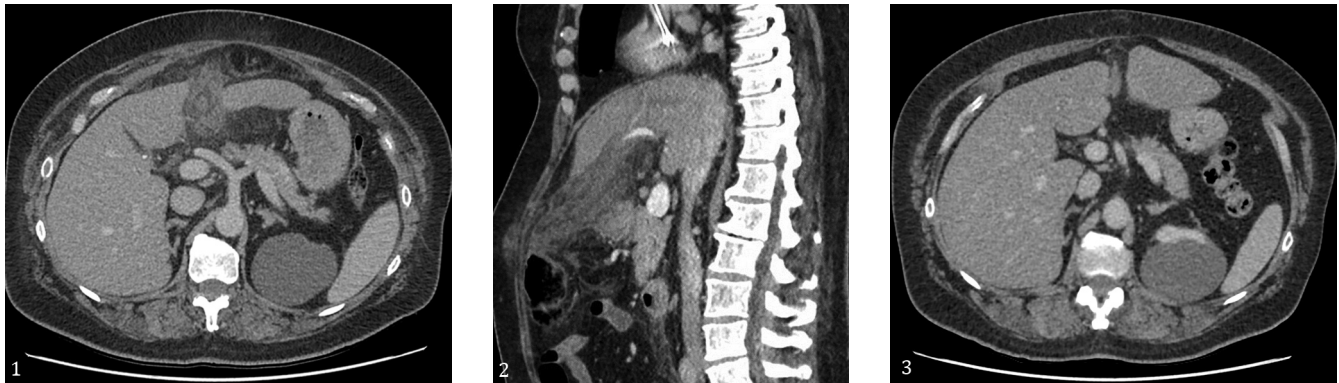


# Thrombophlebitis of the Umbilical Vein: A Rare Complication of Acute Pancreatitis

Megan M.L. Engels<sup>1,2</sup>, Aart Spilt<sup>3</sup>, Johan P. Kuijvenhoven<sup>1</sup>

1) Department of Gastroenterology and Hepatology, Spaarne Gasthuis, Hoofddorp; 2) Department of Gastroenterology and Hepatology, Leiden University Medical Center, Leiden; 3) Department of Radiology, Spaarne Gasthuis, Hoofddorp, The Netherlands



A 65-year-old woman presented with nausea, vomiting, and right upper quadrant pain radiating to her back. Past medical history included biliary pancreatitis with cholecystectomy three years prior, steatohepatitis, diabetes mellitus type II, and congestive cardiomyopathy on vitamin K antagonist therapy (INR 2.4). Inflammatory markers and liver enzymes were mildly elevated with markedly high lipase (2,100 IU/ml). Abdominal ultrasound showed no choledocholithiasis or ductal dilatation, and she was treated for suspected recurrent biliary pancreatitis with intravenous fluids and analgesics.

On day five abdominal pain worsened, accompanied by a fever of 38.5°C and rise in C-reactive protein (15mg/L to 410 mg/L). A computed tomography (CT) scan in the portal venous phase demonstrated an obliterated umbilical vein and surrounding fat infiltration, suggestive of proximal umbilical vein thrombophlebitis (Figs. 1 and 2). In a retrospect CT, the umbilical vein of our patient showed enhancement in portal venous phase five years earlier (Supplementary file).

To prevent thrombus expansion, anticoagulant therapy was switched to low molecular weight heparin. No antibiotics were administered in absence of infected necrosis; blood cultures remained negative. Symptoms improved and the patient was discharged one week later. Four months later, the umbilical vein was non-patent with near-complete resolution of surrounding fat infiltration (Fig. 3). Initial vitamine-K-antagonist therapy was resumed. One-year follow-up was unremarkable with no recurrent thrombosis or pancreatitis.

Acute pancreatitis induces local and systemic inflammation causing a thrombogenic state [1]. Portal vein thrombophlebitis is frequently caused by necrotizing pancreatitis [2], but thrombotic complications are less prevalent in cases without necrosis, and

rarely include extension in the umbilical vein. In literature, two causes for thrombophlebitis and recanalization in the umbilical vein have been proposed. One case suggested portal vein thrombosis induced portal hypertension, causing umbilical vein recanalization and thrombus expansion [3]. Another attributed recanalization and thrombus development to local inflammation, as portal vein thrombosis was absent in this case [4].

**Corresponding author:** Megan M.L. Engels, [m.m.l.engels@lumc.nl](mailto:m.m.l.engels@lumc.nl)

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## REFERENCES

1. Sissingh NJ, Groen JV, Koole D, et al; Dutch Pancreatitis Study Group. Therapeutic anticoagulation for splanchnic vein thrombosis in acute pancreatitis: A systematic review and meta-analysis. *Pancreatology* 2022;22:235–243. doi:[10.1016/j.pan.2021.12.008](https://doi.org/10.1016/j.pan.2021.12.008)
2. Choudhry AJ, Baghdadi YMK, Amr MA, Alzghari MJ, Jenkins DH, Zielinski MD. Pylephlebitis: a Review of 95 Cases. *J Gastrointest Surg* 2016;20:656–661. doi:[10.1007/s11605-015-2875-3](https://doi.org/10.1007/s11605-015-2875-3)
3. Foster RJ, Cowell GW. Acute paraumbilical vein recanalization: an unusual complication of acute pancreatitis. *BJR Case Rep* 2015;1:20150021. doi:[10.1259/bjrcr.20150021](https://doi.org/10.1259/bjrcr.20150021)
4. Lim HQC, Lee XWJ, Mathias N. A Rare Finding of Falciform Ligament Thrombosis as a Sequel of Acute Pancreatitis. *Case Rep Radiol* 2017;2017:2879568. doi:[10.1155/2017/2879568](https://doi.org/10.1155/2017/2879568)