Nebojsa Paunkovic, Jane Paunkovic

TRANSITORY THYROTOXICOSIS DUE TO HEMORRHAGIC THYROID INFARCTION DURING MULTIPLE TROMBOEMBOLIC DISEASE – a Case Report

Abstract: An elderly female patient with a painful thyroid nodule and thyrotoxicosis was referred the Out-patient thyroid clinic. Few days before referral patient was treated in the General hospital for an acute embolic obstruction of left radial artery, and few days after recorded thyroid events she suffered an ischemic cerebral stroke. Patient previously had a dilatative cardiomyopathy with absolute arrhythmia and consecutive events were considered as tromboembolic obstructions of arteries corresponding organs (arm, thyroid, brain). The results of diagnostic procedures and the clinical course of disease are presented as a case report. Authors highlight that authentic occlusive thyroid infarctions are seldom reported in the medical literature.

Key words: Transitory thyrotoxicosis, Tromboebolic disease, Hemorrhagic infaction.

Introduction

The hemorrhagic infarction of thyroid gland is a focal necrotic process caused by inadequate blood supply. The cause of this arterial occlusion may be tromboembolic mass. As a consequence of this necrosis thyroid hormones are released into the blood stream and may cause hyperthyroid state.

We had an occasional opportunity to observe a female patient with previously diagnosed multinodular euthyroid goiter. Prior to admission in the out-patient clinic she suffered acute tromboembolic attack of left radial artery with near total obstruction. Few days later new thyroid nodule and the increase of thyroid hormones in blood were diagnosed.

Case Presentation

A 67-year-old woman was referred from the hospital where she was treated for occlusion of left radial artery for the consultation on the status of her thyroid disease. She was previously registered at the out-patient Thyroid clinic with euthyroid multinodular goiter. During the treatment for arterial occlusion at the hospital, manifestation new thyroid nodule and an increase of thyroid hormones in blood were recorded.

At presentation patient was slightly hypermetabolic, with absolute arrhythmia. The pulse on left radial artery was almost non palpable. Recently manifested nodule in

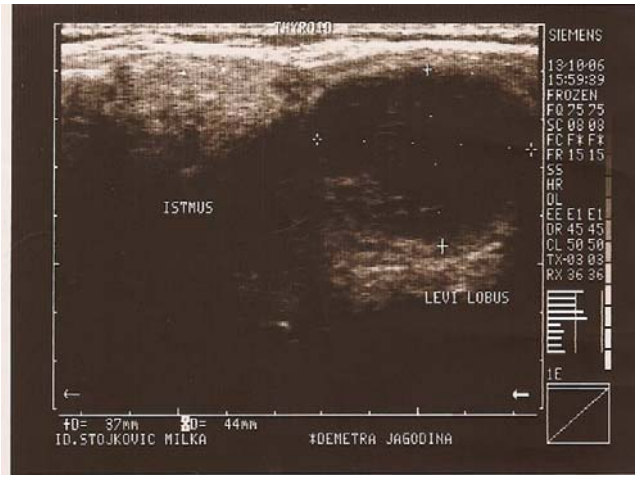


Figure 1. Thyroid ultrasonography at presentation

Figure 2. Thyroid scintiscan at presentation

the left thyroid lobe was painful. We have completed ultrasonography and scintigraphy of thyroid. The nodule was hypoechogenic and cold (figure 1 and 2). A fine needle biopsy has provided few milliliters of destructed tissue and hematinized blood. We have also completed hormonal and immunological tests: FT4 was increased, „ultrasensitive" TSH suppressed and hTRAb negative. Follow-up of FT4 concentration in blood is presented on graph 1.

Upon the completion of the diagnostic procedures we have assumed that patient probably has a hemorrhage in thyroid tissue secondary to anticoagulant therapy prescribed in the hospital for radial artery embolism. For mild thyrotoxic process caused by destruction of the thyroid tissue we have prescribed only symptomatic therapy.

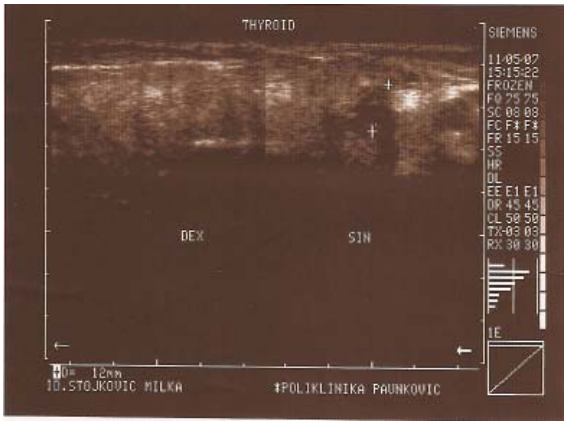
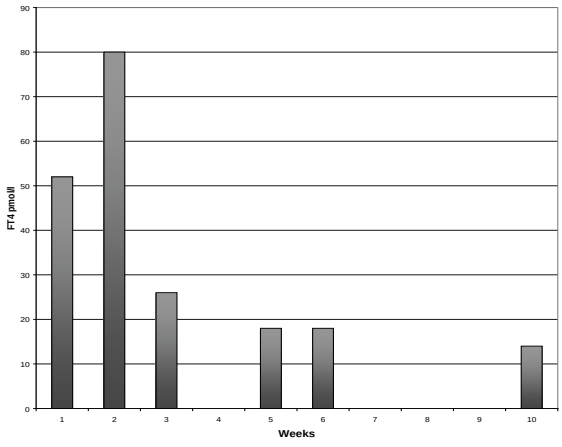


Figure 3. Thyroid ultrasonography 6 weeks after presentation



Graph 1. Follow-up of FT4 concentration during tromboembolic disease

Few days later patient suffered a cerebral ischemic stroke, with right hemiplegia. Anticoagulant therapy with strictly controlled coagulant parameters was continued in intensive care hospital unit and recovery with mild hemiparesis was attained.

Patient was under regular supervision by our thyroid specialist in the following weeks. Fine needle aspiration of nodule was repeated twice, and blood concentrations of thyroid hormones and TSH (graph 1) was regularly performed. Few weeks later nodule was almost in complete recovery (figure 3).

The hemorrhagic infarction of thyroid gland has been reported frequently in the literature [1-7]. Most of these cases were related to hemorrhagic necrosis caused by trauma [1], anticoagulant therapy [3,4] or aspiration biopsy [5,6,7]. Reports of the real infarction of the thyroid (necrosis caused by nutritional artery obstruction) are rare.

Discussion

Thyrotoxic syndrome (increased thyroid hormones concentrations with consecutive hypermetabolism) is frequently a consequence of thyroid hyperfunction (hyperthyroidism), and rarely caused by the thyroid tissue destruction (predominantly subacute de Quervaine's thyroiditis) We have reported a case of a female patient previously diagnosed as multinodular euthyroid goiter for many years, with novel manifestation of thyroid nodule and thyrotoxicosis in the course of acute tromboembolic disease.

We have presented a female patient with previously existent cardiac and thyroid diseases and multiple tromboembolic complications in the left branches of aortic arc. Consecutive clinical manifestations related to the thyroid included new thyroid nodule and transitory thyrotoxicosis.

Post-festum we have concluded that our patient with previously existent multinodular euthyroid goiter and dilatation of left heart chamber and arrhythmia have suffered trombotic process in left heart chamber with embolic complications on left radial artery, left thyroid lobe and left cerebral hemisphere. The consecutive pathologic conditions included ischemic disease of left arm, thyroid hemorrhagic infarct and ischemic cerebral infarct. The clinical manifestations were ischemic pain in left arm, manifestation of the new painful thyroid nodule and thyrotoxicosis, and right hemiplegia. Patients have recoverd after few weeks. Six months later our patient has absolute arrhythmia, multinodular euthyroid goiter and mild right hemiparesis and she is under constant anticoagulant treatment.

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