# Coeliac artery and splenic artery thrombosis complicated with splenic infarction 7 days following the first dose of Oxford vaccination, causal relationship or coincidence?

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## SUMMARY

The novel coronavirus SARS-CoV-2 became a global pandemic in late 2019, and is still ongoing in 2021 causing significant morbidity and mortality. The advent of vaccinations heralded the turning of the tide. The Oxford jab, a vector-based vaccine was favoured due to its low cost and ease of storage. However, its potential association with thromboembolic adverse events resulted in controversy and disrupted its roll-out and use. The aetiopathogenesis of these thromboembolic events and its association with the Oxford vaccine are still speculative and uncertain, more so in the background of SARS-CoV-2 infection being highly thrombogenic in its own right. This paper presents a case of an otherwise healthy 50-year-old Caucasian man who developed acute abdominal pain 7 days following the first dose of Oxford vaccine and was found to have coeliac and splenic artery thrombosis complicated with splenic infarction.

## BACKGROUND

Corona viruses (CoV's) have resulted in pandemics across Asia and the Middle East over the last two decades, for example, SARS-CoV-2 and Middle East respiratory syndrome-CoV. The novel coronavirus SARS-CoV-2 which originated in late 2019 became a global pandemic and has caused significant worldwide morbidity and mortality, with the global death toll reaching 3 million by April 2021.[1] The advent of different vaccinations heralded the turning of the tide as messenger RNA, protein subunit and vector-based vaccines showed good immune response against SARS-CoV-2. The Oxford vaccine (ChAdOx1 nCov-19) which is based on a chimpanzee adenovirus vector with inclusion of genetic sequences of the spike protein in SARS-CoV-2 has shown to elicit good immune response with an efficacy of 81.3% with two doses spaced 12 weeks apart.[2] It became a forerunner among its competitors due to its ease of storage, low cost per dose and was being globally deployed as it had been proven efficacious and safe by phase III clinical trials.[2] However, concerns have been raised regarding its safety after a number of reports of thromboembolic events across Europe following its use.[3] The European Medicine Agency (EMA) recently acknowledged the potential causal relationship between the Oxford vaccine and thromboembolic events.[4] Cerebral vascular accidents and cardiopulmonary embolic complications together with deep vein thrombosis make up most of these noted events.[5] Only one prior splenic infarction was recorded by AstraZeneca's postmarketing adverse event safety database.[5] We report a case of coeliac and splenic artery thrombosis with associated splenic infarction a week following the first dose of the Oxford vaccine.

## CASE PRESENTATION

A previously healthy 50-year-old Caucasian man presented with sudden-onset left-sided abdominal pain of 6 hours' duration to the emergency department. The pain was sharp in nature and persisted and worsened throughout the day to an intensity of 8/10 with poor response to oral paracetamol, necessitating hospital admission. He had been well prior to his presentation on the same morning with no other gastrointestinal or urological symptoms. He denied constitutional symptoms as well. He had not experienced any recent respiratory symptoms or fever and had not had COVID-19 previoulsy . Other than smoking 5–10 cigarettes per day for a decade, he had no significant past medical, surgical or family history of relevance and was not on any routine medication, prescription or off-label. He denied any history of palpitations. He had received the first dose of the Oxford vaccine 7 days prior to his current presentation.

The general examination was normal. He was afebrile with a temperature of 36.3°C. His pulse was 73 beats per minute and regular with a blood pressure of 125 mm Hg systole and 62 mm Hg diastole. His on-air saturation was 99%. His respiratory and cardiovascular system examination was normal. The abdomen on inspection appeared normal with no asymmetry or distension. It was soft on palpation with tenderness being noted on left flank and lower quadrant on deep palpation with no rebound tenderness. Percussion note was normal with auscultation revealing only bowel sounds of normal frequency.

## INVESTIGATIONS

His 12-lead electrocardiograph demonstrated normal sinus rhythm. The initial venous blood gas demonstrated a marginally elevated serum lactate level of 2.45 mmol/L (0.2–1.80) with a pH of 7.335 (7.35–7.45). The whole blood analysis revealed primarily an isolated leucocytosis with elevated count of $18.0 \times 10^9$/L (4–11) with predominant

1

**Table 1** Baseline laboratory parameters

| Investigations and reference ranges | Values |
|---|---|
| **Full blood count** | |
| White blood cells (x10$^9$/L (4–11)) | 18 |
| Neutrophil (x10$^9$/L (7.5–11)) | 15.3 |
| Lymphocytes (x10$^9$/L (1.5–4.0)) | 1.9 |
| Haemoglobin (g/dL (11-15)) | 167 |
| PCV (L/L (0.4–054)) | 0.469 |
| Platelet counts (x10$^9$/L (150 000–450 000)) | 340 |
| **Renal functions** | |
| Serum sodium (mmol/L (135–145)) | 139 |
| Serum potassium (mmol/L (3.5–5.1)) | 4.2 |
| Serum creatinine (µmol/L (44–97)) | 61 |
| Blood urea (mmol/L (2.5 to 7.1)) | 4.6 |
| EGFR mL/min/1.73 m$^2$ | >90 |
| **Inflammatory markers** | |
| C reactive protein (<5 mg/dL) | 5 |
| **Coagulation screen** | |
| aPTT (23.0–31.0 s) | 21.5 |
| PT (9.5–13.5 s) | 12 |
| TT (13.7–19 s) | 15 |
| Fibrinogen (1.8–3.5 g/L) | 3.3 |
| **Liver functions** | |
| Bilirubin (0–21 umol/L) | 10 |
| Alanine transaminase (<41 IU/L) | 39 |
| Albumin (35–50 g/L) | 49 |
| Alkaline phosphate (30–130 U/L) | 105 |
| Total protein (60–80 g/L) | 70 |
| Amylase (28–100 IU/L) | 62 |
| **Thrombotic screening** | |
| Anticardiolipin Ab IgG (GPL U/mL (<10)) | 0.8 |
| Anticardiolipin Ab IgM (MPL U/mL (<10)) | 2.7 |
| B 2 glycoprotein IgG Ab (u/mL (<10)) | <1 |
| B 2 glycoprotein IgM Ab (u/mL (<10)) | <3 |
| D –dimer ((ug/L) <500) | 3026 |
| Fibrinogen (g/L (1.8–3.5)) | 3.3 |
| JAK2 V617F Mutation | Negative |
| **Viral screening** | |
| Hepatitis B surface antigen | Negative |
| Hepatitis C antibodies | Negative |
| Anti HIV ½ | Negative |
| SARS-CoV-2 RT polymerase | Negative |
| **Lipid profile** | |
| Cholesterol (<5.2 mmol/L) | 4.9 |
| Triglycerides (0.8–2.3 mmol/L) | 1.9 |
| HDL (0.9–1.6 mmol/L) | 0.8 |
| Non-HDL | 4.1 |

aPTT, Activated partial thromboplastin time; EGFR, Estimated glomerular filtration rate; GPL, IgG Phospholipid Units; HDL, High-density lipoproteins; MPL, IgM Phospholipid Units; PCV, Packed cell volume; PT, Prothrombin time; TT, Thrombin time.

neutrophilia of 15.3×10$^9$/L (2–7.5). The platelet count was normal with a value of 340×10$^9$/L (150–450). The rest of the investigations including liver functions, renal functions, coagulation profile(except for a marginally prolonged aPTT) and C reactive protein were normal (table 1).

Serum fibrinogen level was normal with a value of 3.3 g/L (1.8–3.5). The urine was dipped and was negative for leukocytes, blood, protein and nitrites. His SARS-CoV-2 nasal and throat swab reverse transcription polymerase chain reaction (RT-PCR)



**Figure 1** Contrast-enhanced CT mesenteric artery angiogram (indicated by arrow) demonstrates a thrombus in the proximal coeliac axis just before giving off the left gastric artery branch.

test was negative. In view of the disproportionate level of symptoms, an urgent contrast-enhanced CT of the abdomen and pelvis was done which demonstrated a non-occlusive thrombus in the coeliac trunk with 50% luminal obstruction, extending into the distal splenic artery with concomitant regional splenic infarct with no bowel ischaemia and no associated renal abnormality or lymphadenopathy. To further clarify this, a selective CT mesenteric angiogram was done which confirmed the thrombus in the proximal coeliac axis (figure 1) with non-opacification of left gastric artery, and thrombus involving the splenic artery (figure 2) causing lower pole splenic infarction (figure 3). The D-dimer levels were checked in retrospect to imaging findings immediately and was grossly elevated with a value of 3026 ug/L (<500). Subsequent investigations with anticardiolipin antibodies and beta-2 glycoprotein antibodies were found to be negative (table 1). Transthoracic two dimensional echocardiogram did not reveal left ventricular thrombus and normal valve morphology was noted.

## TREATMENT
The patient was given analgesia in the form of oral codeine 60 mg, intravenous morphine 5 mg along with intravenous ondansetron 4 mg for pain relief. Following the discovery of coeliac artery and splenic artery thrombus and after haematology consult, he was initiated on anticoagulation with subcutaneous low-molecular-weight heparin (LMWH) of 125 mg as platelet counts remained normal (and the likelihood of anti-PF4/heparin



**Figure 2** Contrast-enhanced CT mesenteric artery angiogram (indicated by arrow) demonstrates a thrombus in the splenic artery.

**Figure 3** Contrast-enhanced CT mesenteric artery angiogram (indicated by arrow) demonstrates splenic infarction of the lower pole.

antibody mechanism being the primary pathology was less likely). He was reviewed by the surgical team and the vascular team and as he remained clinically stable it was decided for conservative management. He was observed for 3 days as an inpatient and with no further evolution of symptoms he was discharged with a plan for follow-up with haematology and vascular team as an outpatient with the continuation of LMWH at the same dose.

## OUTCOME AND FOLLOW-UP
Following discharge, he was asymptomatic. He was reviewed in the vascular surgical clinic and he remains well to date.

## DISCUSSION
Coeliac artery thrombosis is very uncommon. It can occur due to embolic phenomenon by atrial fibrillation, or thrombosis due to atherosclerosis, vasculitis, malignancies and coagulation disorder.[6 7] Advanced age is considered a risk for this phenomenon. The spectrum of presentation can also be highly variable, for example, from dyspeptic symptoms to acute abdomen, and symptoms depend on the extent of ischaemic territory and collateral perfusion.[8 9] This can lead to misdiagnosis, stressing the importance of imaging in its accurate diagnosis.[8] Below we consider the possible mechanisms that may have led to this phenomenon which includes vaccine mediation, thrombotic event due to asymptomatic post-COVID-19, inherent risk factors including atherosclerosis resulting in plaque rupture and prothrombotic states and discuss their individual merits in our patient.

Based on the updated release of the COVID-19 vaccine summary of yellow card reporting on the 9th of June 2021 by the Medicine and Healthcare products Regulatory Authority, 390 thromboembolic events were documented across the UK following the use of AstraZeneca vaccine. The affected age of these patients ranged from 18 to 93 years with most being less than 60 years. The events were also relatively more notable in females (53%).[10] The spectrum of events was extensive and included cerebrovascular accidents, acute myocardial infarction, deep vein thrombosis, pulmonary embolism, multiple thrombosis and cerebral venous sinus thrombosis (CVST).[5 11 12] Coeliac artery thrombosis following AstraZeneca vaccine has only been reported rarely.[5] The mean age for CVST was 46 years and for all other thromboembolic events combined it was 54 years.[10] Thromboembolic events have been noted even during initial clinical trials of the Oxford vaccine, although the number was overall lower in the vaccinated group without an increased risk of bleeding.[13] But in a population based cohort study by Pottergård

*et al*, in Norway and Denmark it was noted that the overall risk of venous thromboembolism to be overall doubled, with disproportionately higher risk in certain subtypes, for example, CVST by as much as 20-fold.[14] As this population mainly composed of healthcare staff, this could very well be an underestimation when extrapolating to the general population as a whole.

The exact aetiopathogenesis resulting in these thrombotic events following the Oxford vaccine are still under investigation and are speculative. However, certain associations are being more frequently observed. Greinacher *et al*, in a paper in preprint, postulates the role of anti-PF4/heparin antibodies and a mechanism similar to heparin-induced thrombocytopaenia with an alternate serological profile as a potential cause for the Oxford vaccine to cause thrombosis in those with simultaneous presence of thrombocytopaenia complicated with a thromboembolic event.[15] Additionally, in certain cases the presence of thrombocytopaenia associated with bleeding complicated with thromboembolism questions the potential role of disseminated intravascular coagulation in its aetiology.[11] A novel alternate mechanism has also been suggested by Kowarz *et al*, in a paper in pre-print who hypothesise that after vaccination, a coded protein precipitates thromboembolic events when it binds to ACE-2 expressed in endothelial cells in the blood vessel. They named this 'vaccine-induced COVID-19 mimicry'.[16] Regardless of the mechanism, this potential association of thromboembolic events following the use of the Oxford vaccine has eroded the faith of the public and governments alike and resulted in the disruption of the vaccine roll-out in Europe and Canada as some nations ceased its deployment pending further investigation.[3 13] The European Medicine Authority and the WHO both initially independently impressed on the lack of causal association of these events due to the Oxford vaccine and encouraged its use as the overall benefit of the vaccine outweighed its risks.[3] Furthermore, an independent analysis of the Danish National patient registry by Østergaard *et al*, comparing the incidence of thromboembolic events in the prevaccine era and following the use of Oxford vaccine roll-out concluded that the use of Oxford vaccine did not increase the relative incidence of thromboembolic events in comparison to the expected incidence among the general population.[12]

It is not possible to unequivocally associate an adverse event following immunisation to the vaccine directly. The event can be due to the vaccine product itself or quality of the product. It can be due to inappropriate handling of the product or can even be anxiety related. Otherwise it can be completely coincidental. To arrive at a conclusion there has to be an assessment at individual and the population level with data analysis in a structured pathway as specified by the WHO.[17 18] However, a recent update by the EMA in April 2021 suggested a causal relationship between thrombotic events in the presence of low platelet count as plausible, while still re-enforcing that the overall the benefit outweighs the risk.[4] This has led to recognition of the phenomenon of SARS-CoV-2 vaccine induced immune thrombotic thrombocytopaenia.[19] Based on available evidence an initial guidance was released where time since vaccination (between 4 and 28 days since vaccination) and low platelet counts ($<150 \times 10^9$/L) were the key determining factors for at risk individuals. This was followed by a release of a broad guidance for investigating and overall management of gastrointestinal manifestations secondary to vaccine-induced thrombosis. The guidelines however suggest to establish definitive thrombocytopaenia before proceeding with further haematological and imaging investigations in pursuit of thromboembolic events suspected of being vaccine induced. The role of D-dimer and

fibrinogen levels have been relegated to a second tier for confirmation rather than being used for initial primary workup.[20 21] However, the mechanism and relationship of the Oxford vaccine causing thromboembolic events in the population in the absence of thrombocytopaenia is still uncertain and remains to be elucidated despite the number of reported incidences. Our patient had a normal platelet count, but grossly elevated D-dimer levels in the presence of thrombosis. This brings into question the role of extending the spectrum of initial screening mechanisms when clinical suspicion is high to ensure a clinical diagnosis is not missed, given the relative lack of information on the different potential mechanisms that maybe leading to thromboembolic phenomenon following use of the Oxford vaccine.

It is also established that COVID-19 infection is prothrombotic. An extensive spectrum of arterial and venous thrombotic and thromboembolic events have been documented in literature during acute infections, even when adequately anticoagulated, impressing on the role thrombogenesis plays in the aetiopathogenesis of COVID-19.[22] A case report by Del Nonno et al, documents an asymptomatic patient who was initially positive and subsequently repeatedly tested negative who thereafter went on to develop a fatal arterial thrombotic event a month following initial diagnosis,[23] further stressing the role of thrombogenicity and its duration of impact following COVID-19 even in asymptomatic patients. Thus, it is not unreasonable to question these episodes of thrombo-embolism as a spectrum of post-COVID-19 syndrome or period,[11] occurring incidentally in the postvaccination context. Our patient had no symptoms preceding his vaccination period and was well. Furthermore, he was rapid antigen negative on admission and when subsequently tested he was also SARS-CoV-2 RT-PCR negative thus making an active infection unlikely and asymptomatic post-COVID-19 period less likely as well.

The other potential differential which could have caused coeliac artery thrombosis in our patient was atherosclerotic plaque rupture as this is an established risk factor for this phenomenon. Our patient had no other risk factor predisposing him to atherosclerosis other than a history of smoking. He was not a known vasculopath nor did he have any suggestive symptoms and he had no prior cardiac symptoms of relevance. Furthermore, coeliac artery thrombosis secondary to this aetiology is usually in the setting of chronic atherosclerosis, which casts doubt of it being the primary aetiopathogenesis in our patient.[24] Another alternate consideration is an intrinsic prothrombotic state. Our patient had no family history and his prothrombotic screen was negative, making an intrinsic cause less likely. But he did have a mildly shortened aPTT, which is a recognised risk factor for mostly venous but also arterial thrombosis.[25 26] However, this hypothesis and association has also been challenged.[27] Furthermore, our patient never had prior venous embolic events and incidences of low aPTT being the sole cause for coeliac artery thrombosis have not been previously reported making this association rather unlikely although not impossible.

Our patient had overlapping risk factors for both coeliac trunk thrombosis (age and potential atherosclerosis) as well as postvaccine thromboembolism (age and timeline of vaccination) and hence falls into the spectrum of a candidate who could be considered as at risk for thromboembolic complications due to the AstraZeneca vaccine. However a definitive causal relationship cannot be proven in our case report other than to speculate based on the association between the time of vaccine administration and the thromboembolic presentation in this otherwise healthy subject. Regardless, there is a scarcity of data in terms of demographics of the populace affected by these events (which may be skewed by the mechanism of vaccine roll-out dependant on age and priority), the time since vaccination to symptom onset and the varying presentation of the clinical picture due to lack of reporting or under-recognition.[12] Thus, it would be prudent to document these thromboembolic events in literature as it would hopefully contribute to greater understanding of this potential phenomenon in future.

## LIMITATIONS

Given the infancy of the state of vaccine roll-out and the knowledge on potential spectrum of postmarketing adverse events, it is still too early to either associate or disregard this arterial thromboembolic event as associated with the Oxford vaccine. As of now, it is still not definitive in its association given the absence of thrombocytopaenia, which is currently the only acknowledged scenario where the Oxford vaccine has been found to cause thrombo-embolic events. However, guidance is rapidly changing and greater recognition of these rare incidences may aid in achieving a better understanding of the potential incidence and spectrum of thromboembolic events following the use of Oxford vaccine.

### Patient's perspective

I initially thought it was trapped wind or constipation. After hearing the diagnosis I thought it could be due to the vaccine. I felt better after receiving the enoxaparin and the pain rapidly subsided. I am feeling better postdischarge, though I still feel anxious.

### Learning points

► Risk stratification and clinical assessment should be used for postvaccine presentations suggestive of thromboembolism. However, if the presentation is highly suggestive, clinical judgement should take precedence to broaden the scope of initial investigation so as to arrive at a diagnosis while still considering alternate aetiologies.
► There is a possible causal relationship between the Oxford vaccine and thromboembolic phenomenona. However, the full spectrum of mechanisms causing this complication need to be better understood and further defined in order to streamline the choice of screening and diagnostic investigations and to decide on appropriate initial management.
► The spectrum of postvaccine thromboembolism both in its manifestations and presentation can be extensive. Reporting of such clinical events will help further our overall understanding of this phenomenon.

## REFERENCES

1 WHO. WHO Coronavirus(COVID 19) Dashboard Word Health Organization, 2021. Available: https://covid19.who.int/table [Accessed 17 Apr 2021].

2 Knoll MD, Wonodi C. Oxford-AstraZeneca COVID-19 vaccine efficacy. *Lancet* 2021;397:72–4.

3 Mahase E. Covid-19: who says rollout of AstraZeneca vaccine should continue, as Europe divides over safety. *BMJ* 2021;372:n728.

4 EMA. AstraZeneca's COVID-19 vaccine: EMA finds possible link to very rare cases of unusual blood clots with low blood platelets: European Medicine Agency, 2021. Available: https://www.ema.europa.eu/en/news/astrazenecas-covid-19-vaccine-ema-finds-possible-link-very-rare-cases-unusual-blood-clots-low-blood [Accessed 16 Apr 2021].

5 Signal assessment report on embolic and thrombotic events (SMQ) with COVID-19 Vaccine (ChAdOx1-S [recombinant]) – COVID-19 Vaccine AstraZeneca (Other viral vaccines), 2021. Available: https://www.ema.europa.eu/en/documents/prac-recommendation/signal-assessment-report-embolic-thrombotic-events-smq-covid-19-vaccine-chadox1-s-recombinant-covid_en.pdf [Accessed 14 Apr 2021].

6 Keskin HA, Yetisir F, Bayram H, *et al*. Celiac artery thrombosis and superior mesenteric artery stenoses with essential thrombocythemia: a case report. *Case Rep Med* 2012;2012:1–3.

7 Kelekis NL, Athanassiou E, Loggitsi D, *et al*. Acute occlusion of the celiac axis and its branches with perforation of gastric fundus and splenic infarction, findings on spiral computed tomography: a case report. *Cases J* 2010;3:82.

8 Saravana K, Zainal AA, Lee SK. Coeliac artery thrombosis: an uncommon cause of an acute abdomen. *Med J Malaysia* 2011;66:273–5.

9 Jerraya H, Sbaï A, Khalfallah M, *et al*. Acute celiac trunk thrombosis revealed by biliary peritonitis. *Diagn Interv Imaging* 2015;96:1199–201.

10 MHRA. Coronavirus Vaccine - summary of Yellow Card reporting: MHRA, 2021. Available: https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/994726/20210616_Coronavirus_vaccine_-_summary_of_Yellow_Card_reporting__v2_-_clean.pdf [Accessed 20 Jun 2021].

11 Mahase E. Covid-19: AstraZeneca vaccine is not linked to increased risk of blood clots, finds European medicine agency. *BMJ* 2021;372:n774.

12 Østergaard SD, Schmidt M, Horváth-Puhó E, *et al*. Thromboembolism and the Oxford–AstraZeneca COVID-19 vaccine: side-effect or coincidence? *The Lancet* 2021;397:1441–3.

13 Dyer O. Covid-19: EMA defends AstraZeneca vaccine as Germany and Canada halt rollouts. *BMJ* 2021;373:n883.

14 Pottegård A, Lund LC, Karlstad Øystein, *et al*. Arterial events, venous thromboembolism, thrombocytopenia, and bleeding after vaccination with Oxford-AstraZeneca ChAdOx1-S in Denmark and Norway: population based cohort study. *BMJ* 2021;373:n1114.

15 et alGreinacher AT, Warkentin T, Weisser ET. A prothrombotic thrombocytopenic disorder resembling heparin-induced thrombocytopenia following Coronavirus-19 vaccination, 2021. Available: https://www.researchsquare.com/article/rs-362354/v1 [Accessed 1 Apr 2021].

16 Kowarz E, Krutzke L, Reis J, *et al*. *Vaccine-Induced Covid-19 Mimicry" Syndrome: Splice reactions within the SARS-CoV-2 Spike open reading frame result in Spike protein variants that may cause thromboembolic events in patients immunized with vector-based vaccines. Preprint from Research Square*, 2021.

17 WHO. *COVID-19 vaccines: safety surveillance Manual- module: responding to adverse events following COVID-19 immunization (AEFIs)*. WHO, 2020. https://www.who.int/vaccine_safety/committee/Module_AEFI.pdf?ua=1

18 WHO. *Causality assessment of an adverse event following immunization (AEFI)*, 2018: 68.

19 Cines DB, Bussel JB. SARS-CoV-2 vaccine-induced immune thrombotic thrombocytopenia. *N Engl J Med* 2021;384:2254–6.

20 Diagnosis and management of gastrointestinal manifestations of vaccine induced thrombosis & thrombocytopenia (VITT) syndrome, 2021. Available: https://www.rcseng.ac.uk/-/media/files/rcs/coronavirus/vitt-guidance-gi-manifestations-6-may-2021.pdf

21 RCEM R, SAM,EHP,BSNR. Management of patients presenting to the emergency Department/ acute medicine with symptoms, 2021. Available: https://www.rcem.ac.uk/docs/Policy/ED-AM%20%20Vaccine%20pathway%20concerns%20-%20RCP%20-%20SAM%20-%20RCEM.pdf [Accessed 16 Apr 2021].

22 Ahmed S, Zimba O, Gasparyan AY. Thrombosis in coronavirus disease 2019 (COVID-19) through the prism of Virchow's triad. *Clin Rheumatol* 2020;39:2529–43.

23 Del Nonno F, Colombo D, Nardacci R, *et al*. Fatal pulmonary arterial thrombosis in a COVID-19 patient, with asymptomatic history, occurred after swab negativization. *Thromb J* 2021;19:1.

24 Pedersoli F, Schönau K, Schulze-Hagen M, *et al*. Endovascular revascularization with stent implantation in patients with acute mesenteric ischemia due to acute arterial thrombosis: clinical outcome and predictive factors. *Cardiovasc Intervent Radiol* 2021;44:1030–8.

25 Abdullah WZ, Moufak SK, Yusof Z, *et al*. Shortened activated partial thromboplastin time, a hemostatic marker for hypercoagulable state during acute coronary event. *Transl Res* 2010;155:315–9.

26 Legnani C, Mattarozzi S, Cini M, *et al*. Abnormally short activated partial thromboplastin time values are associated with increased risk of recurrence of venous thromboembolism after oral anticoagulation withdrawal. *Br J Haematol* 2006;134:227–32.

27 Shervinrad M, Tormey C, Siddon A. Pt and aPTT are not predictors of thromboembolic events. *Am J Clin Pathol* 2019;152:S36-S.