and Chest X ray were ordered and turned out to be normal. Follow up Liver function tests showed improvement with total bilirubin 10 mg/dl (0.2-1.2), direct bilirubin 6.5 mg/dl (0.0-0.8), indirect bilirubin 3.5mg/dl (0.3-0.8), ALT 118 u/l (0-49), AST 122 u/l (0-46), alkaline phosphatase 137 u/l (40-129), total protein 8.8 gm/dl (6.2-8.3) and albumin 4.6 gm/dl (3.5-5.1). A Thyroid function test was ordered at that time and revealed hyperthyroidism, with Free T3 7.50 pmol/l (3.1-6.8), Free T4 30.10 pmol/l (12-22) and TSH 0.06 uIU/ml (0.27-4.2). A NM Scan Thyroid and Uptake findings were suggestive of thyroiditis with 30 minutes uptake 0.2 % (0.5-5 %).
Further thyroid studies with thyroglobulin antibodies and TPO antibodies were done which were negative. At this point he was diagnosed to be having a post viral thyroiditis due to Hepatitis E and was managed conservatively.

He was followed up in the outpatient department and Six weeks later a Liver function test was repeated, that revealed a marked improvement with a total bilirubin 4 mg/dl (0.2-1.2), direct bilirubin 2.2 mg/dl (0.0-0.8), and indirect bilirubin 1.8 mg/dl (0.3-0.8), ALT 58 u/l (0-49), AST 54 u/l (0-46). Repeat Thyroid profile showed Free T3 7.1 pmol/l (3.1-6.8), Free T4 24.8 pmol/l (12-22) and TSH 0.11 uIU/ml (0.27-4.2). He was further managed symptomatically and was regularly followed up in the outpatient department. He was noted to have significant clinical improvement. A serial Liver function test was followed up monthly that normalized over a two month period and so did the thyroid function test that normalized over a four month period. At present the patient is asymptomatic and has improved completely.

Discussion.

Our Search was futile to find any case report to date that has been published, to Implicate Hep E to be a causative agent for viral associated thyroiditis however numerous case reports have been published and studies done on Hep C induced viral thyroiditis.

Thyroid involvement with HCV infected subjects has been seen with variable frequency [2]. Antonelli and Ferri have reported in their study an increased prevalence of papillary thyroid cancer in patients with hepatitis C [3]. Deutsch [4] reported a higher baseline prevalence of autoimmune thyroiditis in patients known to have been suffering from chronic hepatitis C, especially among female patients and thus it became prudent for female patients with chronic hepatitis C patients to tested for anti-thyroid peroxidase antibodies (ATPO) and to have their thyroid function monitored with TSH and FT4 levels during and after IFN–alpha therapy for chronic Hep C.

An association between HCV infection and thyroid abnormalities, especially autoimmune thyroiditis, had been reported and studied especially on the basis of high prevalence rates of anti HCV among patients with Hashimoto’s disease as well as of thyroid auto antibodies and/or thyroid dysfunction among patients with chronic hepatitis C [5].

Our patient had initially presented with symptoms suggestive of an acute presentation of viral hepatitis and further serologic studies done revealed acute hepatitis E infection. The cause of breathlessness was presumed to be due to his underlying viral thyroiditis that resolved with improvement with his thyroiditis. His nonproductive cough was hypothesized to be caused due to irritation of the recurrent laryngeal nerve due to the underlying viral inflammatory process near the thyroid gland that improved with resolution of the thyroiditis. Earlier studies have never documented hepatitis E virus as a causative agent for thyroiditis or have noted any association between the two infections. The classical timing of the viral stage and the subsequent improvement in thyroiditis over the ensuing months with improvement with hepatic function with no residual thyroid deficits clearly established Hep E induced viral thyroiditis a rare and unknown and undocumented case so far as a cause of thyroiditis.

References.


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