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Splanchnic vein thrombosis associated with SARS-CoV-2 infection: A VALDIG case–control study

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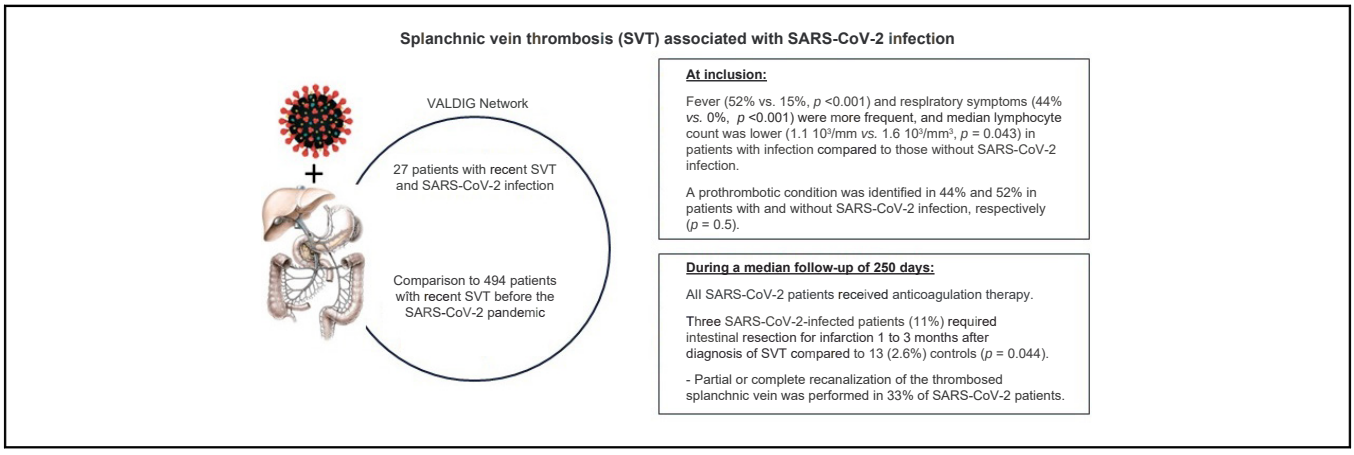
Graphical abstract



Highlights

- We collected data on 27 patients with SARS-CoV-2 with recent splanchnic vein thrombosis.
- Fever and respiratory symptoms were more frequent, and median lymphocyte count was lower in patients with SARS-CoV-2.
- A prothrombotic condition was identified in 44% and 52% of patients with and without SARS-CoV-2 infection, respectively.
- Intestinal resection was required in 11% and 2.6% of patients with and without SARS-CoV-2 infection, respectively (p = 0.044).
- Partial or complete recanalisation of the thrombosed splanchnic vein was performed in 33% of patients with SARS-CoV-2.

Impact and implications

SARS-CoV-2 infection can be associated with recent SVT. SVT occurring during SARS-CoV-2 infection is characterised by a higher frequency of respiratory symptoms and a lower lymphocyte count. Intestinal infarction leading to intestinal resection appears to occur more frequently in patients with SARS-CoV-2.

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Splanchnic vein thrombosis associated with SARS-CoV-2 infection: A VALDIG case–control study

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Background & Aims: Whether severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection is a risk factor for splanchnic vein thrombosis (SVT) is unknown. This study aims to assess the impact of SARS-CoV-2 infection on the presentation and prognosis of recent SVT and to identify specific characteristics of SARS-CoV-2-associated SVT.
Methods: This is a retrospective study collecting health-related data of 27 patients presenting with recent SVT in the context of SARS-CoV-2 infection in 12 Vascular Liver Disease Group (VALDIG) centres and in comparison with 494 patients with recent SVT before the SARS-CoV-2 pandemic.
Results: Twenty-one patients with SARS-CoV-2 had portal vein thrombosis with or without thrombosis of another splanchnic vein, two had superior mesenteric vein thrombosis, one had splenic vein thrombosis, and three had hepatic vein thrombosis. Diagnosis of SVT was made 10 days (95% CI 0–24 days) after the diagnosis of SARS-CoV-2 infection. Fever (52 *vs.* 15%; p <0.001) and respiratory symptoms (44 *vs.* 0%; p <0.001) were more frequent, and median lymphocyte count was lower ($1.1 \times 10^3/mm^3$ *vs.* $1.6 \times 10^3/mm^3$; p = 0.043) in patients with infection than in those without SARS-CoV-2 infection. A prothrombotic condition was identified in 44 and 52% of patients with and without SARS-CoV-2 infection, respectively (p = 0.5). All patients with SARS-CoV-2 received anticoagulation therapy. During a median follow-up of 250 days, three SARS-CoV-2-infected patients (11%) required intestinal resection for infarction 1 to 3 months after diagnosis of SVT compared with 13 (2.6%) controls (p = 0.044). Partial or complete recanalisation of the thrombosed splanchnic vein was performed in 33% of patients with SARS-CoV-2.
Conclusions: SARS-CoV-2 infection can be associated with recent SVT. Intestinal infarction leading to intestinal resection might be more frequent in patients with SARS-CoV-2.
Impact and implications: SARS-CoV-2 infection can be associated with recent SVT. SVT occurring during SARS-CoV-2 infection is characterised by a higher frequency of respiratory symptoms and a lower lymphocyte count. Intestinal infarction leading to intestinal resection appears to occur more frequently in patients with SARS-CoV-2.

Keywords: Splanchnic vein thrombosis; Portal vein thrombosis; SARS-CoV-2 infection.
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Introduction

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection may be associated with haemostasis impairment and may predispose patients to venous and arterial thromboembolism.[1] Although the association between SARS-CoV-2 infection and deep vein thrombosis of the lower limbs and pulmonary embolism has been well documented, little is known about thrombotic events occurring in the splanchnic area.

Several cases of splanchnic vein thrombosis (SVT) that occurred during coronavirus disease-19 (COVID-19) have been reported in the past 3 years.[2–6] Recently, a systematic review summarised the observations made in 21 patients with SARS-CoV-2 infection and acute SVT identified from 21 case reports.[7] This review included a heterogeneous population of patients as some of them had cirrhosis or hepatitis B-related hepatocellular carcinoma, two features that may also be responsible for portal vein thrombosis (PVT). In addition, only half of the patients had a reported workup for an inherited or acquired thrombophilia, and some patients did not receive anticoagulation therapy. Thus, studies including a significant number of well-characterised patients who developed SVT during SARS-CoV-2 infection are needed.

Here, we retrospectively collected health-related data from patients with SARS-CoV-2 infection and recent SVT seen in the hospital centres belonging to the Vascular Liver Disease Group (VALDIG) network. We aimed to assess the prognosis of patients with SARS-CoV-2 presenting with recent SVT. We also aimed to assess patient characteristics and evaluate whether they differ from those of patients presenting with SVT outside the setting of SARS-CoV-2 infection. For this aim, SARS-CoV-2 patients were compared with patients with acute SVT before the SARS-CoV-2 pandemic.

Patients and methods

Patients

This was a retrospective multicentre study supported by the VALDIG network. Patients were included if they presented with recent SVT during a SARS-CoV-2 infection. Data collection started 1 January 2020. The retrospective study design allowed clinicians to include patients in whom SARS-CoV-2 infection and SVT were identified before 1 January 2020. Diagnosis of recent SVT that occurred during SARS-CoV-2 infection was based on the following: (1) imaging by either CT scan or magnetic resonance angiography demonstrating thrombosis of a splanchnic vein, (2) absence of a previous known SVT, and (3) a temporal relationship between the diagnosis of SARS-CoV-2 infection and the occurrence of SVT. Patients were excluded if SVT had been previously identified, in cases of cirrhosis, or when a liver tumour was identified. Diagnosis of SARS-CoV-2 infection was made based on a positive polymerase chain reaction test for SARS-CoV-2.

This study was carried out in compliance with the Declaration of Helsinki and Good Clinical Practice Guidelines. The study protocol was approved by the ethical committee of CUB Hopital Erasme, Brussels, Belgium (local reference: P2021/140). According to its advice, informed consent was waived for the use of anonymous clinical routine data in the setting of a retrospective study. The prospective database from which patients from the control group without SARS-CoV-2 infection were included was approved by the ethical committee (local reference: Sud Mediterranée V on 23 11 2021 2021-A02148-33; France). All these patients gave informed consent.

Data collection and study endpoints

Clinical, biological, and radiological data and data related to anticoagulation therapy, as well as data related to evolution of thrombosis, liver-related events, and patient outcomes, were collected in patients with SARS-CoV-2 and in controls. Determination of SARS-CoV-2 variants was not done.

The primary endpoints were SVT-related events and 6-month mortality. Secondary endpoints were prevalence of an underlying prothrombotic condition and rate of splanchnic vein recanalisation.

Statistical analysis

Quantitative variables are expressed as median and IQR, and categorical variables are expressed as absolute and relative frequencies. Quantitative variables were compared using the Mann–Whiney test. Comparison of qualitative variables was performed using the Chi-square or Fisher exact test, as appropriate. All tests are two-sided, and $p \leq 0.05$ was considered significant.

All data analyses were performed using NCSS 2016 software (NCSS, Kaysville, UT, USA).

Results

Patients

Twenty-seven patients were recruited at 12 VALDIG centres (Table S1). Among the 27 patients, 21 had PVT with or without thrombosis of another splanchnic vein, two had superior mesenteric vein thrombosis, one had splenic vein thrombosis, and three had hepatic vein thrombosis. Diagnosis of SVT was made 10 days (95% CI 0–24 days) after the diagnosis of SARS-CoV-2 infection.

A total of 494 patients with acute SVT without SARS-CoV-2 infection were included in the control group (Table 1). The site and extension of SVT did not differ between patients with and without SARS-CoV-2 infection. Fever (52 *vs.* 15%; p <0.001) and respiratory symptoms (44 *vs.* 0%; p <0.001) were more frequent in patients with SARS-CoV-2 infection than in those without SARS-CoV-2infection. The median lymphocyte count was lower in patients with SARS-CoV-2 infection than in those without SARS-CoV-2 infection ($1.1 \times 10^3/mm^3$ [95% CI $0.9 \times 10^3/mm^3$ to $1.5 \times 10^3/mm^3$] *vs.* $1.6 \times 10^3/mm^3$ [95% CI $1.5 \times 10^3/mm^3$ to $1.7 \times 10^3/mm^3$]; $p = 0.043$). A prothrombotic condition was identified in 44% of patients with SARS-CoV-2 and in 52% of patients without SARS-CoV-2 ($p = 0.5$). Prothrombotic conditions that were identified are reported in Table 2. No differences in associated prothrombotic conditions were observed between patients with and without SARS-CoV-2 infection.

Follow-up data

Three patients received treatment for SARS-CoV-2 infection, and four required ventilation support. All patients with SARS-CoV-2 were treated with anticoagulation. Initial treatments received were low-molecular-weight heparin in 22 patients and direct oral anticoagulants in five patients. During a median follow-up of 250 days (95% CI 83–394 days), no patient with SARS-CoV-2 experienced portal hypertension-related gastrointestinal bleeding or another liver-related event. Three patients (11%) with SARS-CoV-2 infection required intestinal resection for infarction 1 to 3 months after the diagnosis of SVT, which was significantly more frequent than that in the control group (n = 13 [2.6%]; $p = 0.044$). The median time to surgery was 33 days (range 32–84 days) in patients with SARS-CoV-2 and 363 days (95% CI 2–1,210 days) in patients without SARS-CoV-2 ($p = 0.5$). Interestingly, among the three patients with SARS-CoV-2 who required intestinal resection, two had G20210A prothrombin gene mutations (67%) *vs.* one among those who did not require intestinal resection (5%; $p = 0.03$). No patients with SARS-CoV-2 died. At the end of the follow-up period, partial or complete recanalisation of the thrombosed splanchnic vein was observed in eight of 24 patients (33%; three patients had missing data), a frequency that was not different from patients without SARS-CoV-2

Table 1. Characteristics of patients with recent SVT with and without SARS-CoV-2 infection.

Characteristics	Patients with SARS-CoV-2 infection (n = 27)	Patients without SARS-CoV-2 infection (n = 494)	*p* value
Age (range), years	51 (36–53)	47 (45–49)	0.9
Male sex, n (%)	18 (67)	298 (61)	0.7
BMI	26.7 (24.3–29.3)	26.2 (25.6–26.9)	0.5
Fever, n (%)	14 (52)	74 (15)†	<0.001
Respiratory symptoms, n (%)	12 (44)	0 (0)	<0.001
Dyspnoea, n (%)	8 (30)	0 (0)	<0.001
Anosmia, n (%)	6 (25)*	0 (0)	<0.001
Ageusia, n (%)	6 (25)*	0 (0)	<0.001
Abdominal pain, n (%)	24 (89)	404 (82)‡	0.6
Ascites, n (%)	7 (26)	105 (22)§	0.4
CRP (mg/dl)	42 (14–106)	35 (21–45)	0.5
D-dimers (ng/ml)	3298 (850–10,000)	NA	NA
White blood cells ($\times10^3$/mm^3)	6.1 (4.0–10.0)	7.0 (6.6–7.2)	0.5
Polymorphonuclear neutrophils ($\times10^3$/mm^3)	3.8 (2.7–6.2)	4.1 (3.9–4.4)	0.8
Lymphocytes ($\times10^3$/mm^3)	1.1 (0.9–1.5)	1.6 (1.5–1.7)	0.04
Eosinophils ($\times10^3$/mm^3)	0.03 (0–0.10)	0.10 (0.10–0.10)	0.002
Platelets ($\times10^3$/mm^3)	220 (178–301)	257 (240–273)	0.6
AST (IU/ml)	38 (24–43)	32 (30–34)	0.3
ALT (IU/ml)	41 (25–52)	40 (37–43)	0.7
Alkaline phosphatase (IU/ml)	90 (75–134)	83 (79–88)	0.4
Bilirubin (mg/dl)	0.9 (0.6–1.1)	0.6 (0.6–0.7)	0.03
INR	1.2 (1.1–1.3)	1.1 (1.0–1.1)	0.01
Creatinine (mg/dl)	0.9 (0.7–1.0)	0.8 (0.8–0.8)	0.3

Continuous variables are presented as median (IQR). Quantitative variables are expressed as median and IQR, and categorical variables are expressed as absolute and relative frequencies. Quantitative variables were compared using the Mann–Whiney test. Comparison of qualitative variables was performed using the Chi-square or Fisher exact test, as appropriate. All tests are two-sided and $p \leq 0.05$ was considered significant.
ALAT, alanine aminotransferase; ASAT, aspartate aminotransferase; CRP, C-reactive protein; INR, international normalised ratio; NA, not available; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; SVT, splanchnic vein thrombosis.
* Available in 24 patients.
† Available in 482 patients.
‡ Available in 491 patients.
§ Available in 488 patients.

Table 2. Site of SVT and prothrombotic conditions.

Characteristics	Patients with SARS-CoV-2 infection (n = 27)	Patients without SARS-CoV-2 infection (n = 494)	*p* value
Main portal vein thrombosis, n (%)	21 (78)	316 (64)	0.2
Intrahepatic thrombosis, n (%)	18 (69)*	392 (79)	0.2
Superior mesenteric vein thrombosis, n (%)	14 (52)	292 (60)¶	0.4
Splenic vein thrombosis, n (%)	10 (37)	179 (37)**	1.0
Other thrombosis, n (%)	5 (19)	68 (14)††	0.6
Prothrombotic condition, n (%)	11 (44)*	256 (52)	0.5
Myeloproliferative disorder	4 (15)*	57 (14)	0.8
Polycythaemia vera	3 (11)	32 (7)	
Essential thrombosis	1 (4)	27 (6)	
Myelofibrosis	0 (0)	1 (0)	
Unclassified	0 (0)	7 (1)	
Jak-II V617f mutation	3 (12)†	59 (12)‡‡	1.0
Calreticulin mutation	0 (0)‡	5 (3)§§	1.0
Factor V gene mutation	3 (12)§	25 (5)	0.15
G20210A prothrombin gene mutation	3 (12)§	33 (7)¶¶	0.5
Antiphospholipid syndrome	2 (8)*	15 (3)***	0.5
Protein C (%)	88 (74–100)	n.a.	n.a.
Protein S (%)	73 (62–86)	n.a.	n.a.
Antithrombin (%)	94 (87–100)	n.a.	n.a.

Continuous variables are presented as median (IQR). Quantitative variables are expressed as median and interquartile range (IQR), and categorical variables are expressed as absolute and relative frequencies. Quantitative variables were compared using the Mann–Whiney test. Comparison of qualitative variables was performed using the Chi-square or Fisher exact test, as appropriate. All tests are two-sided, and $p \leq 0.05$ was considered significant.
n.a., not available; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; SVT, splanchnic vein thrombosis.
* Available in 26 patients.
† Available in 24 patients.
‡ Available in 12 patients.
§ Available in 25 patients.
¶ Available in 483 patients.
** Available in 480 patients.
†† Available in 492 patients.
‡‡ Available in 488 patients.
§§ Available in 198 patients.
¶¶ Available in 486 patients.
*** Available in 489 patients.

infection (32.7%; $p = 1.0$). No difference was observed in terms of rates of recanalisation between patients with SARS-CoV-2 without a prothrombotic condition and those with a prothrombotic condition (36 *vs.* 30%; $p = 1.0$). The site or extension of SVT was not associated with the rate of recanalisation ($p = 0.2$).

Discussion

Although venous thrombosis is a common feature among patients with SARS-CoV-2 infection, SVT has only been described in case reports or small series. A recent systematic review was not able to provide a clear view on the impact of SARS-CoV-2 infection on SVT presentation or on SARS-CoV-2 outcomes.[7] Thanks to a joint effort of members of the VALDIG network, despite the rarity of SARS-CoV-2-associated SVT, this study is able to fill this gap and improve our knowledge of this complication of SARS-CoV-2 infection.

The main result of this study is that SARS-CoV-2 infection wase identified in a significant number of patients with recent SVT. Whether SARS-CoV-2 infection is directly responsible for SVT remains uncertain. Of note, SARS-CoV-2 infection was the only prothrombotic condition encountered in most patients, whereas another prothrombotic condition was identified in 44% of patients in this study. Overall, more than 90% of patients had a complete workup to investigate the possible presence of additional prothrombotic disorders. This result indicates that when identifying SARS-CoV-2 infection and acute SVT, full evaluation for other aetiologies is needed. In line with these results, no differences in the prevalence of another underlying prothrombotic condition or regarding associated prothrombotic conditions were observed between patients with and without SARS-CoV-2 infection. The main characteristics of SARS-CoV-2-associated SVT were the presence of fever, dyspnoea, anosmia, ageusia, and lymphopaenia, all features that are frequently encountered during SARS-CoV-2 infection.[8] Hence, testing for SARS-CoV-2 should be performed in all patients presenting with SVT, especially when respiratory symptoms and/or lymphopaenia are present.

Another important point is that SARS-CoV-2 infection may be associated with a higher risk of intestinal infarction requiring resection despite a shorter follow-up than that in the control group. Indeed, 11% of the present study population who had SARS-CoV-2 infection underwent intestinal resection, a higher incidence than that in the control group (2.4%) and in a prospective study performed in 102 patients with acute PTV (2.1%).[9] This finding suggests that severity of thrombotic events occurring in the splanchnic area during SARS-CoV-2 infection may be more important than that outside the setting of SARS-CoV-2 infection, although the small sample size of this study precludes drawing robust conclusions. However, even if long-term follow-up data were not available, no patients presented with portal hypertension-related gastrointestinal bleeding or another liver-related event. Anticoagulation therapy was started early on in all patients with SARS-CoV-2 and allowed partial or complete recanalisation of the thrombosed splanchnic vein in 33%, a percentage not different from that in the control group and similar to the one reported in patients with acute PVT (38%).[9] Overall, SVT disease trajectories did not appear to differ between patients with and without SARS-CoV-2 infection with the notable exception of a possible increased risk of intestinal ischaemia requiring resection. This study also did not suggest that SVT impaired the prognosis of SARS-CoV-2 infection as no patients died. However, the short follow-up and the absence of a control group of patients with SARS-CoV-2 infection and without SVT hampered our ability to draw a firm conclusion.

We acknowledge that our study has several limitations that are inherent to its retrospective design. We cannot be sure that all cases of SVT occurring in the setting of SARS-CoV-2 infection within the participating centres have been reported. More specifically, SVT occurring in patients who died from SARS-CoV-2 infection may have been overlooked if imaging dedicated to identifying SVT was not performed. However, the limited number of cases previously reported and the number of patients included in this European registry do not suggest that SVT is a frequent event during SARS-CoV-2 infection, even if the retrospective study design and the small sample size of this study did not allow us to draw robust conclusions.

In conclusion, SARS-CoV-2 infection can be associated with recent SVT. SVT occurring during SARS-CoV-2 infection is characterised by a higher frequency of respiratory symptoms and a lower lymphocyte count. Intestinal infarction leading to intestinal resection appears to occur more frequently in patients with SARS-CoV-2.

Abbreviations

COVID-19, coronavirus disease-2019; PVT, portal vein thrombosis; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; SVT, splanchnic vein thrombosis; VALDIG, Vascular Liver Disease Group.

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The authors did not receive any funding for this study.

Conflicts of interest

The authors have no competing interests to declare.

Please refer to the accompanying ICMJE disclosure forms for further details.

Authors' contributions

Study concept and design: PD. Acquisition of data: PD, P-ER, Apl. Analysis and interpretation of data: PD, Apa, LE, VLM, FA, AB, J-PC, LC, IC, EL, BP, DS, CB, OG, IO, AN, P-ER, Apl. Drafting of the manuscript: PD, P-ER, Apl. Critical revision of the manuscript for important intellectual content: PD, Apa, LE, VLM, FA, AB, J-PC, LC, IC, EL, BP, DS, CB, OG, IO, AN, P-ER, Apl. Statistical analysis: PD. Study supervision: PD. Approved the final version of the manuscript: all authors.

Data availability statement

Data are available on request.

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Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jhepr.2023.100894.

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