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ORIGINAL RESEARCH

# Chronic abdominal aortic occlusive disease related to antiphospholipid syndrome: a rare presentation

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## ABSTRACT

**Objective** Chronic abdominal aortic occlusive disease (CAAOD) is an uncommon manifestation of antiphospholipid syndrome (APS), impacting cardiovascular health and peripheral arterial circulation. We investigated CAAOD in antiphospholipid antibodies (aPL)-positive patients, aimed to offer comprehensive clinical and radiological insights.

**Methods** aPL-positive patients with arterial thrombotic events were categorised into CAAOD and non-CAAOD. Extensive data, including clinical features, radiological images and outcomes, were analysed.

**Results** This case-control study involved 114 patients who experienced arterial events from 2013 to 2021, revealing 12 patients with abdominal aortic stenosis or occlusion. The CAAOD group, predominantly young (36.67±11.83) males (75.00%), exhibited significantly higher rates of critical smoking habits (66.67% vs 25.49%, p=*0.006*) and hyperhomocysteinaemia (66.67% vs 31.37%, p=*0.026*). Radiological findings showed long-segment infrarenal aorta stenosis in CAAOD, occasionally involving renal and common iliac arteries. The lesions presented varying degrees of stenosis, including smooth lumen narrow and total vascular occlusion. Treatment modalities typically involved interventions or surgery, complementing anticoagulation therapy.

**Conclusion** The study shed light on the rare occurrence of CAAOD in APS, highlighting the roles of smoking and hyperhomocysteinaemia as notable risk factors. These findings emphasised the significance of early diagnosis and management of CAAOD.

### WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Current evidence has reported large vascular stenosis/occlusion of renal and cerebral arteries in patients with antiphospholipid syndrome (APS). Yet, the lesion rarely occurs in the abdominal aorta.

### WHAT THIS STUDY ADDS

⇒ This study provides a comprehensive examination of the clinical and radiological features of chronic abdominal aortic occlusive disease (CAAOD) in aPL-positive patients, as well as symptoms and potential risk factors that require attention.

### HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE, OR POLICY

⇒ The study advocates for a more proactive strategy in identifying CAAOD, addressing risk factors and initiating timely surgical intervention in APS. These findings may impact healthcare practices and the establishment of standardised treatment protocols, thus prompting further comprehensive investigations into the pathophysiology.

## INTRODUCTION

Antiphospholipid syndrome (APS) is a systemic autoimmune disease characterised by vascular thrombosis and obstetric complications, accompanied by persistent positive antiphospholipid antibodies (aPL).[1] Approximately 40% of patients with aPL experience arterial events, particularly affecting cerebral and cardiovascular vessels.[2] In addition to well-recognised classical thromboembolisms, emerging evidence suggests that APS presents a wide range of vascular complications.[3 4] Several studies have reported arterial stenosis/occlusion and vessel wall proliferation without thrombus, especially in the renal and cerebral arteries.[5 6] Despite this progress, abdominal aortic stenosis continues to be sporadically reported and limited to anecdotal cases.[7]

Chronic abdominal aortic occlusive disease (CAAOD) is a rare form of peripheral arterial disease (PAD) characterised by the narrowing or occlusion of the abdominal aorta and/or its branches, often affecting the infrarenal aorta.[8] PAD primarily affects elderly male patients, leading to various clinical presentations such as progressive claudication, lower limb amputation and pulse abnormalities. Chronic lesions can also manifest as asymptomatic stenosis due to the development of a

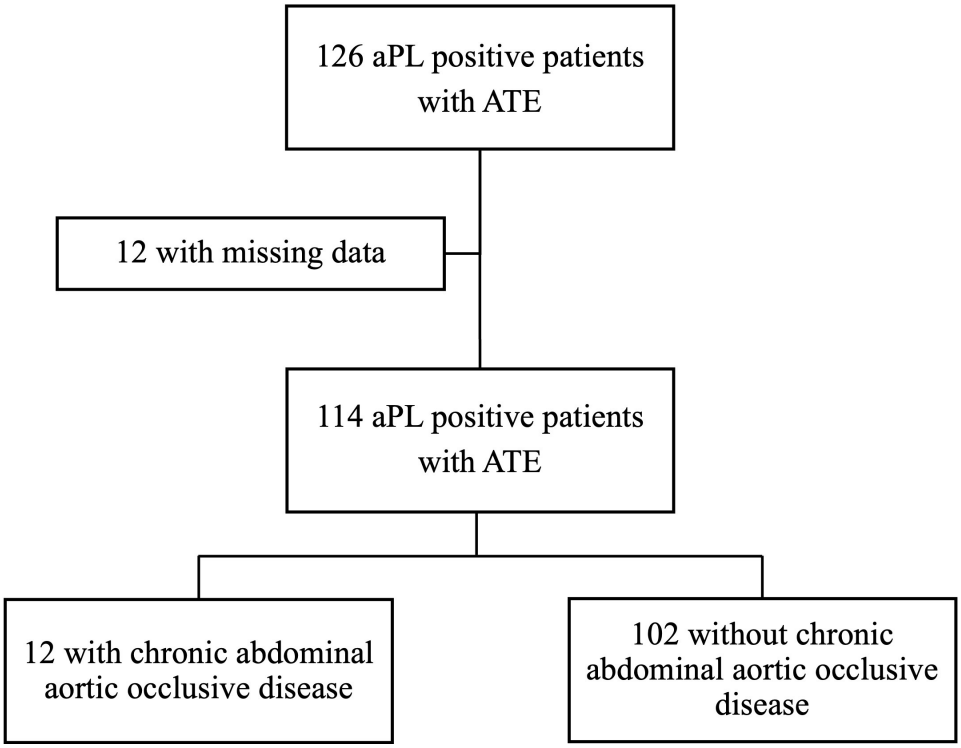


**Figure 1** Flow chart. Totally 126 antiphospholipid antibodies (aPL)-positive patients had arterial lesions including thrombosis or vasculopathy, 12 patients lost follow-up, 12 patients presented chronic abdominal aortic occlusive disease and the left 102 patients did not show abdominal aortic occlusive disease. ATE, arterial thromboembolism.

strong collateral circulation. Widespread atherosclerosis and arterial thrombosis are major contributors to the aetiology.[9] Additionally, potent hypercoagulable states precipitate CAAOD in younger patients. In our cohort, several patients with APS reported complete abdominal aortic occlusion with involvement of the iliac or renal arteries. This intriguing observation provides a glimpse into a potential relationship between APS and CAAOD, which has been rarely documented in previous literature. Our study offers a detailed description of the clinical characteristics and radiological features of CAAOD, to improve the recognition and diagnosis of CAAOD in patients with APS.

## METHODS

### Participates

This case-control study used the database of Peking Union Medical College Hospital from January 2013 to January 2021. The study enrolled patients with positive aPL who had presented with arterial lesions. Patients with chronic stenosis or occlusion in the abdominal aorta were classified as group CAAOD. Others were classified as group non-CAAOD. All participants had written informed consent.

### Clinical data collection

Demographic characteristics, lifestyle behaviours, comorbidities, medical histories and physical examinations were carefully recorded by an experienced rheumatologist at each visit. Information on the treatment process, laboratory indices and outcomes were collected from the Hospital Information System. Serum anti-cardiolipin antibody (aCL) and anti-β2glycoprotein I antibody (aβ2GPI) were detected using ELISA. The cut-offs for each aPL were set according to the manufacturer's recommendations: aCL IgG/IgM levels≥12 GPLU/mL, and aβ2GPI IgG/IgM levels≥20 AU/mL. The positivity of aCL and aβ2GPI was considered as a titre over the cut-off values. Lupus anticoagulant (LA) was detected by dilute Russell viper venom time and activated partial thromboplastin time, following the recommendations of the International Society on Thrombosis and Hemostasis.[10] Patients were considered positive for LA if the ratio was ≥1.2. Patients with any aPL positivity were confirmed with an interval of at least 12 weeks. APS was diagnosed in patients who experienced vascular events or obstetric complications and had persistent medium-to-high titres of aPL, according to the Sapporo criteria.[11]

### Radiology

Based on the rigorous process of clinical evaluation and preliminary screening, computed tomographic angiography (CTA) was performed on individuals who raised suspicions of abdominal aortic disease or unexplained ischaemic conditions in either the visceral or lower limbs. The specific 'string of beads' appearance indicative of fibromuscular dysplasia was excluded. Potential arteritis was meticulously excluded, as defined by systemic inflammation or occlusion of the carotid/cervical arteries. Arrhythmia and valvular diseases were ruled out using

**Table 1** Clinical characteristics of antiphospholipid antibodies (aPL)-positive patients with and without chronic abdominal aortic occlusive disease

| | Arterial thrombotic events | | |
|---|---|---|---|
| | CAAOD (n=12) | Non-CAAOD (n=102) | P value |
| Male | 9 (75.00) | 45 (44.12) | 0.065 |
| Age onset (y) | 36.67±11.83 | 33.33±13.91 | 0.421 |
| Secondary APS (n/%) | 1 (8.33) | 24 (23.53) | 0.459 |
| Cardiovascular risk factors (n/%) | | | |
| Smoking | 8 (66.67) | 26 (25.49) | 0.006* |
| Hypertension | 4 (33.33) | 34 (33.33) | 1.000 |
| Diabetes | 1 (8.33) | 7 (6.86) | 1.000 |
| Obesity (BMI≥30 kg/m$^2$) | 0 | 8 (7.84) | 1.000 |
| Dyslipidaemia | 9 (75.00) | 61 (59.80) | 0.365 |
| Hyperhomocysteinaemia | 8 (66.67) | 32 (31.37) | 0.026† |
| APS manifestations except ATE (n/%) | | | |
| First site of ATE | | | |
| Stroke/TIA | 1 (8.33) | 64 (62.75) | <0.001* |
| MI/angina | 1 (8.33) | 8 (7.84) | 1.000 |
| lower limb artery | 7 (58.33) | 12 (11.76) | <0.001* |
| Visceral ATE/organ infarction | 9 (75.00) | 13 (12.75) | <0.001* |
| Retinal arterial occlusion | 0 | 5 (4.90) | 1.000 |
| VTE | 4 (33.33) | 42 (41.18) | 0.759 |
| Deep venous thrombosis | 4 (33.33) | 27 (26.47) | 0.732 |
| Pulmonary embolism | 0 | 16 (15.69) | 0.212 |
| Visceral venous thrombosis | 1 (8.33) | 4 (3.92) | 0.433 |
| Cranial venous sinus thrombosis | 1 (8.33) | 4 (3.92) | 0.433 |
| Retinal venous thrombosis | 0 | 3 (2.94) | 1.000 |
| Non-criteria manifestations | 3 (25.00) | 56 (54.90) | 0.068 |
| Thrombocytopenia | 1 (8.33) | 40 (39.22) | 0.053 |
| Hemolytic anaemia | 0 | 14 (13.73) | 0.356 |
| Valve vegetation | 0 | 10 (9.80) | 0.596 |
| APS nephropathy | 0 | 11 (10.78) | 0.603 |
| Non-stroke CNS manifestation | 0 | 14 (13.73) | 0.356 |
| Cutaneous ulcer | 1 (8.33) | 1 (0.98) | 0.200 |
| Livedo reticularis | 0 | 5 (4.90) | 1.000 |
| Superficial vein thrombosis | 1 (8.33) | 1 (0.98) | 0.200 |
| aPL phenotypes (n/%) | | | |
| aCL IgG/IgM positivity | 9 (75.00) | 74 (72.55) | 1.000 |
| anti-β2GPI IgG/IgM positivity | 10 (83.33) | 84 (82.35) | 1.000 |
| LA positivity | 12 (100.00) | 89 (87.25) | 0.355 |
| Triple aPL positivity | 8 (66.67) | 61 (59.80) | 0.761 |
| aGAPSS (median±SD) | 13.50 (11.75, 14.00) | 13.00 (9.00, 14.00) | 0.633 |

*p<0.01.
†p<0.05.
‡
aCL, anti-cardiolipin antibody; aGAPSS, adjusted global antiphospholipid syndrome score; aPL, antiphospholipid antibodies; APS, antiphospholipid syndrome; APS nephropathy, acute thrombotic microangiopathy or chronic intrarenal vascular lesions; ATE, arterial thromboembolism; BMI, body mass index; CAAOD, chronic abdominal aortic occlusive disease; CNS, central nervous system; LA, lupus anticoagulant; VTE, venous thromboembolism; β2GPI, Beta-2-Glycoprotein I.

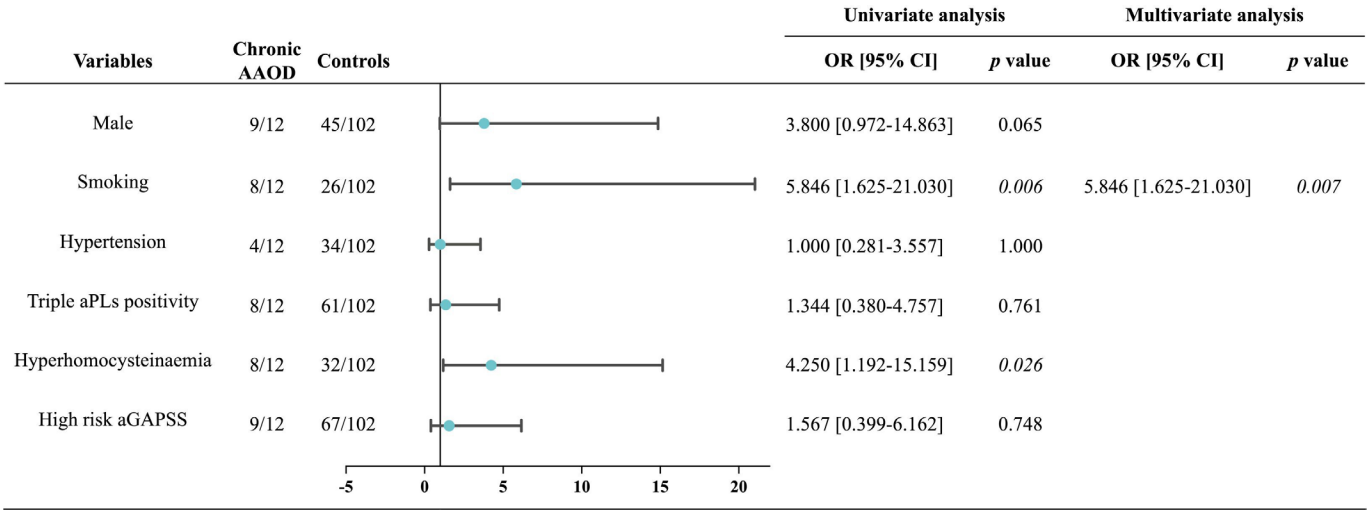


**Figure 2** Forest plot. In 114 antiphospholipid antibodies (aPL)-positive patients with arterial thrombotic events, risk factors for chronic abdominal aortic occlusive disease (CAAOD) were analysed using $\chi^2$ test, OR values and 95% CI. In univariate regression analysis, smoking and hyperhomocysteinaemia presented specifically increased risk. After multivariate analysis, smoking reserved strong association with CAAOD. aGAPSS, adjusted global antiphospholipid syndrome score.

an ECG and echocardiogram. Other secondary conditions, such as tissue compression, congenital thrombophilia, Marfan syndrome and small-vessel vasculitis, were excluded as much as possible.

A radiology professor supervised all radiological reports. The assessment involved a comprehensive quantification of stenotic indices, including the percentage of stenosis degree, calculation of the average wall thickness across four distinct quarters at the narrowest point, and the measurement of the stenotic length. Renal artery ultrasound was used to measure the peak systolic velocity of the main renal artery in patients with CAAOD.

### Statistical methods

Dichotomous factors were expressed as numbers and percentages and analysed using Fisher's exact test (T<5) or the $\chi^2$ test as appropriate. Continuous variables were presented as mean and SD or median and IQR and analysed using Student's t-test or the Wilcoxon-Mann-Whitney test as appropriate. Regression and forest plots were used to analyse the risks associated with abdominal aortic vasculopathy, presented as OR and 95% CIs. Data analysis was conducted using SPSS V.27 (IBM Corp.). A two-sided p value of less than 0.05 was considered statistically significant.

## RESULTS

### Baseline characteristics and risk factors

Among 126 patients who visited our department with arterial lesions and positive aPL, 114 had complete data. Among them, 12 patients were identified with CAAOD and met the APS criteria. The remaining 102 patients presented non-CAAOD, 95 of them were diagnosed with APS and 7 of them only had positive aPL (figure 1). Most of the patients with CAAOD admitted to the hospital complained of progressive claudication, lower extremity rest pain, numbness or abdominal discomfort for at least 3 months (table 2). When comparing groups, it was found that most of the patients with CAAOD were male (75.00%). Although cardiovascular risk factors were commonly detected in both groups, smoking (66.67% vs 25.49%, p=0.006) and hyperhomocysteinaemia (66.67% vs 31.37%, p=*0.026*) were more frequent in group CAAOD. Patients with CAAOD had a lower incidence of venous thrombosis (33.33% vs 41.18%, p=0.759) and non-criteria manifestations (25.00% vs 54.90%, p=0.068), particularly thrombocytopenia (8.33% vs 39.22%, p=0.053). But statistical difference was not found between the groups (table 1). During the regression analysis, smoking (OR=5.846, 95% CI 1.625 to 21.030) and hyperhomocysteinaemia (OR=4.250, 95% CI 1.192 to 15.159) showed an association with CAAOD. After conducting multivariate regression, smoking showed the strongest association (figure 2).

### Radiological findings

The CTA scans revealed a range of stenotic conditions within the abdominal aorta, ranging from mild stenosis to complete occlusion. The lesions were mainly located within the region from the infrarenal aorta to the iliac artery. Three patients showed involvement of the renal artery, while only one patient had chronic renal failure. The average stenotic length was 77.46±30.82 mm, and the narrowest luminal diameter averaged 9.43±5.14 mm. The mural thickness did not increase significantly in CAAOD, remaining mostly symmetrical in all four quarters of the artery vessel. Patient 12 presented with secondary thromboembolism in the lumen (table 2). The burden of calcified plaque was generally moderate, and positron emission tomography-CT of patient 7 showed no increased uptake in the vessel walls of the aorta. Four out of seven patients with complete renal artery ultrasound

**Table 2** Baseline demographic and radiology of antiphospholipid antibodies-positive patients with chronic abdominal aortic occlusive disease

| ID | Age onset | Symptoms and duration before diagnosis | Arteries presentation | Stenotic aortic length (mm) | Average wall thickness (mm) | Renal artery PSV (cm/s) | Antiplatelet | Anticoagulant | Immunosuppressive therapy | Surgical therapy | Outcome |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Patient in their 40s | Claudication 2 years | Infrarenal aortoiliac occlusion, SMA stenosis | 92.3 | 2.23 | 92/83 | Aspirin | Warfarin | Steroid+CYC/sirolimus | Endarterectomy, aortobifemoral bypass | Artificial vessel thrombus at 1 year with aspirin monotherapy, patent after embolectomy |
| 2 | Patient in their 20s | Nausea, chest tight, lower limb oedema 10 years | Pararenal aortic occlusion, left RAS | 75 | 1.45 | –/– | Aspirin | Warfarin | Sirolimus | None | Stable at 3 years |
| 3 | Patient in their 20s | Claudication 2 years | Infrarenal aortoiliac stenosis (right CIA) 50%–70% | 20 | 1.00 | –/– | Aspirin | Warfarin | None | None | Stable at 7 years |
| 4 | Patient in their 50s | Numbness, claudication 4 years | Infrarenal aortoiliac occlusion, SMA occlusion | – | – | –/– | Aspirin | Warfarin | TAC | Balloon dilation, stenting | Stable at 2 years |
| 5 | Patient in their 20s | Claudication, ulcer/gangrene 5 years | Infrarenal aortoiliac stenosis 50%–70%, bilateral CIA occlusion | 105 | 2.13 | 155/88 | Aspirin | Rivaroxaban | Steroid+CYC | None | Stable at 1 year |
| 6 | Patient in their 40s | Claudication 3 years | Infrarenal aortoiliac occlusion, bilateral RAS, SMA occlusion | 86.6 | 2.30 | –/284 | Aspirin+clopidogrel | None | None | Aortobifemoral bypass | Stable at 1 years |
| 7 | Patient in their 30s | Claudication 4 years | Pararenal aortoiliac occlusion (left CIA), bilateral RAS, SMA occlusion | 106.8 | 1.95 | 114/165 | Aspirin | Warfarin | Steroid | Endarterectomy, aortobifemoral bypass | Stable at 3 years |
| 8 | Patient in their 40s | Toes rest pain 3 years | Abdominal aortic occlusion | – | – | –/– | Aspirin | Warfarin | None | Balloon dilation, stenting | Slight restenosis at 5 months with DAPT, stable after changing into warfarin |

Continued

**Table 2** Continued

| ID | Age onset | Symptoms and duration before diagnosis | Arteries presentation | Stenotic aortic length (mm) | Average wall thickness (mm) | Renal artery PSV (cm/s) | Antiplatelet | Anticoagulant | Immunosuppressive therapy | Surgical therapy | Outcome |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 9 | Patient in their 20s | Toes rest pain 2 years | Abdominal aortic occlusion | – | – | 120/118 | Aspirin | Warfarin | Steroid+CYC | Stenting | Stable at 4 years |
| 10 | Patient in their 50s | Numbness, claudication 3 months | Infrarenal aortoiliac occlusion | 100 | 1.75 | –/– | Aspirin | Warfarin | None | Aortobifemoral bypass | Stable at 10 years |
| 11 | Patient in their 20s | Asymptomatic | Terminal aortoiliac stenosis (right CIA), <50% | 34.4 | 1.80 | 65/67 | Aspirin | Warfarin | None | None | Stable at 6 years |
| 12 | Patient in their 50s | Claudication 6 months | Infrarenal abdominal aortic stenosis, 50%–70% | 77 | 1.28 | 65/95 | Aspirin | Warfarin | Steroid+TAC/sirolimus | None | Stable at 5 years, discontinuation of aspirin since ITP |

CIA, common iliac artery; CYC, cyclophosphamide; DAPT, dual antiplatelet therapy; HCQ, hydroxychloroquine; PSV, peak systolic velocity, normal PSV of renal artery is 50-100 cm/s; RAS, renal artery stenosis; SMA, superior mesenteric artery; TAC, tacrolimus.

data exhibited increased renal artery PSV (>100 cm/s) (figure 3).

## Outcomes

During the observation of patients with CAAOD, most received antiplatelet and anticoagulant therapy, except for one patient who discontinued aspirin due to thrombocytopenia. Several patients were additionally treated with steroids and immunosuppressants due to thrombocytopenia, vascular proliferation or potential immune response in the early stages of treatment. Steroid doses were reduced to a minimum, and cyclophosphamide was discontinued within 6 months for all patients. Patient 1 and patient 8 experienced restenosis before being diagnosed with APS and receiving anticoagulant therapy later. The other patients with CAAOD experienced symptoms of improvement or graft patency during treatment (table 2).

## DISCUSSION

Our study suggests that CAAOD represents a rare and distinct arterial manifestation of APS, which is associated with male sex, smoking habits and hyperhomocysteinaemia. Most patients experienced chronic symptoms that required surgical interventions.

APS presents a wide range of abdominal manifestations, including thrombosis, infarction, organ damage and microthrombi.[12] Recently, a significant narrowing of large blood vessels in the abdomen has been observed.[13] Initial reports suggest that aPL carriers may have arterial stenosis, which is linked to thrombosis and vasculitis.[14] Subsequent cases have documented stenosis of visceral vessels such as the coeliac trunk, mesenteric artery and renal artery.[15–17] Sangle *et al* report renal artery stenosis (RAS) in over 20% of aPL carriers with resistant hypertension, as visualised on magnetic resonance angiography (MRA).[18 19] Notably, abdominal aortic stenosis in APS remains poorly reported.[7 20] Alfayate *et al* report a case of a young woman with APS who developed a total occlusion of the infrarenal aorta and bilateral iliac arteries.[21] Karaolanis *et al* have reviewed all 12 sporadic cases of aortic lesions related to APS. The lesions mainly affect middle-aged patients, and most of them receive surgical or interventional treatment.[22]

Aortic occlusion is a rare occurrence associated with various underlying lesions, distinguished by imaging.[23] Atherosclerosis manifests as eccentric plaques and calcified lesions on CTA, which are more prominent in the distal abdominal aorta and branch Ostia.[24] Takayasu arteritis predominantly affects young women and involves non-specific inflammation.[25 26] The CTA reveals concentric mural thickening with a 'double ring' enhancement pattern.[27] The $^{18}$F-fluorodeoxyglucose positron emission tomography can indicate increased vascular uptake.[28] Acute aortic mural thrombosis typically presents as hypodense filling defects attached to the vessel walls with a floating component.[29] Two types of APS-related

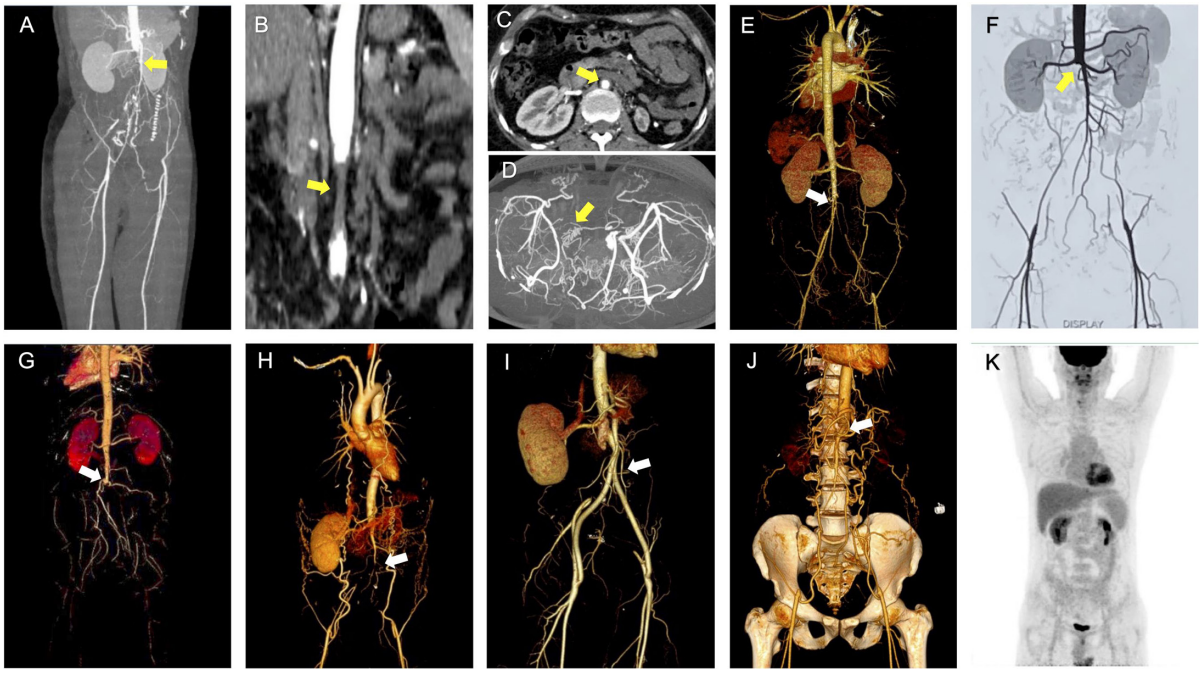


**Figure 3** Radiology imaging of chronic abdominal aortic occlusive disease in antiphospholipid antibodies (aPL)-positive patients. Patient 1 presented complete occlusion from the infrarenal aorta, accompanied by restenosis within the graft vessel (A). Patient 2 presented a collapsed distal segment of the abdominal aorta, (B) characterised by a reduction in lumen diameter (C). Patient 3 presented a well-developed collateral circulation (D) and stenosis of the distal aorta (E). Patient 4 presented occlusion of infrarenal aorta and common iliac arteries. (F) Patient 5 showed shorter occlusion of distal aorta. (G) Patient 6 presented infrarenal aortic occlusion, inclusive of left renal artery involvment (H), and the observation of patent graft vessel (I). Patient 7 presented severe occlusion including pararenal aorta and bilateral renal artery (J), and $^{18}$F-fluorodeoxyglucose positron emission tomography-CT was normal in vessel wall (K).

RAS are reported in radiology: one with smooth non-critical stenosis and another resembling atherosclerosis-like lesions involving the aorta.[30] Our study found both types in the abdominal aorta. As aortic occlusion is often diagnosed at an advanced stage with severe blockage, we hypothesise that these two presentations may represent distinct stages of CAAOD. Further research is needed to investigate the implications of these various presentations and their connection to disease progression.

From a clinical perspective, we have found that smoking is the most related risk factor for CAAOD. Smoking has traditionally played a pivotal role in PAD by promoting vascular damage through the increase of reactive oxygen species and oxidative stress in endothelial cells. Smoking cessation has been shown to improve vasodilation and reduce the risk of limb ischaemia.[31] Our study has revealed a strong correlation between smoking and abdominal aortic lesions. These findings underscore the importance of promoting smoking cessation in APS management.

The mechanisms of APS-related arterial lesions are multiple, and the pathology of aortic occlusive disease remains undetermined. Pathogenic aPL stimulate endothelial and platelet activation, monocyte tissue factor production and the release of neutrophil extracellular traps, ultimately leading to thrombus formation.[32 33] APL also binds to circulating oxidised low-density lipoprotein, enhancing its uptake by macrophages, promoting macrophages' differentiation into foam cells, and accelerating the development of atherosclerotic plaque.[34 35] Furthermore, aPL is associated with chronic vasculopathy, characterised by intimal thickening and hyperplasia, similar to arteriosclerotic hypertension.[36] This condition is characterised by the proliferation of smooth muscle cells, increment of extracellular matrix and constriction of the lumen.[37] The phosphoinositide-3 kinase-AKT-mammalian target of rapamycin complex (mTORC) pathway is involved in endothelial proliferation in aPL-related vasculopathy.[38] Treatment with an mTORC inhibitor can alleviate the lesions.[39]

Traditional abdominal aortic occlusion, which is often associated with an increased risk of amputation, cardiovascular diseases and mortality, is managed with antiplatelet agents to improve patency and minimise cardiovascular morbidity.[8 40] Anticoagulant therapy is not routinely recommended due to concerns about the risk of bleeding and limited benefits in preventing cardiovascular diseases.[41] Surgical and stenting interventions are preferred depending on the extent of the lesion. Surgery offers a higher patency rate but also entails a higher mortality rate.[42 43] APS-related CAAOD lacks established treatment recommendations. Sangle *et al* report improvement in resistant hypertension and patency rate in RAS with a target international normalised ratio of 3.0–4.5.[30] Individual cases of abdominal aortic thrombosis have been treated with anticoagulation or surgical

procedures.[44] In our cohort, two patients with antiplatelet treatment after revascularisation surgery experienced restenosis or thrombosis. While others who were treated with anticoagulants or dual antiplatelet therapy showed stable outcomes after revascularisation. Thus, appropriate anticoagulation and individualised treatment are necessary. The early screening for aPL in patients with abdominal aortic occlusion also influences the treatment decisions.

Timely identification of CAAOD holds significance due to its potential adverse health outcomes. Clinicians are advised to focus on young to middle-aged males diagnosed with APS and presenting multiple cardiovascular risk factors, notably highlighting smoking and hyperhomocysteinaemia. This group may have an increased susceptibility to CAAOD. Healthcare practitioners should remain vigilant to the symptoms of potential CAAOD, such as claudication, lower limb rest pain or numbness, open ulcers, distal pulselessness, renal complications, or unexplained gastrointestinal discomfort, including postprandial pain, vomiting and nausea. An elevated PSV in the renal artery might suggest possible ischaemia, prompting additional imaging examinations such as ultrasound, CTA, or MRA to evaluate suspected CAAOD cases.

This study has several limitations. The inability to perform histopathological analysis in the abdominal aorta limited the pathological characterisation of the disease. The reliance on imaging leaves the possibility of various pathologies. Additionally, the rarity-based small sample size constrains the strength of the study. In the future, a cohort study is imperative to screen the abdominal aorta in patients with APS systemically. Further research into the underlying mechanisms is essential to improve our understanding of this distinctive disease.

## CONCLUSION

In conclusion, this study provides valuable clinical and radiological insights into the rare occurrence of CAAOD in patients with APS. It highlights risk factors such as smoking and hyperhomocysteinaemia, emphasising the importance of early identification of CAAOD in APS and the necessity of therapeutic interventions.

**Contributors** HJ contributed to the statistical analysis and manuscript writing. YS contributed to the collection and management of the clinical data. WL contributed to review the radiological imaging. BL and YX-C contributed to assessment of surgical therapy. MT-L, QW, XP-T and XF-Z equally contributed to the recruitment of patients. JL-Z and YZ provided guidance, checked the data and revised the content. JL-Z acted as the guarantor of the work.

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**Competing interests** None declared.

**Patient consent for publication** Not applicable.

**Ethics approval** This study involves human participants and was approved by Committee of Peking Union Medical College Hospital (JS-2038). Participants gave informed consent to participate in the study before taking part.

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**Data availability statement** Data sharing not applicable as no datasets generated and/or analysed for this study.



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