

# Successful treatment of acute portal vein thrombosis with rivaroxaban

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Splanchnic vein thrombosis (SVT) is a potentially life-threatening disease (1) and anticoagulant therapy with low-molecular-weight heparin or vitamin K antagonists is recommended (2). However, these agents lack evidence from randomised prospective trials, are not specifically approved and have significant limitations (2, 3). Modern oral anticoagulants such as rivaroxaban may offer pharmacologic and logistic benefits (3, 4).

Our *T&H Image* demonstrates the successful treatment of a 56-year-old male patient with acute symptomatic portal vein thrombosis, using the direct oral anticoagulant rivaroxaban. The patient, suffering from hemochromatosis for more than 20 years, complained of acute epigastric pain, dysphagia and fatigue for three weeks. Abdominal duplex ultrasound confirmed extensive acute portal vein thrombosis involving the main branch, ramus principalis dexter et sinister and most segmental branches. The clot could clearly be seen in b-mode ultrasound (► Figure 1a). Furthermore, colour-coded duplex demonstrates incomplete occlusion of the portal vein with partial perfusion between clot and vessel wall (► Figure 1b). Based on clinical and pharmacological considerations, we obtained written informed consent and decided to treat the patient's portal vein thrombosis using rivaroxaban at a dosage of 20 mg once daily.

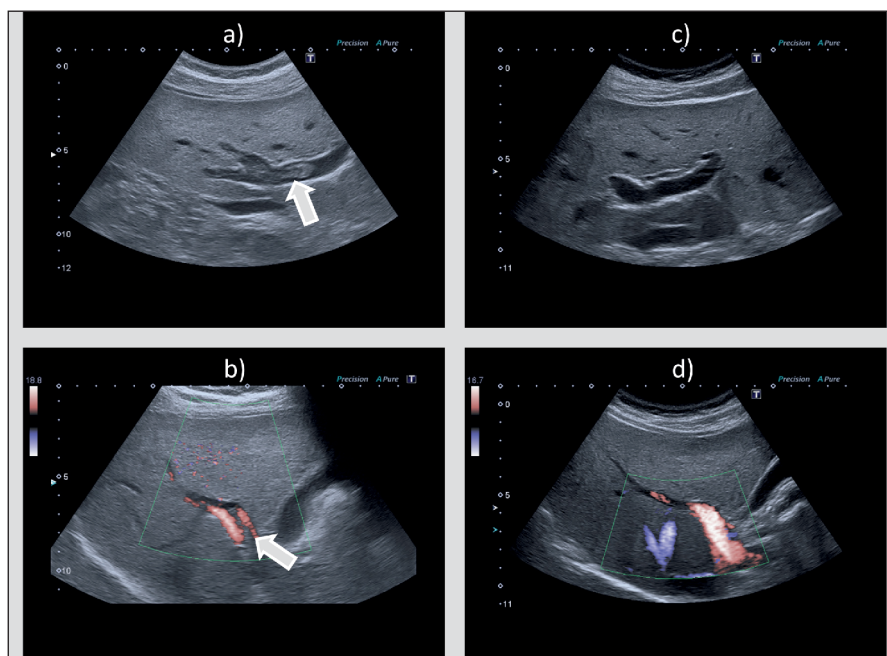
In a scheduled ultrasound control after four weeks, complete recanalisation of the

portal vein thrombosis including recanalisation of all segmental branches was found (► Figure 1c), and colour-coded duplex ultrasound demonstrated complete normalisation of blood flow (► Figure 1d). The patient tolerated rivaroxaban well and did not suffer any complications. After four weeks of treatment, the patient was completely asymptomatic.

Rivaroxaban is a direct factor Xa inhibitor, which has recently become approved for the treatment of venous thromboembolism. It has a reliable dose-response-relationship and a favourable safety profile (4). Rivaroxaban is rapidly absorbed in the upper gastrointestinal tract in its active form. Consequently, high plasma concentrations should be achieved in the portal vein at the site of acute SVT. Therefore, the pharmacological profile makes rivaroxaban a very interesting alternative for anticoagulant therapy of acute SVT.

Of note, rivaroxaban is a potent anticoagulant. As with every type of anticoagulation, the risk-benefit evaluation needs to include the assessments of bleeding risks, which is increased in SVT patients due to intestinal infarction, portal vein hypertension, thrombocytopenia or impaired coagulation parameters. Furthermore, drug-drug interactions are rare with rivaroxaban, but include some antibiotic and antimycotic agents, which needs to be considered especially in SVT patients with underlying malignant disease.

Rivaroxaban has been shown to be able to resolve clots rapidly (5), and our observation supports this concept. However, we need to point out that our report is only based on a single case and data from larger cohorts of SVT patients need to be collected to assess efficacy and safety of novel oral anticoagulants in this difficult-to-treat population.



**Figure 1: Liver ultrasound images (a and c = B-mode ultrasound, b and d = colour-coded duplex ultrasound). Left side: acute portal vein thrombosis in main stem (arrows) and segmental branches (a) and impaired blood flow (b). Right side: after four weeks of rivaroxaban treatment. Portal vein is completely recanalised without residual thrombi (c). Blood flow also is normalised (d).**

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Received: May 21, 2013

Accepted after minor revision: June 10, 2013

Prepublished online: July 11, 2013

doi:10.1160/TH13-05-0407

Thromb Haemost 2013; 110: 626–627

**Conflicts of interest**

None declared.

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