

De Quervain thyroiditis in the course of H1N1 influenza infection

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Abstract

Background/aim: Viral infections have been frequently associated with subacute (De Quervain) thyroiditis and autoimmune thyroid diseases. In the present case report we document a rare case of De Quervain thyroiditis in the course of H1N1 influenza infection.

Description of the case: A 17-year-old previously healthy female that was treated in the General Hospital of Kalamata developed an influenza-like syndrome that was accompanied by palpitations, thyroid enlargement, and increased C-reactive protein. Polymerase chain reaction assay confirmed the diagnosis of H1N1 virus infection. Serum thyroid-stimulating hormone was suppressed to zero while the levels of free thyroxine and triiodothyronine were increased. The patient was treated with non-steroidal anti-inflammatory drugs and thyroid function was gradually restored without evolving to a hypothyroid phase.

Conclusion: To our knowledge this is the second case described in the literature of De Quervain thyroiditis associated with H1N1 influenza infection. Hippokratia 2014; 18 (1): 86-87.

Keywords: H1N1 virus, De Quervain thyroiditis

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Introduction

Viral infections are frequently implicated in subacute (De Quervain) thyroiditis and autoimmune thyroid diseases¹. A few years ago, Dimos et al published a rare case of De Quervain thyroiditis in the course of acute novel H1N1 influenza infection². This was the first case report of this kind in the literature. We came across a similar case in the Internal Medicine Department at the General Hospital of Kalamata, Greece. The patient was diagnosed with acute novel H1N1 influenza infection when evaluated for a 4-day history of gradually worsening fever, and sore throat.

Description of case

A 17-year-old, otherwise healthy female, was admitted to the hospital complaining of gradually worsening fever over 4 days and sore throat. The patient's symptoms were initially attributed to pharyngitis by her primary care physician, and she was already being treated with amoxicillin / clavulanic acid when she was admitted to the hospital. The patient was not a smoker and did not mention any previous health problems. Physical examination revealed tachycardia (110 beats per minute); the patient denied the presence of palpitations in the past. Her blood pressure was within normal range (110/70 mmHg), while her temperature was 38.9 °C. Palpation of the mildly enlarged thyroid gland did not elicit significant patient discomfort.

The thyroid was soft, with no alterations of the overlying skin and there were no nodules felt. The chest was clear and the abdomen was soft to palpation. Chest x-ray and ECG were negative for pathological findings. The diagnosis of H1N1 viral infection was based on polymerase chain reaction testing of nasopharyngeal / oropharyngeal swabs by the reference center located in the University of Athens, Medical School.

Laboratory workup showed significantly increased levels of triiodothyronine (T3) 3.79 ng/ml [normal values (NV) 0.6-1.85 ng/ml], free triiodothyronine (FT3) 11.60 pg/ml (NV: 2.3-4.2 pg/ml), thyroxine (T4) 20.37 µg/dL (NV: 4.5-11 µg/dL), and free thyroxine (FT4) 5.24 ng/dL (NV: 0.8-1.8 ng/dL), while thyroid-stimulating hormone (TSH) levels were suppressed to almost zero (0.01 mIU/L, NV: 0.5-5 mIU/L). Thyroid autoantibodies (thyroglobulin and thyroid peroxidase antibodies) were negative. Erythrocyte sedimentation rate (ESR) was 110 mm/h (NV: 0-20 mm/h) and C - reactive protein levels were elevated (CRP: 11.41 mg/dl, NV: <0.5 mg/dl). Ultrasound of the thyroid that was performed during the acute phase showed a diffuse inhomogeneous increase in size of the right lobe of the thyroid gland. Non-steroidal anti-inflammatory drugs were prescribed and the patient was followed-up weekly with clinical evaluation of thyroid tenderness and measurement of thyroid hormone levels.

Technetium-99m-pertechnetate scintigraphy a month following the initial infection showed diffuse and homogeneous high technetium trapping. Thyroid levels were restored and the patient did not progress to a hypothyroid phase.

Discussion

De Quervain thyroiditis is an uncommon cause of hyperthyroidism and affects women more often than men³. Subacute thyroiditis is presumed to be caused by a viral infection or a post-viral inflammatory process and most patients have a history of an upper respiratory infection prior to the onset of thyroiditis (typically two to eight weeks). Clusters of cases have been reported in association with Coxsackievirus, EBV, mumps, measles, adenovirus, influenza and other viral infections³. The thyroid injury in subacute thyroiditis is considered to be the result of cytolytic T-cell recognition of viral and cell antigens present in an appropriate complex^{3,4}.

This seems to be, to our knowledge, the second case of subacute thyroiditis related to H1N1 influenza virus. It is worth noting that, as with the first case report, the viral infection was active and well documented. As far as the involvement of thyroid gland during viral infections is concerned, there is controversy regarding the exact nature of their possible relation³. In many cases of thyroiditis the involvement of a viral infection was based on serology, i.e., on the presence of antibodies, which constitutes an indication of recent or past infection⁵. In the present case, the documentation of the presence of H1N1 virus by polymerase chain reaction assay indicates the active form of the infection, and reinforces the etiopathogenetic role of influenza virus infection in the development of subacute thyroiditis.

It is interesting to mention that, in the literature, cases have been reported of thyroid storm triggered by H1N1 infection⁶. Furthermore, recently, there was a case report of subacute thyroiditis following the H1N1 vaccine⁷. The fact that a second case of H1N1 virus-induced subacute thyroiditis has been documented should make physicians

aware of a possible involvement of the thyroid gland as subacute thyroiditis when managing patients with flu-like symptoms. These two reported cases along with the documentation of H1N1 infection-induced thyroid storm support the hypothesis that the disorder of thyroid gland could be well included in the cluster of clinical complications that can occur in the course of H1N1 infection. Therefore, physicians should keep this complication in mind when assessing cases with H1N1 infection in order not to misattribute thyroid-related symptoms to the pharyngitis that is often also present in such cases. Further studies should be conducted in order to clarify the exact pathophysiological mechanisms implicated in the association of H1N1 infection and subacute thyroiditis.

Conflict of interest

The authors have nothing to disclose

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