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Infective pylephlebitis complicating chronic portal vein thrombosis in a patient with hepatitis B-related cirrhosis: a case report and review of the literature

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Case Report

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Abstract

Background

Infective pylephlebitis is suppurative thrombophlebitis of the portal venous system. It is rare, lethal when recognition is delayed, and almost always described in patients without pre-existing portal vein thrombosis (PVT). In cirrhosis with chronic PVT, anticoagulation is constrained by varices and coagulopathy, and no guidance covers the infected thrombus.

Case presentation:

A 68-year-old Han Chinese man with hepatitis B-related decompensated cirrhosis, previous variceal bleeding treated by splenectomy, and chronic PVT maintained on low-molecular-weight heparin (LMWH) for 5 years presented with fever, abdominal pain and back distension. Contrast-enhanced computed tomography (CT) showed occlusion of the main portal vein and superior mesenteric vein, periportal collaterals and right colonic mural oedema; blood culture grew non-O1/non-O139 *Vibrio cholerae*. Continuous intravenous unfractionated heparin (UFH) targeting an activated partial thromboplastin time (APTT) of 50–70 s was started together with cefoperazone/sulbactam. On day 3 the patient declined the infusion pump and was changed to subcutaneous LMWH. On day 5 the pain worsened, the APTT fell to 31.3 s, the D-dimer rose from 2.07 to 18.74 mg/L and CT showed thrombus extending into the collateral vessels. Continuous UFH was resumed after multidisciplinary review. Over the next two weeks the fever and abdominal pain resolved and the diarrhoea improved; C-reactive protein fell from 20.14 to 0.79 mg/L, and the portal vein was patent on day 12 and fully patent by day 31. Ascites that had persisted for years and diarrhoea present for 5 years resolved completely.

Conclusions

Chronic PVT does not protect against superimposed infective pylephlebitis, and bloodstream infection can convert a stable thrombus into a propagating one within days. High fever, worsening abdominal or back pain and an abrupt rise in D-dimer should prompt immediate contrast-enhanced CT. Once the diagnosis is established, culture-directed antimicrobials combined with continuous intravenous heparinization, titrated to APTT and interrupted when bleeding risk rises, can achieve recanalization despite previous variceal haemorrhage. Clinical improvement lags behind thrombus resolution, and a high D-dimer in the first week is not a reason to stop heparin.

Background

Portal vein thrombosis (PVT) is one of the most frequent vascular complications of decompensated cirrhosis. Data summarised in the Chinese expert consensus on the management of PVT in cirrhosis indicate a prevalence of 5–20% and an annual incidence of 3–17%; the one-year cumulative incidence is

about 4% in predominantly Child–Pugh class A cohorts but exceeds 16% in class B and C patients [1]. Because chronic PVT produces no specific symptoms and is easily masked by the underlying liver disease, it is often found incidentally, and surveillance with imaging and D-dimer measurement has been proposed for patients at high risk [2].

Infective pylephlebitis — suppurative thrombophlebitis of the portal venous system — is a different and much rarer disorder. A pyogenic focus drained by the portal circulation reaches the portal vein through the mesenteric veins and causes acute suppurative inflammation of the vessel wall with superimposed or accelerated thrombosis. Reported annual incidences are of the order of 0.37–2.7 per 100,000 persons [3]. Mortality is high when recognition is delayed, largely because the early picture is indistinguishable from spontaneous bacterial peritonitis, acute biliary infection or gastroenteritis.

When pylephlebitis develops in a patient who already has cirrhosis and chronic PVT, two difficulties converge. The first is diagnostic: a patient who has lived with abdominal discomfort and diarrhoea for years, and a clinician who has learnt to attribute new symptoms to decompensation, will both tend to dismiss a febrile episode. The second is therapeutic. Anticoagulation in cirrhosis is constrained by thrombocytopenia, coagulopathy and oesophagogastric varices, and active infection adds further uncertainty because sepsis-associated coagulopathy may aggravate both thrombosis and bleeding [4]. The evidence on anticoagulation for cirrhosis-associated PVT has been generated almost exclusively in patients with sterile thrombus [5–9]. How to anticoagulate a patient whose chronic PVT has become infected, and whether the route of administration matters in that situation, is essentially undocumented.

We describe a patient with hepatitis B-related cirrhosis and a 5-year history of chronic PVT maintained on LMWH who developed non-O1/non-O139 *Vibrio cholerae* (NOVC) bacteraemia and infective pylephlebitis with acute thrombus propagation. Continuous intravenous heparinization was followed by complete recanalization of the portal vein and by the unexpected resolution of long-standing ascites and chronic diarrhoea.

Case presentation

This report follows the CARE reporting guideline, and the completed checklist is provided as Additional file 1.

A 68-year-old Han Chinese man was admitted to the Department of Infectious Diseases on 16 September 2026 with a 5-hour history of fever, abdominal pain and back distension. He had hepatitis B-related cirrhosis with a 20-year history of decompensation, including several episodes of bleeding from oesophagogastric varices, for which he had undergone splenectomy; he had since taken tenofovir alafenamide fumarate and ursodeoxycholic acid regularly. Chronic PVT had been diagnosed 5 years earlier and was managed with LMWH 3800 U subcutaneously once daily. On this regimen he had had no further bleeding or documented thrombus extension, and his D-dimer had remained between 0.5 and 1.8 mg/L, but he had persistent mild ascites and five or more loose stools a day. One year before admission he had undergone minimally invasive treatment of a liver malignancy.

On examination the temperature was 39.2°C, pulse 87 beats/min, respiratory rate 22 breaths/min and blood pressure 122/65 mmHg. There was no scleral icterus; palmar erythema and spider naevi were present. The abdomen was flat with a surgical scar, tender in the right upper and mid abdomen without rebound tenderness or guarding; shifting dullness was positive and bowel sounds were 5 per minute.

Laboratory investigations showed a white cell count of 8.3 × 10^9/L, haemoglobin 110 g/L, C-reactive protein 20.14 mg/L and procalcitonin 0.986 ng/mL. Liver biochemistry showed alanine aminotransferase 65 U/L, aspartate aminotransferase 98 U/L, albumin 36.69 g/L, albumin/globulin ratio 1.0, lactate dehydrogenase 308 U/L and total bile acids 24.3 μmol/L. Coagulation studies showed a D-dimer of 2.07 mg/L (1.49–2.49 mg/L over the first 48 hours), prothrombin time 21.0 s and APTT 50.8 s. Contrast-enhanced CT of the abdomen (Fig. 1A) showed post-operative changes of the liver malignancy, cirrhosis with ascites, occlusion of the main portal vein and superior mesenteric vein with multiple periportal collateral vessels, and mural oedema and thickening of the ascending colon and part of the transverse colon with stranding of the adjacent fat. Blood culture subsequently grew NOVC [10, 11].

Continuous intravenous UFH was begun on the day of admission, titrated to a target APTT of 50–70 s, together with intravenous cefoperazone/sulbactam 3.0 g every 12 hours. On day 3 the patient declined the infusion pump and was converted to subcutaneous LMWH 3800 U every 12 hours. On day 5 (20 September) at 17:42 he reported worsening pain in the right abdomen radiating to the right back. The APTT had fallen to 31.3 s (the sample was drawn before the infusion was interrupted) and the D-dimer had risen to 18.74 mg/L. Repeat contrast-enhanced CT (Fig. 1B) showed progression of thrombus within the main portal vein and superior mesenteric vein, new thrombus within several of the collateral vessels, incipient cavernous transformation of the portal vein, and marked tortuosity and dilatation of the abdominal wall and rectal venous plexuses. After multidisciplinary discussion, and after a planned 24-hour interruption for bleeding risk, continuous UFH was resumed at the previous APTT target on 21 September.

Cefoperazone/sulbactam was continued until the inflammatory markers had normalised. UFH was interrupted for 24 hours on two occasions (20 and 24 September) when the bleeding risk was judged to have risen (red columns in Fig. 2) and was converted to LMWH 3800 U every 12 hours before discharge. Ademetionine and ursodeoxycholic acid were given for cholestasis, oral hydrochlorothiazide with spironolactone for ascites, and intravenous albumin, electrolyte replacement and acid suppression as supportive measures; tenofovir alafenamide was continued.

The subsequent course is summarised in Fig. 2. The pain score fell from 4 on 20 September to 3, 1 and 0 on 24 September, 28 September and 17 October. Stool frequency fell from 20 per day at the peak to 10, 7 and finally 1 formed stool per day. Ascites, 39 mm deep on admission, increased to 45 and 50 mm on 20 and 24 September, then fell to 19 mm on 28 September and was undetectable on 17 October. Fever resolved during the second week, with a single transient rise to 38.3°C on 28 September. C-reactive protein fell from 20.14 to 0.79 mg/L and procalcitonin returned to normal. The D-dimer, having peaked at 24.46 mg/L on 24 September, during the second planned interruption, fell to 14.36 mg/L on 28

September and 1.21 mg/L on 17 October. The APTT was 14.4 s at the nadir of the second planned interruption (24 September) and 36.2–37.2 s thereafter. Serial ultrasonography and CT documented the evolution of the portal venous system from occlusion of the main portal vein and superior mesenteric vein with collateral formation on 16 September, through cavernous transformation with partial thrombosis of the collaterals on 20 and 24 September, to a patent main trunk with residual thrombus in its sagittal portion on 28 September and a fully patent main trunk and collaterals on 17 October.

Two features of the recovery were unexpected. Ascites that had been present continuously for years resolved completely once the portal vein had recanalized, and diarrhoea that had been present for 5 years and had been attributed to portal hypertensive enteropathy also resolved; the patient reported one formed stool daily, a pattern he had not experienced for half a decade.

Patient perspective

At the 1-month follow-up visit the patient reported that the change in bowel habit mattered most to him: he now passes one formed stool each morning, which he had not done for five years. He also described complete resolution of the abdominal distension that had been present for years and said that he had returned to his usual daily activities. He had previously accepted both symptoms as unavoidable consequences of his cirrhosis.

Discussion and conclusions

The evidence for anticoagulation in cirrhosis-associated PVT has strengthened considerably over the past decade, although it has been generated in patients with sterile thrombus. In a prospective Italian cohort, Senzolo and colleagues treated patients with cirrhosis and PVT with nadroparin and reported portal vein recanalization in 36% of anticoagulated patients, a significantly lower rate of thrombus progression than in untreated controls and no excess of major bleeding [5]. A systematic review of 16 observational studies [6] and a multicentre study [7] reached concordant conclusions. The meta-analysis by Loffredo and colleagues, pooling 8 studies and 353 patients, reported recanalization in 71% of anticoagulated patients compared with 42% of untreated patients and thrombus progression in 9% compared with 33%, without an increase in overall or variceal bleeding [8]. A network meta-analysis [9] and the Chinese expert consensus [1] both recommend that anticoagulation be actively considered when there is no active bleeding. Chinese series have addressed the risk of portal venous thrombosis after splenectomy with devascularization for portal hypertension, and the factors that predict it [12–17]; such patients form a substantial part of the population in whom maintenance anticoagulation has to be considered. Our patient extends these observations to the very long term: despite recurrent variceal haemorrhage and splenectomy, he received LMWH for 5 years without further bleeding or thrombus extension, with D-dimer stable between 0.5 and 1.8 mg/L. That course supports the feasibility of prolonged maintenance anticoagulation, but it also exposes the limitation of the strategy, because sustained LMWH did not prevent the abrupt thrombotic deterioration that followed bloodstream infection.

The early picture of pylephlebitis superimposed on chronic PVT is notoriously non-specific. The diagnosis should be considered when a patient with cirrhosis develops pain of new character or increased severity, rigors or high fever that cannot be explained by the underlying liver disease, watery or loose diarrhoea, or a rapid accumulation of ascites; all four were present in our patient (Fig. 3). The trap is that each of these features has a plausible alternative explanation in decompensated cirrhosis. Conventional inflammatory markers may mislead, and in patients with bacterial liver abscess the indices that predict progression to sepsis are not the conventional ones alone [18]. In our patient the D-dimer trajectory was the most informative single test: it had been stable between 0.5 and 1.8 mg/L for years and was only 1.49–2.49 mg/L during the first 48 hours, but rose to 18.74 mg/L within hours of the clinical deterioration. Colour Doppler ultrasonography is well suited to surveillance of portal vein calibre, echogenicity, flow direction and portosystemic collaterals [19], but contrast-enhanced CT is required to define the anatomical extent of thrombus, the degree of occlusion and any evidence of intestinal ischaemia, and the consensus recommends early contrast-enhanced CT whenever PVT is suspected clinically or on ultrasonography, or when the patient deteriorates [1]. In our patient two contrast-enhanced CT studies 4 days apart documented the transition from simple occlusion with collateralization to cavernous transformation with fresh thrombus inside the collaterals, a radiological sequence rarely captured so closely within a single admission.

The most instructive aspect of this case is the natural experiment created by the patient's refusal of the infusion pump. Within 48 hours of switching from continuous intravenous UFH to intermittent subcutaneous LMWH, the APTT fell from the therapeutic range to 31.3 s, the D-dimer rose almost tenfold and thrombus extended into vessels that had previously been patent. Compared with subcutaneous LMWH, continuous intravenous UFH has a rapid onset, a half-life of approximately 1–1.5 hours, and an anticoagulant effect that resolves quickly on discontinuation and can be completely reversed with protamine sulfate. In a patient with severe infection, fluctuating hepatic function and a history of variceal bleeding, that controllability is a substantive advantage rather than a matter of convenience, and the episode argues for maintaining the intravenous route through the acute phase before transitioning to LMWH for maintenance (Fig. 4), even though combined and sequential heparin strategies have so far been described mainly as adjuncts to catheter-directed thrombolysis for deep vein thrombosis [20]. Two practical points emerge from the same course. First, clinical improvement lags behind thrombus resolution: the D-dimer continued to rise to 24.46 mg/L on 24 September, during the second planned interruption, and recanalization was demonstrable only on 28 September and complete on 17 October. A rising D-dimer in the first week of apparently adequate anticoagulation is therefore not by itself evidence of treatment failure, and heparin should not be abandoned on that basis. Second, planned brief interruptions were compatible with recovery provided the APTT was monitored closely and the infusion restarted promptly.

The disappearance of long-standing ascites and of 5-year chronic diarrhoea after recanalization deserves comment, although a single case cannot establish causation. Both had previously been attributed to the natural history of cirrhosis and to portal hypertensive enteropathy, and neither had responded to diuretics or to antiviral therapy. Their simultaneous resolution once antegrade portal flow

was restored suggests that, in patients with cirrhosis and PVT, ascites that is disproportionate to the synthetic function of the liver and chronic diarrhoea unexplained by other causes should prompt an assessment of portal vein patency, and that restoration of portal flow may be worth pursuing as a therapeutic goal in its own right rather than as an incidental radiological endpoint. Prospective data are needed before this can be recommended, and we present it as a hypothesis generated by the present case.

Several limitations should be acknowledged. This is a single observation; the anticoagulation regimen was modified partly by patient preference rather than by protocol; and the APTT target of 50–70 s was chosen pragmatically rather than derived from evidence in this population. After the second interruption the APTT ran at 36–37 s, below the acute-phase target, without further thrombus progression, which raises the question whether a target derived from venous thromboembolism practice is appropriate here. The maintenance dose of LMWH (3800 U once daily) is lower than weight-adjusted prophylactic dosing in many guidelines, and the D-dimer range of 0.5–1.8 mg/L that we associate with stability is an observation in one patient rather than a validated target. Finally, follow-up extends to 1 month after admission; whether recanalization is sustained, and whether long-term anticoagulation should continue indefinitely, remains to be determined.

In conclusion, chronic PVT in cirrhosis does not protect against superimposed infective pylephlebitis, and the transition from a stable chronic state to acute thrombus propagation can occur within days of bloodstream infection. The combination of high fever, worsening abdominal or back pain and an abrupt rise in D-dimer should be treated as a warning triad and should prompt immediate contrast-enhanced CT. Once infective pylephlebitis is diagnosed, culture-directed antimicrobial therapy should be combined with continuous intravenous heparinization, titrated to APTT and interrupted liberally when the bleeding risk rises; a history of variceal haemorrhage is a reason for close monitoring rather than an absolute contraindication. Clinicians should also expect a delay between clinical improvement and radiological resolution, and should not interpret a persistently elevated D-dimer in the first week as failure of anticoagulation.

Abbreviations

APTT
activated partial thromboplastin time
CT
computed tomography
LMWH
low-molecular-weight heparin
NOVC
non-O1/non-O139 *Vibrio cholerae*
PVT
portal vein thrombosis

UFH
unfractionated heparin.

Declarations

Ethics approval and consent to participate

This case report was conducted in accordance with the Declaration of Helsinki. The study was reviewed and approved by the Ethics Committee of Tiantai Hospital Affiliated to Hangzhou Medical College (approval number 202610261613000527879).

Consent for publication

Written informed consent for publication of this case report and any accompanying images was obtained from the patient. A copy of the consent form is available for review by the Editor-in-Chief of this journal.

Competing interests

The authors declare that they have no competing interests.

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Author Contribution

JHY and JWY conceived the report. JHY collected and interpreted theclinical data, prepared Table 1 and Figures 2 and 3, and drafted themanuscript. JWY supervised the clinical management, coordinated themultidisciplinary review and critically revised the manuscript. LYcontributed to the clinical management and to data collection. JCperformed and interpreted the contrast-enhanced computed tomographystudies and prepared Figure 1. MMT contributed to the initialassessment and emergency management of the patient. LCC performed andinterpreted the ultrasonographic studies and contributed to thefollow-up imaging. All authors read and approved the final manuscriptand agree to be accountable for all aspects of the work.

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Availability of data and materials

All data generated or analysed during this study are included in this published article and its additional files (Additional file 1: CARE checklist). The original imaging studies are available from the corresponding author on reasonable request.

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Figures

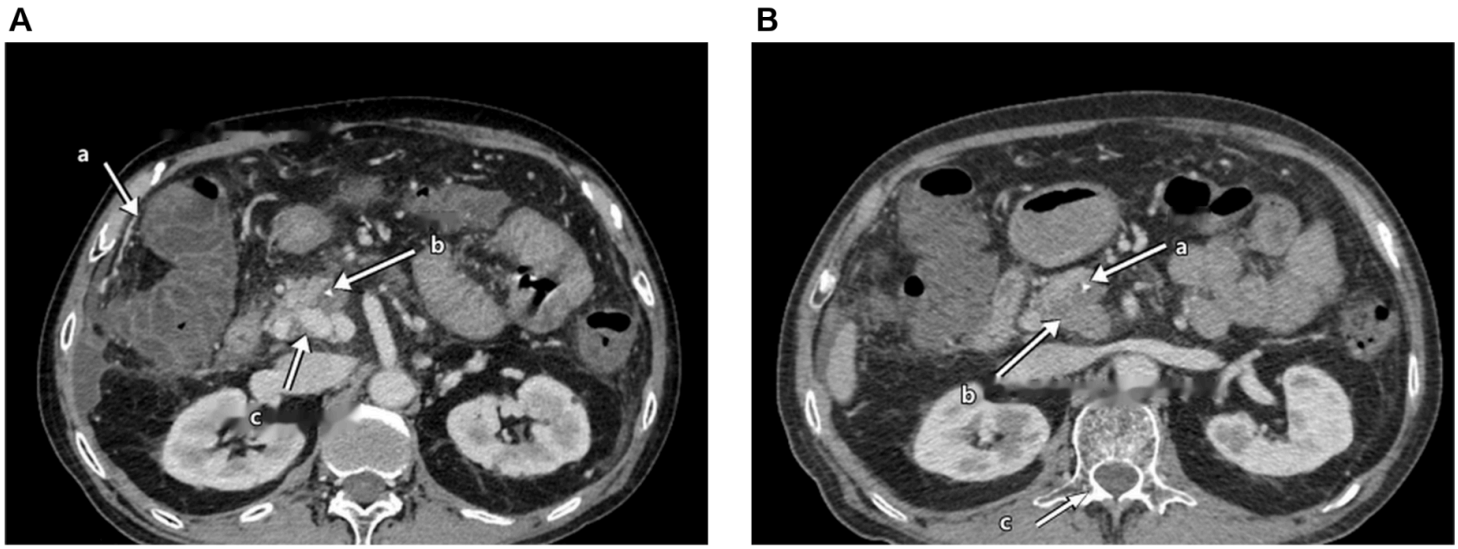


Figure 1

Contrast-enhanced abdominal computed tomography in the portal venous phase. (A) 16 September 2026: marked mural oedema of the ascending colon (arrow a), thrombus in the portal vein (arrow b) and periportal collateral vessels (arrow c). (B) 20 September 2026: persistent thrombus in the portal vein (arrow a), new thrombus within the collateral vessels (arrow b) and an incidental degenerative change of a vertebral appendage (arrow c).

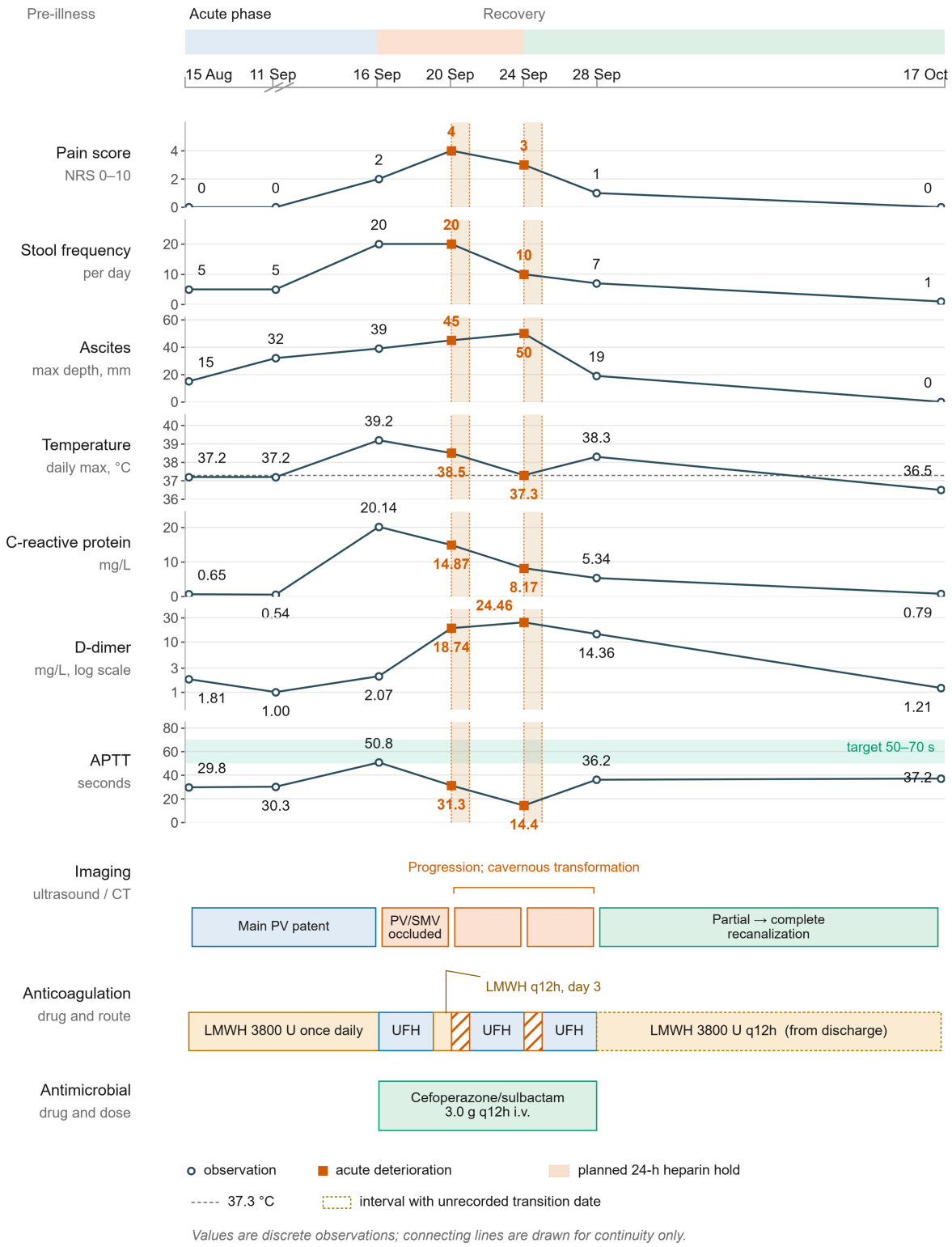


Figure 2

Clinical course, laboratory kinetics and treatment from 15 August to 17 October 2026. Time is drawn to scale; the axis break compresses the pre-illness interval. Rows show, from top to bottom, pain (numeric rating scale 0–10), stool frequency per day, maximum ascites depth on ultrasonography (mm), maximum daily temperature (°C; dashed line, 37.3 °C), C-reactive protein (mg/L), D-dimer (mg/L, logarithmic scale) and activated partial thromboplastin time (s; shaded band, target 50–70 s). Coloured

bars summarise imaging findings (blue, stable or patent; orange, occlusion or progression; green, recanalization), anticoagulation and antimicrobial treatment. Red squares mark the acute deterioration and red columns the two planned 24-hour interruptions of the heparin infusion (20 and 24 September); the blood sample showing an APTT of 31.3 s was drawn before the infusion was stopped. Values are discrete clinical observations and connecting lines are drawn for continuity only; dotted outlines mark intervals whose exact transition dates were not recorded. APTT, activated partial thromboplastin time; CRP, C-reactive protein; LMWH, low-molecular-weight heparin; PV, portal vein; SMV, superior mesenteric vein; UFH, unfractionated heparin.

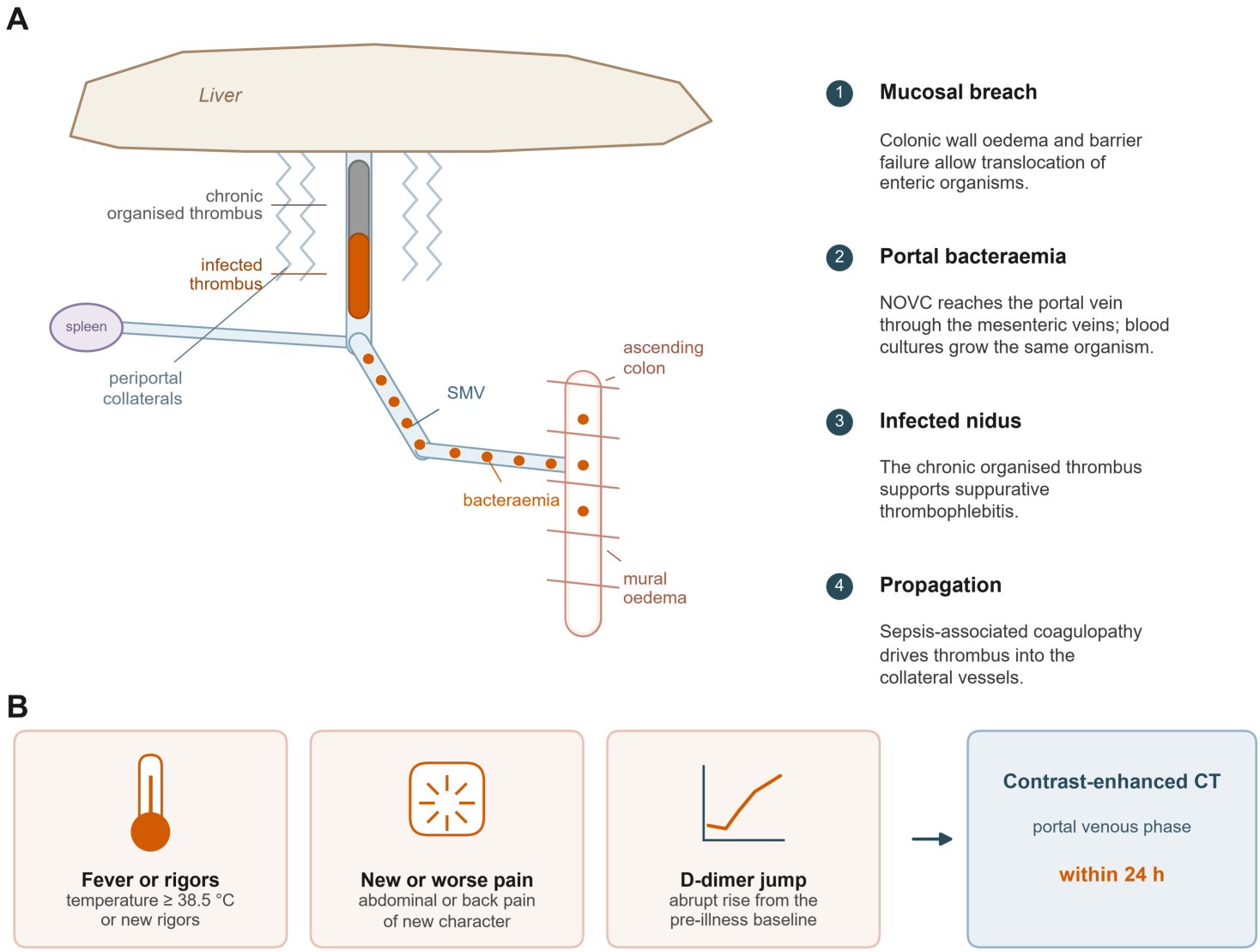
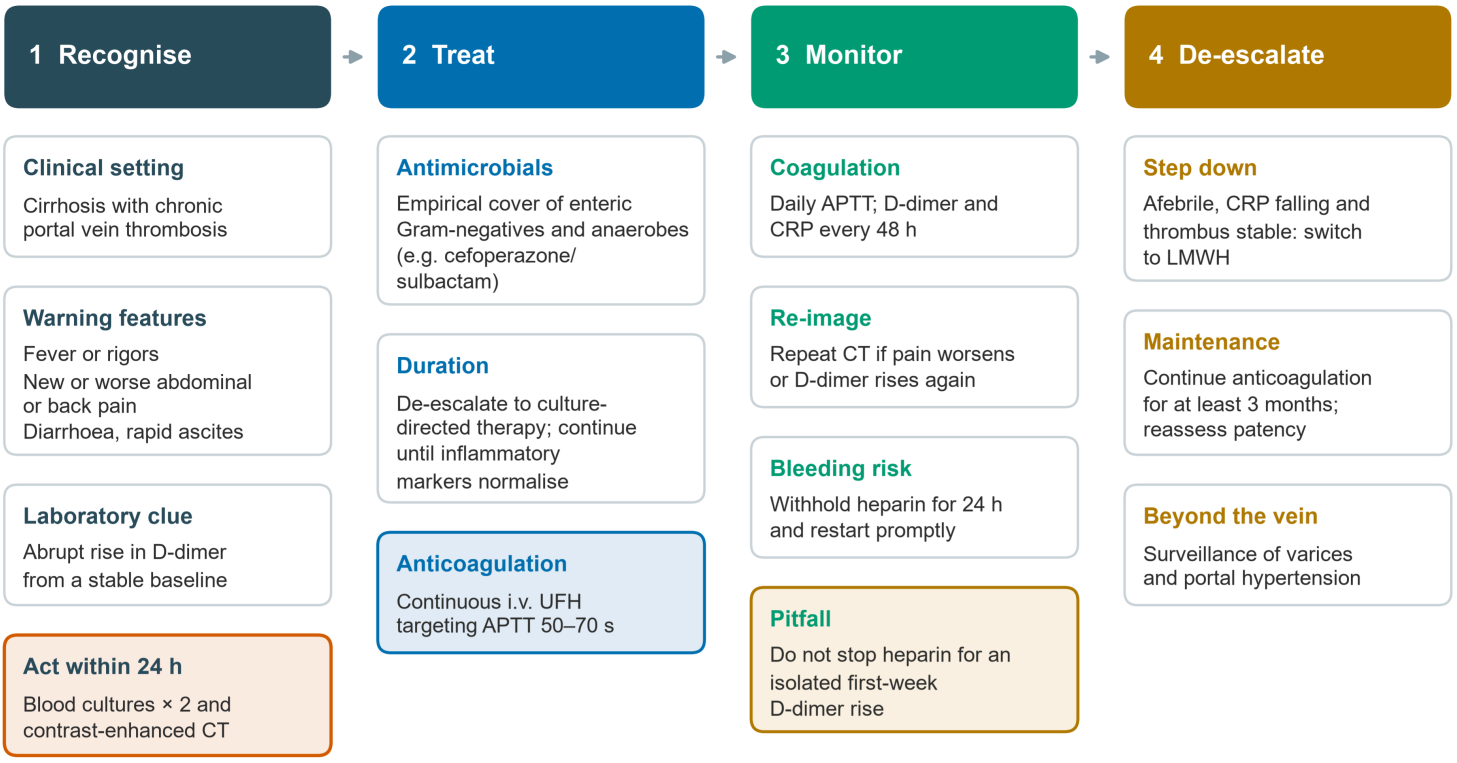


Figure 3

From chronic portal vein thrombosis to infective pylephlebitis: mechanism and warning triad. (A) Schematic (not to scale). A pyogenic focus in the ascending colon, drained by the ileocolic and mesenteric veins, seeds the portal circulation; the pre-existing organised thrombus acts as a nidus for suppurative thrombophlebitis, and sepsis-associated coagulopathy drives propagation into the collateral

vessels. (B) The clinical warning triad—high fever or rigors, new or worsening abdominal or back pain, and an abrupt rise in D-dimer—should prompt contrast-enhanced CT within 24 hours. NOVC, non-O1/non-O139 *Vibrio cholerae*; SMV, superior mesenteric vein.



Derived from a single case; proposed as a hypothesis-generating pathway, not a guideline.

APTT, activated partial thromboplastin time; CRP, C-reactive protein; CT, computed tomography; LMWH, low-molecular-weight heparin; PVT, portal vein thrombosis; UFH, unfractionated heparin.

Figure 4

Proposed management pathway for suspected infective pylephlebitis superimposed on cirrhosis-associated chronic portal vein thrombosis: recognition, antimicrobial and anticoagulant treatment, monitoring, and de-escalation to long-term low-molecular-weight heparin. The pathway is derived from a single case and is hypothesis-generating rather than a guideline. APTT, activated partial thromboplastin time; CRP, C-reactive protein; CT, computed tomography; LMWH, low-molecular-weight heparin; PVT, portal vein thrombosis; UFH, unfractionated heparin.