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Secondary antiphospholipid syndrome on oral anticoagulant presented with subdural hematoma: A case report

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CASE STUDY

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ABSTRACT

A 50-year-old female known case of systemic lupus erythematosus with positive antiphospholipid antibody warfarin, presented to hospital complaining of severe headache and vomiting for four days. CT brain showed bilateral chronic subdural haemorrhage more on the left side. Her laboratory investigation showed normal Platelet and INR of 3.10. Warfarin reversed and the patient treated conservatively. Later on, admission, she became drowsy again CT brain repeated and showed extensions of hematoma and increased effacement of brain parenchyma. She underwent left sided burr hole to evacuate hematoma. More studies are needed to guide the management of APLS with bleeding.

Key Words
Antiphospholipid syndrome, systemic lupus erythematosus, subdural bleeding, thrombosis, anticoagulation

Implications for Practice:

1. What is known about this subject?
The antiphospholipid syndrome is an autoimmune disease present with multiple scenarios, including venous and/or arterial thrombosis, pregnancy loss and thrombocytopenia.

2. What new information is offered in this case study?
The balance between benefits of anti coagulation and risk of bleeding is a challenge to avoid a life-threatening event, either thrombosis or bleeding.

3. What are the implications for research, policy, or practice?
All patients with antiphospholipid syndrome should be screened for connective tissue diseases such as SLE, besides closed monitoring of INR.

Background

The antiphospholipid syndrome (APS) is an autoimmune disease associated with variety of clinical disorders, including venous and/or arterial thrombosis, pregnancy loss and thrombocytopenia. The autoantibodies are generated against different epitopes of the participant of the coagulation system (mainly against phospholipid-binding proteins) that result in thrombosis. These antiphospholipid antibodies are a heterogeneous group of autoantibodies. Lupus anticoagulant (LA), anti-cardiolipin antibodies (ACA) and anti-β2 glycoprotein 1 (aβ2GPI) are the most important among them It may occur in context of autoimmune disease, mainly systemic lupus erythematosus named as secondary antiphospholipid syndrome or alone without existing autoimmune disease owning the name primary antiphospholipid syndrome.

Diagnosis of APS is based on the Sapporo criteria (proposed in 1999 and updated in 2006 after a conference in Sydney, Australia). Since thrombosis is the main complication of APLS, preventing this complication by using of prophylactic anticoagulation is warranted.

Till nowadays, no sufficient data regarding duration or intensity of anticoagulation. Treatment with warfarin producing INR more than or equals 3 was significantly effective in preventing recurrence of thrombosis.[1]

Bleeding is unusual first presentation of APLS but could be in sever thrombocytopenia, acquired thrombocytopathy, or an acquired factor VIII inhibitor.[2] Intracranial haemorrhage account as main complication of oral anticoagulation usage.[3] Intracranial haemorrhage could be life-threatening

and difficult to manage. Here we report a case of SLE associated antiphospholipid syndrome presented with acute on top of chronic subdural hematoma.

Case details

A 50-year-old female known case of systemic lupus erythematosus with positive antiphospholipid antibody (lupus anticoagulant and anticardiolipin antibodies), mixed anxiety and depressive disorder and history of deep venous thrombosis, pulmonary embolism and one miscarriage on warfarin. She was receiving moderate dose of steroid, hydroxychloroquine and Azathioprine presented to hospital complaining of severe headache and vomiting for four days. CT brain showed bilateral chronic subdural haemorrhage more on the left side. Creatinine level was 71micmol/L (reference range 53–115), haemoglobin was 11g/dl, Platelet (270 10^9/L) and INR 3.10. Warfarin reversed and the patient treated conservatively. On the second day CT brain repeated showed new fresh blood on the left side acquiring diagnosis of acute on top of chronic subdural haemorrhage. Prophylactic apixaban started. On day 13, she became drowsy again CT brain repeated and showed extensions of hematoma and increased effacement of brain parenchyma (Figure 1). Lab wise platelet level dropped to (47 10^9/L). For treatment of thrombocytopenia, six units platelets were transfused, pulse methylprednisolone 500mg IV once daily for three days, then shifted to oral dose 60mg PO OD. When platelet count increased more than (100 10^9/L), she underwent left sided burr hole to evacuate hematoma. Postoperative CT scan showed resolution of bleeding (Figure 2). Two week later, she became stable post operative and switched to therapeutic dose of apixaban. During six months of follow up the patient did not develop thrombotic nor bleeding events.

Discussion

The antiphospholipid syndrome is an autoimmune disease present with multiple scenarios, including venous and/or arterial thrombosis, pregnancy loss and thrombocytopenia. It may occur as primary antiphospholipid without underlying connective tissue disease or secondary antiphospholipid syndrome if associated with connective tissue diseases such as SLE. Our patient known SLE associated with secondary antiphospholipid syndrome.

The main aim of treatment in APLS is to prevent recurrent thrombosis by anticoagulants. Our patient has history deep venous thrombosis, pulmonary embolism and one miscarriage in correlation with her underlying SLE and positive antiphospholipid antibodies, lifelong anticoagulation was indicated.

In a retrospective study, they found that the risk of recurrent thrombosis in patients with the antiphospholipid-antibody syndrome is high and they recommend Long-term anticoagulation therapy with international normalized ratio is maintained at or above three and they found that it was preventing thrombotic events in 90% of patient over five years period.[1]

In our patient she presented with SDH while she was receiving warfarin with INR in the therapeutic level 3.10.

The balance between benefits of anti coagulation and risk of bleeding is a challenge to avoid a life-threatening event, either thrombosis or bleeding. Uncontrolled blood pressure, concurrent of aspirin and azathioprine were present at the time of bleed in large number of patients.[5,6]

Holding anticoagulation is first step in managing anticoagulated patient presenting with bleeding. Intracranial haemorrhage in anticoagulated patient carries a greater risk of hematoma expansion, neurological deterioration and death necessitating an anticoagulation reversal.[6]

In a case report, Kaaroud et al., found that initiation of steroid, immunosuppressive agents and/or plasma exchange was beneficial for a patient presented with bleeding and can help in resolution of the bleeding.[7] Reinstitution of anticoagulation after resolution of bleeding is essential. Omitting warfarin was associated higher recurrence rate of thrombosis within six months than untreated patient.[1]

The presentence of thrombocytopenia in APLS is common but usually mild and asymptomatic. Thrombocytopenia could be the only manifestation of APLS and found to be associated with thrombotic events more than hemorrhagic due to high platelet function.[8]

Conclusion

At the end, this patient with such presentation represents a dilemma for physician to weight between risk and benefit of anticoagulation and we suggest more studies to focus on management of patient antiphospholipid syndrome presenting with bleeding.

References

1. Khamashta MA, Cuadrado MJ, Mujic F, et al. The management of thrombosis in the antiphospholipid antibody syndrome. N Engl J Med. 1995;332(15):993–7.
2. Forastiero R. Bleeding in the antiphospholipid syndrome. Hematology. 2012;17 Suppl 1:S153–S155. doi:10.1179/102453312X13336169156654
3. Landefeld CS, Goldman L. Major bleeding in outpatients treated with warfarin: incidence and prediction by factors known at the start of outpatient therapy. Am J Med. 1989;87:144–152.

4. Hart RG, Boop BS, Anderson DC. Oral anticoagulants and intracranial hemorrhage. Facts and hypotheses. Stroke. 1995;26(8):1471–1477. doi:10.1161/01.str.26.8.1471
5. Castellino G, Cuadrado MJ, Godfrey T, et al. Characteristics of patients with antiphospholipid syndrome with major bleeding after oral anticoagulant treatment. Ann Rheum Dis. 2001;60(5):527–530. doi:10.1136/ard.60.5.527
6. Flaherty ML. Anticoagulant-associated intracerebral hemorrhage. Semin Neurol. 2010;30(5):565–572. doi:10.1055/s-0030-1268866
7. Kaaroud H, Beji S, Guermazi S, et al. Bleeding and thrombosis in a patient with secondary antiphospholipid syndrome. Saudi J Kidney Dis Transpl. 2008;19(2):227–231.
8. Gadó K, Domján G. Antifoszfolipid-szindróma és a terhesség [Antiphospholipid syndrome and pregnancy]. Orv Hetil. 2012;153(31):1207–1218. doi:10.1556/OH.2012.29425

PEER REVIEW

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CONFLICTS OF INTEREST

The authors declare that they have no competing interests.

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PATIENT CONSENT

The authors, *Alanazi T and Garib A* declare that:

1. They have obtained written, informed consent for the publication of the details relating to the patient(s) in this report.
2. All possible steps have been taken to safeguard the identity of the patient(s).
3. This submission is compliant with the requirements of local research ethics committees.

Figure 1: Preoperative CT scan

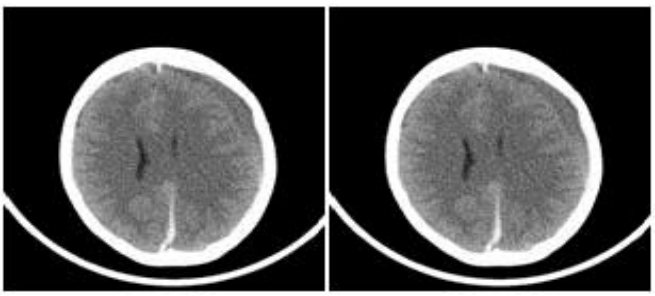

Figure 2: Postoperative CT scan