Pregnancy-triggered triple autoimmunity (Hashimoto’s thyroiditis, antiphospholipid syndrome and systemic lupus erythematosus)

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Abstract

Introduction
We describe a case of 22 year old female with her first pregnancy triggered Hashimoto’s thyroiditis (HT), Antiphospholipid Syndrome (APS) and Systemic Lupus Erythematosus (SLE).

Case Report
A 22 year old female was diagnosed with HT on levothyroxine during the early first trimester. During 21 weeks of gestation patient has intrauterine fetal demise and underwent medical abortion. SLE work up including antinuclear antibody and anti-double stranded DNA were positive. She underwent kidney biopsy, which revealed membranous and mesangial proliferative lupus nephritis. Diagnosis of SLE and APS was made. Treatment with anticoagulation therapy was started. SLE therapy was initiated with prednisone, mycophenolate mofetil and hydroxychloroquine with complete resolution of symptoms.

Discussion
APS is a prothrombotic disorder with various manifestations, most commonly venous and arterial thromboembolism and recurrent pregnancy loss. Pregnancy may trigger an underlying APS, which may well be the causative for the miscarriage. New onset SLE during pregnancy is rare. However, in our case, the anemia, thrombocytopenia, and proteinuria led us to the correct diagnosis of SLE. HT is associated with higher rates of infertility and early miscarriages, due to the associated hormonal changes and instability. However, the association of APS and HT is not well recognized in pregnant women.

Conclusion
We present here a challenging case of new-onset triple autoimmune disorders trigged by pregnancy. Clinicians should be aware of this association and initiate early autoimmune work up for SLE and APS in patients with new onset of HT during pregnancy.

A successful neonatal and maternal outcome was achieved in this case. The patient's history revealed membranous MGN stage II-III. Herein, we present a case of successful pregnancy and foetal outcome in a young woman with APLA syndrome and MN.

Keywords
Pregnancy triggered Hashimoto’s thyroiditis, Antiphospholipid Syndrome and Systemic Lupus Erythematosus.

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Introduction
Although the association between autoimmune thyroid and rheumatic disorders has been studied in nonpregnant women and there are no data on the frequency of this association during pregnancy and its impact on reproductive outcomes. We present a case of 22 year old female with her first pregnancy triggered Hashimoto’s thyroiditis (HT), Antiphospholipid Syndrome (APS) and Systemic Lupus Erythematosus (SLE).

Case Report
A 22 year old female diagnosed with HT during the early first trimester. Her thyroid work up include anti-thyroid peroxidase antibody was positive, she was stated on levothyroxine. She was admitted at 21 weeks of gestation for labor induction secondary to intrauterine fetal demise and underwent medical abortion. Laboratory results was significant for thrombocytopenia, prolongation activated partial thromboplastin time, positive IgG and IgM anticardiolipin antibodies, anti-beta2- glycoprotein I and lupus anticoagulant. Placental pathology showed placental infarcts with hypoxia ischemic changes. Due to suspicion of APS and therefore risk of thromboembolism, the patient was started on prophylactic Lovenox 40mg SC daily. She presented to the emergency room 4 weeks later with sudden onset of focal neurologic deficit. Computerized tomography angiogram showed distal right middle cerebral artery segment M1 occlusion. Patient was started on therapeutic anticoagulation and focal weakness was resolved in 5 days. SLE work up initiated, antinuclear antibody and anti-double stranded DNA were positive. Anti-smith antibody, anti-RNP antibody, anti-Ro, anti-La antibodies were reported negative with normal C3 and C4 complement levels. 24hr urine protein was between 1.56 and 2gm. She underwent kidney biopsy, which revealed membranous and mesangial proliferative lupus nephritis, ISA/RPS class V and II. Diagnosis of SLE and APS was made. Treatment with anticoagulation therapy was started with warfarin and aspirin. SLE therapy was initiated with prednisone, mycophenolate mofetil and hydroxychloroquine with complete resolution of proteinuria. For HT levothyroxine was adjusted.

Discussion
APS is a prothrombotic disorder with various manifestations, most commonly venous and arterial thromboembolism and recurrent pregnancy loss. Diagnosis of APS can be challenging due to evolving criteria and overlapping characteristics with other prothrombotic thrombocytopenic disorders. Thrombotic complications within the uteroplacental circulation has also been proposed as a contributing mechanism. Pregnancy may trigger an underlying APS, which may well be the causative for the miscarriage [1-3]. In our case given the placental findings the fetal death was due to APS. New onset SLE during pregnancy is rare. However, in our case, the anemia, thrombocytopenia, and proteinuria led us to the correct diagnosis of SLE. When SLE is first suspected during pregnancy, the diagnostic criteria are not different from those for nonpregnant women. Renal disorders appeared to be more common at the onset of SLE in pregnant patients than in nonpregnant patients. Meanwhile, HT is associated with higher rates of infertility and early miscarriages, due to the associated hormonal changes and instability. However, the association of APS and HT is not well recognized in pregnant women [4-7].

Conclusion
We present here a challenging case of new-onset triple autoimmune disorders trigged by pregnancy. Our case confirms a close association between autoimmune thyroiditis, SLE and APS during pregnancy. Clinicians should be aware of this association and initiate early autoimmune work up for SLE and APS in patients with new onset of HT during pregnancy.

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References


