Table 2. Urine Test results.

Test	Result	Test	Result
Pus cells	10_12/HPF (High Power Field)	Red blood cells (RBCs)	2_26/HPF
Protein	+++	Cast	5RBCs

Table 3. Laboratory data on day three.

Tests	Results
WBC	$10.3x10^3$
Hemoglobin	7.1 g/dl
Platelet	$241x10^3$
Urea	64mg/dl
Creatinine	2.6mg/dl
Na	132mmol/l
K	2.2mmol/l
RBG	89mg/dl(80_120mg/dl)
CXR	normal.
ECG	sinus tachycardia.
Anticardiolipin IgG	0.9U/L (Up to 10U/L)
Anticardiolipin IgM	1.2U/L (Up to 10U/L)
Lupus anticoagulant	32(18_45 sec)
AntiB2glycoprotein 1 IgM	10.3U/L (+ve >8U/L)
AntiB2glycoprotein 1 IgG	13.4U/L (+ve>8U/L)
Rheumatoid factor	<8(_ve)
AntiCCP	8.6Iu/l (<20Iu/l _ve)
ANA profile	+ve for PCNA and Anti ribosomal P antibodies, -ve for all antibodies including anti dsDNA.
Abdominal ultrasound	Normal size kidneys
Histological finding/ Skin biopsy	Taken from fingertips and showed lecuocytoclastic vasculitis extensive small vessel thrombosis.

Table 4. Laboratory data on discharge and post discharge.

Test	On discharge	Post discharge follow up
WBC	4.8	$5.7x10^9$
Hemoglobin	10.2	9.2
Platelet	249	451
Urea	34	24
Creatinine	0.8	1.1
Na	134	143
K	3.2	3.6
Protein	6.5	7.4
Albumin	2.9	3.9
AST	34	14
ALT	27	12
INR	1.15	2.2
PT	13.2	_
APTT	26.2	_
ESR	20	15
CRP	19	14
Urine analysis	0_1. RBCs: 3_4. No more protein or casts.	No sediment
C3	71.4mg/dl	(90_180mg/dl)
C4	C4:27.8mg/dl	(9_36mg/dl)
Echocardiography	Echocardiography: EF: 62%, normal study. EF:62% and normal parameters.	

DISCUSSION

Antiphospholipid syndrome (APL) is an acquired autoimmune disease characterized by the production of autoantibodies (anticardiolipin, lupus anticoagulant and B2 glycoprotein 1) that are primarily responsible for widespread thrombosis including arterial, venous and small vessels

alongside pregnancy morbidity [1,3]. CAPS is a frightening, extremely rare type of APL syndrome that occurs rapidly and was first recognized in 1992 by Ronald Asherson [2,5,6]. Although rare, it affects multiple organs [2,5,7] and has high mortality which necessitates the early recognition and prompt accurate intervention.

CAPS is classified into definite and probable according to certain preliminary criteria [4,8] which are:

1. Involvement of at least 3 organs or systems
2. Rapid onset of clinical features in less than one week
3. Histopathological confirmation of small vessels occlusion in at least one organ or tissue
4. Presence of antiphospholipid antibodies

Definite CAPS requires the presence of all 4 criteria while probable CAPS requires 3 out of 4 criteria to be fulfilled. The exact cause of CAPS is still elusive but different mechanisms may play a fundamental role in the pathogenesis such as infections, endothelial damage with complement activation, upregulation of adhesion molecules and reduction of fibrinolytic factors which collectively lead to microthrombi formation and occlusion of small vessels resulting in tissue ischemia and release of many cytokines leading to a systemic inflammatory response which is the major culprit of inflammatory and clinical features [4,9,10]. CAPS is a fatal condition, early recognition and accelerated management is the cornerstone to safe life [11].

Infections such as malaria, which is caused by a parasitic infection and classified as an endemic disease in Sudan, was conceived to be the main causative agent in this case as it is considered to be a major precipitating reason for CAPS through immune system activation [6,12,13].

CAPS is treated with glucocorticoids, anticoagulants and antiplatelets while in severe cases, plasma exchange and intravenous immunoglobulin are best recommended alongside treating any infections or triggering factors. If there is no response, cyclophosphamide for SLE flare or Rituximab can be considered [2,5,14].

SLE is an autoimmune disorder that is associated with immune complexes deposition that can affect the kidneys badly and leads to lupus nephritis which is diagnosed by the presence of proteinuria >0.5gm/24h (urinary protein+++) or urine albumin/creatinine ratio >0.5 and active urinary sediment (RBCs cast >5, WBCs >5) after the exclusion of any underlying causes. A renal biopsy is considered an added criterion [14]. In this case, the patient fulfilled the criteria having >5 RBCs cast and urine albumin /creatinine ratio 1.2. A renal biopsy was requested but the patient refused to have it done.

PCNA is a protein that is essential for DNA repair and replication through its role in enhancing the action of DNA polymerase. It correlates with disease activity and associates with renal involvement [15,16] as presented in our patient's case who was positive for PCNA.

SLE is a multisystem connective disease, associated with a myriad of clinical manifestations and associations, APL being one of the serious ones. CAPS is a lethal variant of APL that can be followed with sepsis as suspected in our patient [17].

Although the patient exhibited a fulminant clinical presentation, the patient's clinical outcome is being satisfactory and acceptable given the resource and financial constraints that affected the patient's care.

DECLARATIONS

Ethics approval and consent to participate

The patient in our case report has signed a written consent form and she has no concerns. A copy of the consent form is available upon request.

CONSENT TO PUBLISH

Written consent was obtained for publication and she reviewed all data included in the manuscript before submission.

AVAILABILITY OF DATA AND MATERIALS

Not applicable.

COMPETING INTERESTS

The authors declare that they have no competing interests.

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AUTHORS' CONTRIBUTIONS

HA was involved in patient care and case selection. AR was involved in patient care, writing and reviewing the abstract, case description, discussion, and the conclusion of the manuscript. Both authors contributed to the planning of the case report, manuscript revision and final approval of the manuscript.

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