Case Study

Antiphospholipid antibody syndrome - A case report

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Abstract

Anti-phospholipid antibody (APLA) syndrome is defined by the presence of thrombo-embolic complications and pregnancy morbidity in the presence of persistently increased titers of APLA syndrome. Its clinical presentation can be diverse and any organ can be involved with a current impact in the most surgical and medical specialties. Here, the case of a 34-year-old young lady with APLA syndrome presented with the cerebral venous thrombosis and subsequently deep vein thrombosis of the left leg veins. Three classes of APLAs (IgG, IgM and activated protein C) were elevated. There were no clinical or laboratory evidence for other autoimmune or systemic illnesses. The patient is under treatment of *Ruksha Tikshna Virechana* (purgation) with *Haritaki* (*Terminalia chebula* Retz.) and *Goarka* (extract of cow's urine) with the concept of *Kaphaja Shotha* (nonpitting edema) and got significant result in both subjective and objective parameters.

Keywords: *Goarka*, *Haritaki*, *Ruksha Virechana*, thromboembolism

Introduction

Anti-phospholipid antibody (APLA) syndrome is complex diseases rarely seen in day-to-day practice. APLA is very diverse and can occur in a variety of clinical presentation. The authors therefore present the case of a 34-year-old female with multiple thrombotic events and the presence of APLAs, in the light of *Ruksha Tikshna Virechana* (purgation) with *Haritaki* (*Terminalia chebula* Retz.) and *Goarka* (extract of cow's urine), is one of the remedies to treat *Kaphaja Shotha*.

Case Report

A 34-year-old female presented with pain in all major joints of both upper and lower limb of a 4-year history even after treating with non-steroidal anti-inflammatory drugs (NSAIDs) by a local doctor. Detailed history revealed that on July 15, 2012, she was admitted to the nephrology department for the chief complaints of bilateral pedal edema, peri-orbital edema (since 3 months), mild breathlessness, and sore throat (since 3 days) presented with 3.5 mg/dl serum creatinine, provisionally diagnosed as a case of acute renal failure (ARF) secondary to NSAID and treated symptomatically. Laboratory reports also showed hypoalbuminemia with proteinuria which leads to suspect lupus nephropathy and was treated with ciprofloxacin and salt and fluid restriction. During treatment, she developed severe headache and magnetic resonance (MR) imaging-brain revealed right frontal hemorrhagic infarction suggestive of venous infarction.

The patient had repeated epileptic attacks for three times, and the laboratory reports showed leukocytosis at 14,000 predominantly with eosinophilia, i.e., 22.9%. Laboratory test done on August 28, 2012, showed anticardiolipin (aCL) antibody (antiphospholipid)-IgG positive and aCL antibody (antiphospholipid)-IgM negative, but the same test was repeated on September 24, 2012, as the test should be repeated to confirm diagnosis as aCL antibodies: aCL IgG or IgM antibodies present at moderate or high levels in the blood on two or more occasions at least 6 weeks apart[1] [Table 1]. At the same time, other tests such as factor V Leiden mutation analysis – not detected, anti-thrombin III activity in plasma was 118.00 (80%–120%); near to upper limit. Activated protein C (APC) resistance test was positive; lupus anticoagulant was absent. An MR venogram of the head and neck demonstrated filling defect in the internal jugular vein, sigmoid sinus, and transverse sinus on the left side suggestive of thrombosis.

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Above-explained laboratory parameters confirmed the diagnosis as APLA syndrome. She was advised to continue tablet warfarin and tablet eptoin in the prescribed dose.

With this history, on December 25, 2012, she consulted Kayachikitsa outpatient department, with a chief complaint of the left leg severe pain with edema, unable to sit either on a chair or on ground and pain in joints of both extremities, she was advised for admission. On January 1, 2013, she was admitted to the hospital under Kayachikitsa Department. A color Doppler of the left leg showed the presence of deep vein thrombosis.

***Vyadhi Vinischaya* (diagnosis):** *Kaphaja Shotha*[2] (nonpitting edema).

***Chikitsa Upakaramas*:** In *Kaphaja Shotha*, *Kshara-Katu-Ushna Yukth Dravya* such as *Gomutra, Takra*, *Asava*[3] and *Haritaki* uses with *Gomutra*[4] are indicated.

Treatment given

Ruksha Tikshna Virechana with *Haritaki Choorna* 20 g + *Goarka* 30 ml with *Ushnajala* as *Anupana* [Table 2] and *Sthanika Chikitsa* with *Valuka Sweda* and *Erandmoola Kashaya Pareesheka* to painful joints were given during the course of admission. During menstruation, the treatment was discontinued.

Results

Data based on clinical presentation were collected before, during and after treatment and are presented in the tabular form.

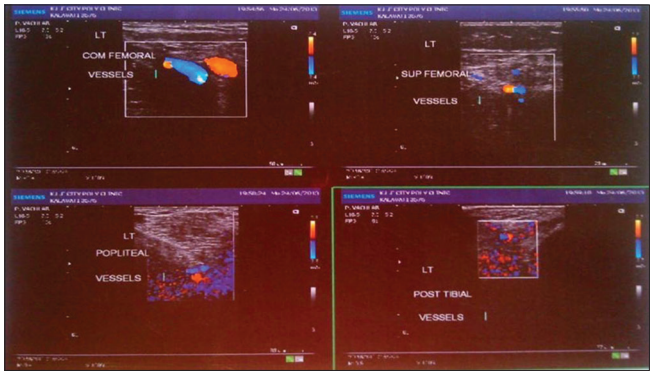

Figure 1: Color Doppler of left lower limb on June 24, 2013 (during treatment)

Table 1: Hematological findings before, during, and after the treatment

Investigations	August 28, 2012	September 24, 2012	June 24, 2013	July 10, 2014
Cardiolipin antibody-IgG	Positive	Negative	Negative	-
Cardiolipin antibody-IgM	Negative	Positive	Negative	-
APC	Positive	-	Negative	-
Homocysteine	Negative	-	-	-
ANA	Negative	-	-	Negative
ESR	-	9 mm/h	9 mm/h	42 mm/h

APC: Activated protein C, ESR: Erythrocyte sedimentation rate, ANA: Antinuclear antibody

Table 2: Course and duration of *Nitya Tikshna Virechana* during admission up to the reduction of *Shotha* (swelling)

IPD number	Date of admission	Date of discharge	Duration of treatment (days)	Weight (kg)
16	January 1, 2013	January 29, 2013	27	59.84
965	February 21, 2013	April 1, 2013	34	53.3
2189	April 27, 2013	May 9, 2013	10	50.2
3079	June 13, 2013	July 6, 2013	22	50.3
4251	August 13, 2013	September 7, 2013	24	49.4
5398	October 24, 2013	October 31, 2013	6	50.4
193	January 9, 2014	January 23, 2014	12	49.8
1041	February 20, 2014	March 4, 2014	10	50.1
2345	May 6, 2014	May 13, 2014	6	49.9
3895	July 10, 2014	July 24, 2014	10	49.7

Table 3: Subjective parameters before, during, and after the treatment

Subjective parameters	January 2, 2013	June 24, 2013	June 22, 2014
Pain in multiple joint	Grade 3	Grade 2	Grade 1
Stiffness in multiple joints	Grade 3	Grade 1	Grade 1
Tenderness in multiple joints	Grade 4	Grade 2	Grade 1
Swelling [Table 3]	All over body	In left lower limb	Absent
Deformity in multiple joints	Absent	Absent	Absent
Seizures episodes	2 episodes before admission	Absent	Absent
Homan's sign	Positive	Positive	Negative
Moses sign	Positive	Negative	Negative
Peripheral pulses	Intact	Intact	Intact
Buerger's test	Negative	Negative	Negative

Table 4: Measurement of both upper and lower limbs in centimeters

Body parts	Date					
	January 2, 2013		June 24, 2013		July 22, 2014	
	Right	Left	Right	Left	Right	Left
Mid-foot	23	26	23	24.5	23	23
At ankle joint	26	27	23.5	25	23	24
Mid-knee joint	34	38	33.5	37	33	33.5
At mid-leg	26	35	27.5	35	27	28
At mid-thigh	45.5	50.5	44.5	49.5	42	43
At wrist	16	16	16	16	15.5	16
At mid-hand	20	22	19	19	18	18.5

Subjective parameters

Buerger's test are the subjective parameters [Table 3] and pain in multiple joint, stiffness in multiple joints, tenderness in multiple joints, swelling [Table 4 and Figures 1, 2], deformity in multiple joints, seizures episodes, Homan's sign, Moses sign and peripheral pulses.

Objective parameters

(i) Hematological investigations [Table 1] such as cardiolipin antibody-IgG, cardiolipin antibody-IgM, APC, homocysteine, antinuclear antibody, erythrocyte sedimentation rate; (ii) prothrombin time international normalized ratio (PT-INR) periodical monitoring [Table 5]; and (iii) radiological findings that are color doppler evaluation of the left lower limb vessels [Table 6 and Figures 3-5] are the objective parameters.

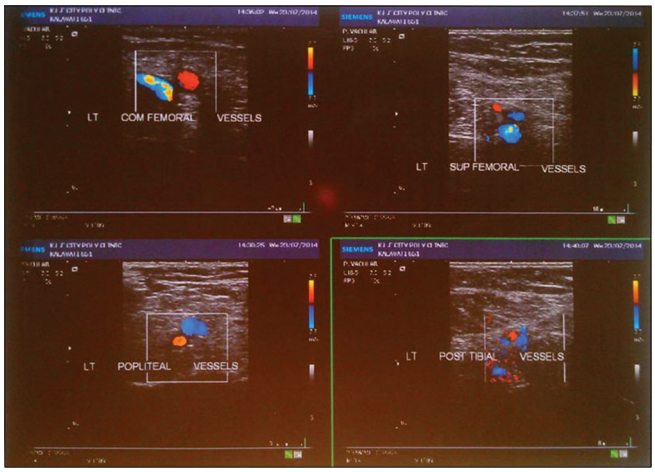

Figure 2: Color Doppler of left lower limb on July 23, 2014 (after treatment)

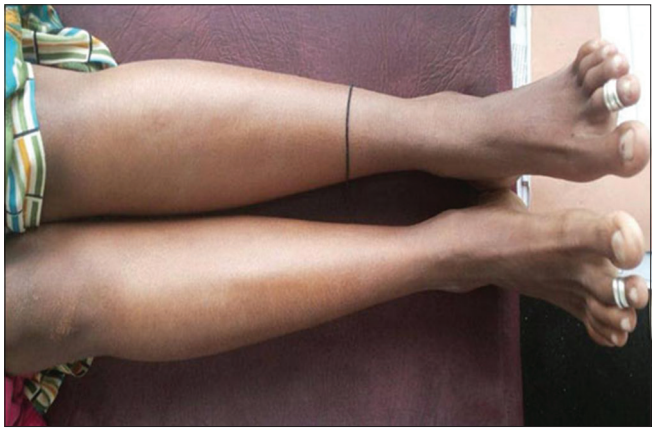

Figure 4: Edema of lower limb after the treatment

Discussion

Antiphospholipid antibody syndrome

The term APLA syndrome denotes the clinical association between APLAs and a syndrome of hypercoagulability.[5,6] It is an autoimmune disease characterized by the presence of thromboembolic complications and pregnancy morbidity in the presence of persistently increased titers of APLAs.[7] The most commonly detected subgroups of APLAs are lupus anticoagulant, aCL, and anti-beta-2-glycoprotein 1 antibodies. Lupus anticoagulant antibodies are associated with

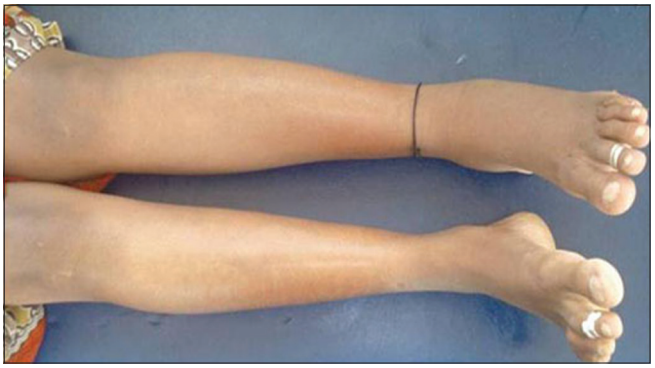

Figure 3: Edema of lower limb before the treatment

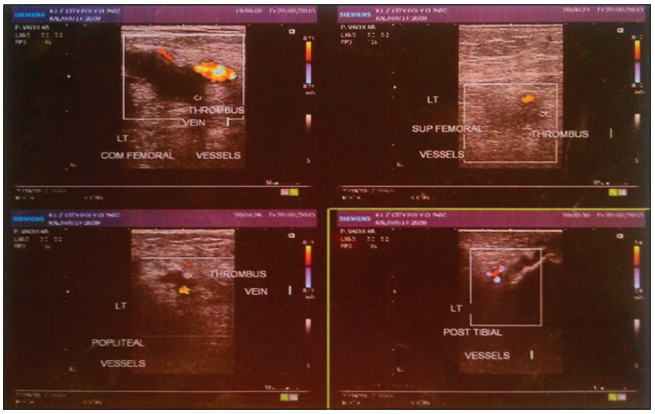

Figure 5: Color Doppler of left lower limb on February 26, 2013 (before treatment)

Table 5: Monthly monitoring of prothrombin time-international normalized ratio (confirmed by coagulometer)

PT	Date					
	August 28, 2013	September 24, 2013	December 11, 2013	February 21, 2014	May 7, 2014	July 10, 2014
Control	15.8	14.4	11.4	12	14	14
Test	16	12.2	16.1	12	12	12
Ratio	1.01	1.19	1.41	1.0	1.1	1.2
INR	1.01	1.19	1.41	1.0	1.2	1.3

INR: International normalized ratio, PT: Prothrombin time

Table 6: Radiological findings: Color Doppler evaluation of left lower limb vessels

Date	Impression
February 22, 2013	Figure 3 shows an organized hypoechoic thrombus seen in the external iliac, common femoral, superficial femoral, popliteal and proximal posterior tibial veins of left lower limb. Thrombus is also extended into the greater saphenous vein with mild dilatation of the lower portion Deep vein thrombosis involving the above-said veins of the left lower limb
June 24, 2013	Figure 4 shows partial recanalization of the old thrombus in external iliac, common femoral, superficial femoral, popliteal and proximal posterior tibial veins of the left lower limb
July 23, 2014	Figure 5 As compared to the previous color Doppler studies done on February 26, 2013 and June 24, 2013, there is significant near-complete recanalization seen in all the veins of the left lower limb On color Doppler examination, there are good color signals visualized with normal respiratory variation. Superficial veins show normal appearance. No evidence of thrombosis in the superficial veins

thromboembolic events rather than clinical bleeding. APLAs can interfere with both pro- and anti-coagulant pathways

Probable mode of action

Gomutra Haritaki Nitya Tikshna Virechana might have acted to prevent further thrombosis along with recanalization of thrombosed veins without any thromboembolism [Figure 3]. Studies have showed that *Gomutra*[8] and *Haritaki*[9] as immune modulatory in action by which we can assume that the other associated symptoms might have reduced.

Anticipated action

In this patient based on subjective and objective findings, it was observed that, near complete recanalization of completely thrombosed veins without the history of thromboembolism. Antithrombotic activities of *Haritaki* and *Goarka* are not yet proven; further studies are needed to find the efficacy of *Goarka-Haritaki* in resolving, preventing further thrombus formation and recanalization of thrombosed vein without a history of any embolism.

Future treatment planning

Treatment will be continued by monitoring PT-INR every month and are planning to conduct MR venogram to rule out the presence of thrombus.

Conclusion

Though APS is an autoimmune condition with limited treatment options, if properly treated as per the basic principles of Ayurveda under the light of *Shotha Chikitsa* promising results can be obtained which gives a hope for its further approach without any adverse effects.

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Conflicts of interest

There are no conflicts of interest.

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