Left Portal Vein Thrombosis Complicated by Acute Cholecystitis Cause Liver Atrophy: A Case Report

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Case report

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Abstract

Background: Portal vein thrombosis (PVT) was infrequently complicated by acute cholecystitis. The clinical signs of PVT are usually non-specific and subclinical. The treatment aim was to re-canalize the portal vein and to avoid serious complications. An early anticoagulation treatment would result in favorable outcome.

Case Report: We present a case of acute cholecystitis with sole left portal vein thrombosis causing left liver atrophy in a 59-year-old woman.

Conclusions: While the surgeon is familiar with this uncommon condition, the PVT could be detected on the pre-operative images through a tailored CT or MRI. The condition of PVT is often subclinical and might complicate liver atrophy, an early anticoagulation treatment would result in a favorable outcome.

Background

Portal vein thrombosis (PVT) resulting from acute cholecystitis is an infrequent condition(1). As so far, only a few cases have been reported about the rare condition of PVT post-acute cholecystitis(2, 3). Choi et al. speculated that the occurrence of PVT post-acute cholecystitis could be due to an inflammation or infectious process that involves the cystic vein(4). The condition of PVT is often subclinical and detected incidentally(1), and early anticoagulation treatment may result in more favorable outcome(5). Here, we present a case of acute calculous cholecystitis with left portal vein thrombosis and a rare consequence of left liver atrophy occurrence years later.

Case Report

A 59-year-old woman without systemic disease complained of relentless pain at the right upper abdomen for days. She visited our emergency room, where physical examination disclosed her pain was localized and aggravated with palpation. Laboratory blood test revealed no leukocytosis, unremarkable liver enzymes, and C-reactive protein elevation to 126 mg/L. An imbalance of protein-C and protein-S was not found. A further survey of the abdomen with intravenous contrast-enhanced computed tomography (CT) was performed. The acute cholecystitis was diagnosed based on findings of gallbladder distention with wall thickening. In addition, sole thrombosis of the left portal vein was noted (Fig. 1). On survey for the PVT, other associated lesions or coexistent conditions were excluded through the CT and testified by hours later MRI (Fig. 2). At the serial follow-up CT images (Fig. 3), the portal vein thrombosis progressed with a consequence of left liver lobe atrophy years later.

Discussion And Conclusions

The rate of incidental detection of PVT is around 1% of the general population(6). In some cases, PVT remains undiagnosed and is incidentally detected during routine examination for other conditions(1). The pathogenesis of PVT is multifactorial and common etiologies include malignancy of the hepato-

gastrointestinal organs, septic thrombophlebitis, liver cirrhosis, hyper-coagulation, and pancreatitis(1). There is no coexistent clinical condition responsible for PVT in our case which describes a rare condition of portal vein thrombosis secondary to acute cholecystitis. The association of PVT secondary to acute cholecystitis is not well known and is usually presented as case series and reports(2, 3). Choi e al. speculated that the acute cholecystitis related to an inflammation or infectious process involving the cystic vein may play a key of PVT(4). A connection between the cystic and portal veins provides a path for thrombus extension. Thus, we could observe PVT post-acute cholecystitis occurring in liver segments II, III, IV, V and VII(4).

The condition of PVT is often subclinical and detected incidentally(1). The clinical signs are usually non-specific, and patients would present with abdominal tenderness and distension, lower grade fever, and even shock(1). The favored treatment aim was not only at the primary condition of acute cholecystitis, but also the thrombotic portal vein to avoid serious complications. However, some studies noticed the PVT could spontaneously resolve (7).The treatment of PVT includes hydration, anticoagulation therapy, and (infrequently) thrombolytic therapy or surgical embolectomy(8). The time lag of starting the anticoagulation treatment dictates the outcome of recanalization (8). It has been reported that early anticoagulation treatment results in favorable outcome(5).

In our case, the patient's condition improved after a laparoscopic total cholecystectomy. As report, PVT could spontaneously resolve (7). Thereby, there was no aggressive interest focusing on the PVT management. Furthermore, the condition of PVT is often subclinical then further vigilance on the PVT might be lack. Therefore, a rare consequence of liver lobar atrophy would be complicated without an aggressive thrombolytic management as in this case.

Surgeons should be familiar with this uncommon condition, that PVT would be detected on the pre-operative images through a tailored image. The condition of PVT is often subclinical and might result in liver atrophy, and early anticoagulation treatment should be arranged.

List Of Abbreviations

PVT, CT, MRI

Declarations

Ethics approval and consent to participate:

All procedures performed in the study involving human participant were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. This case report has obtained IRB approval from "Chang Gung Medical Foundation Institutional Review Board" and the need for informed consent was waived

Consent for publication:

“Not applicable”. Any relevant data of the person was concealed and can’t be decoded from this article.

Availability of data and materials:

The data supporting the conclusion of this article are included within the article.

Competing interests:

The authors declare that they have no competing interests" in this section.

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C-H W drafted the manuscript. B-C L was the surgeon in participant. C-H W and Y-C W, B-C L collected and analyzed the clinical and image data. C-H W performed the critical revision for important intellectual content. C-H W and P.W W edited the manuscript. C-H W and Y-C W reviewed the draft. All authors read and approved the final manuscript.

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References

1. Muneer M, Abdelrahman H, El-Menyar A, Zarour A, Awad A, Al-Thani H. Acute Cholecystitis Complicated with Portal Vein Thrombosis: A Case Report and Literature Review. The American journal of case reports. 2015;16:627–30.

2. Hsu SC, Hsieh CH, Wu SC, Huang HC, Lo HC, Yeh CC. Successful staged treatment for acute cholecystitis complicated by portal vein thrombosis. Am Surg. 2012;78(1):E19–21.
3. El-Wahsh M. A case of portal vein thrombosis associated with acute cholecystitis/pancreatitis or coincidence. Hepatobiliary & pancreatic diseases international: HBPD INT. 2006;5(2):308 – 10.
4. Choi SH, Lee JM, Lee KH, Kim SH, Kim YJ, An SK, et al. Relationship between various patterns of transient increased hepatic attenuation on CT and portal vein thrombosis related to acute cholecystitis. AJR American journal of roentgenology. 2004;183(2):437–42.
5. Turnes J, García-Pagán JC, González M, Aracil C, Calleja JL, Ripoll C, et al. Portal hypertension-related complications after acute portal vein thrombosis: impact of early anticoagulation. Clinical gastroenterology hepatology: the official clinical practice journal of the American Gastroenterological Association. 2008;6(12):1412–7.
6. Ogren M, Bergqvist D, Björck M, Acosta S, Eriksson H, Sternby NH. Portal vein thrombosis: prevalence, patient characteristics and lifetime risk: a population study based on 23,796 consecutive autopsies. World journal of gastroenterology. 2006;12(13):2115–9.
7. Condat B, Pessione F, Helene Denninger M, Hillaire S, Valla D. Recent portal or mesenteric venous thrombosis: increased recognition and frequent recanalization on anticoagulant therapy. Hepatology. 2000;32(3):466–70.
8. Thomas RM, Ahmad SA. Management of acute post-operative portal venous thrombosis. Journal of gastrointestinal surgery: official journal of the Society for Surgery of the Alimentary Tract. 2010;14(3):570–7.

Figures

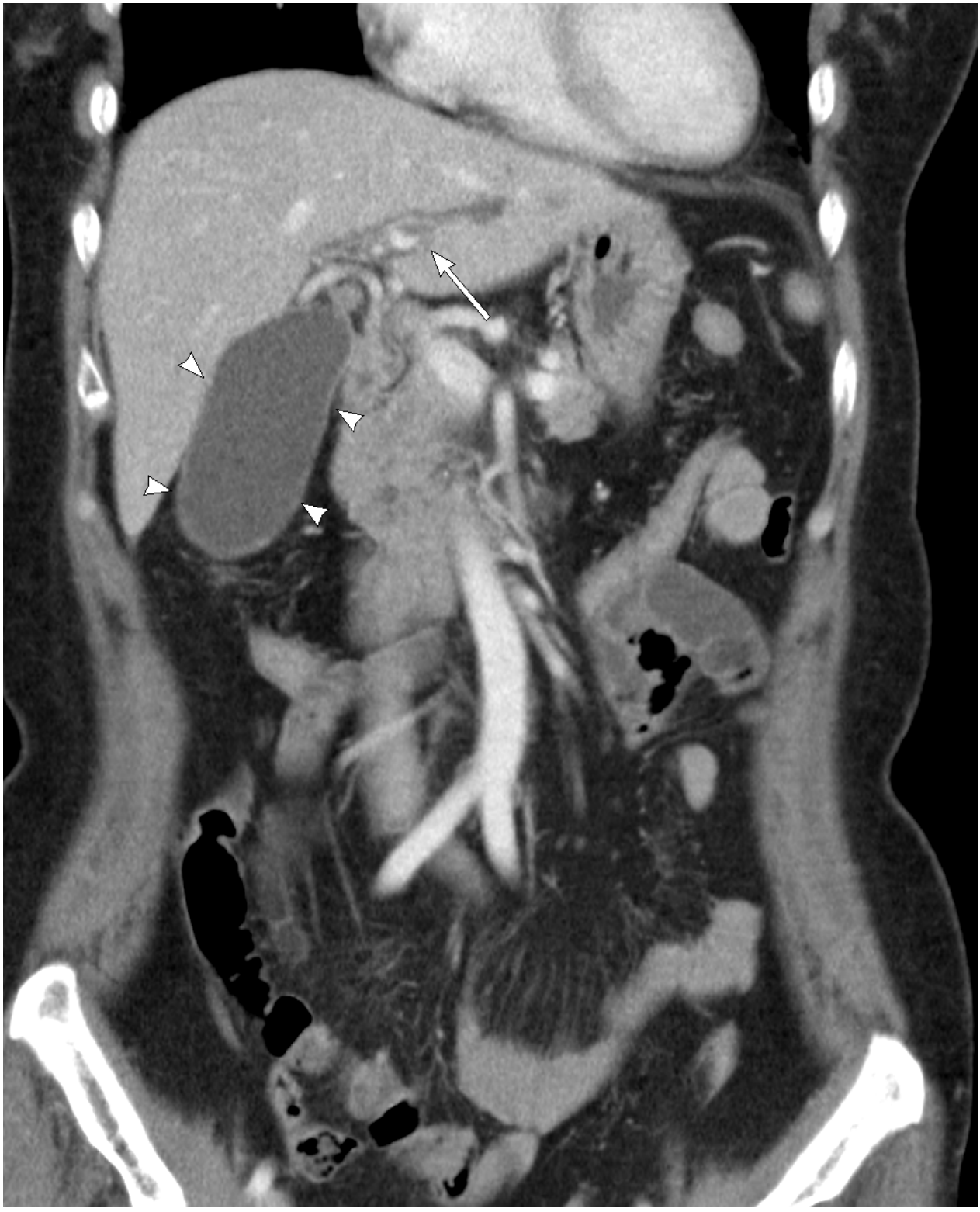

Figure 1

Corona view of the computed-tomographic image (CT) shows the gallbladder distention with wall thickening (arrowheads) and sole thrombosis of the left portal vein (arrow).

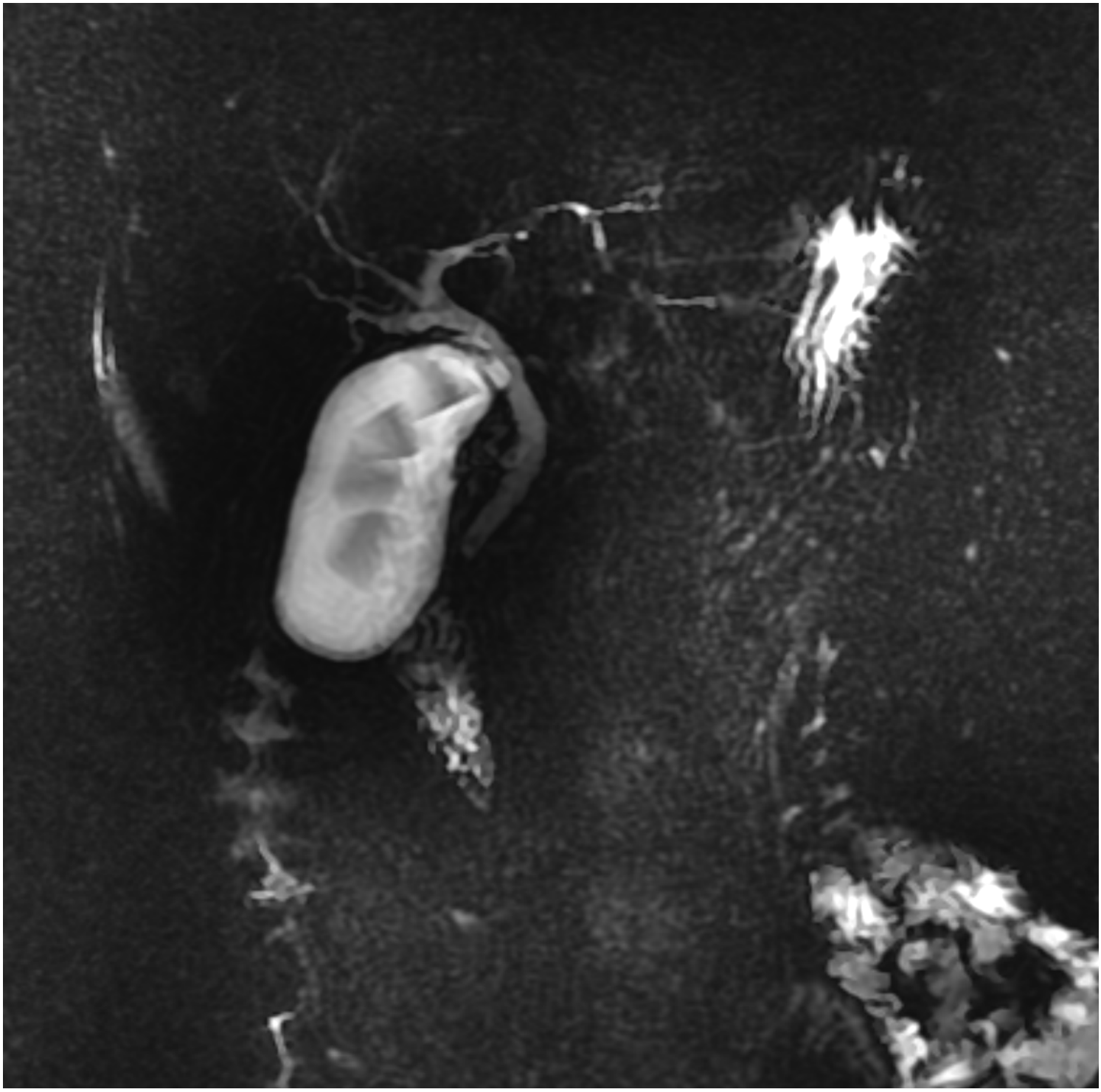

Figure 2

Magnetic Resonance Cholangiopancreatography unveils stones within the distended gallbladder and normal looking of the bile ducts.

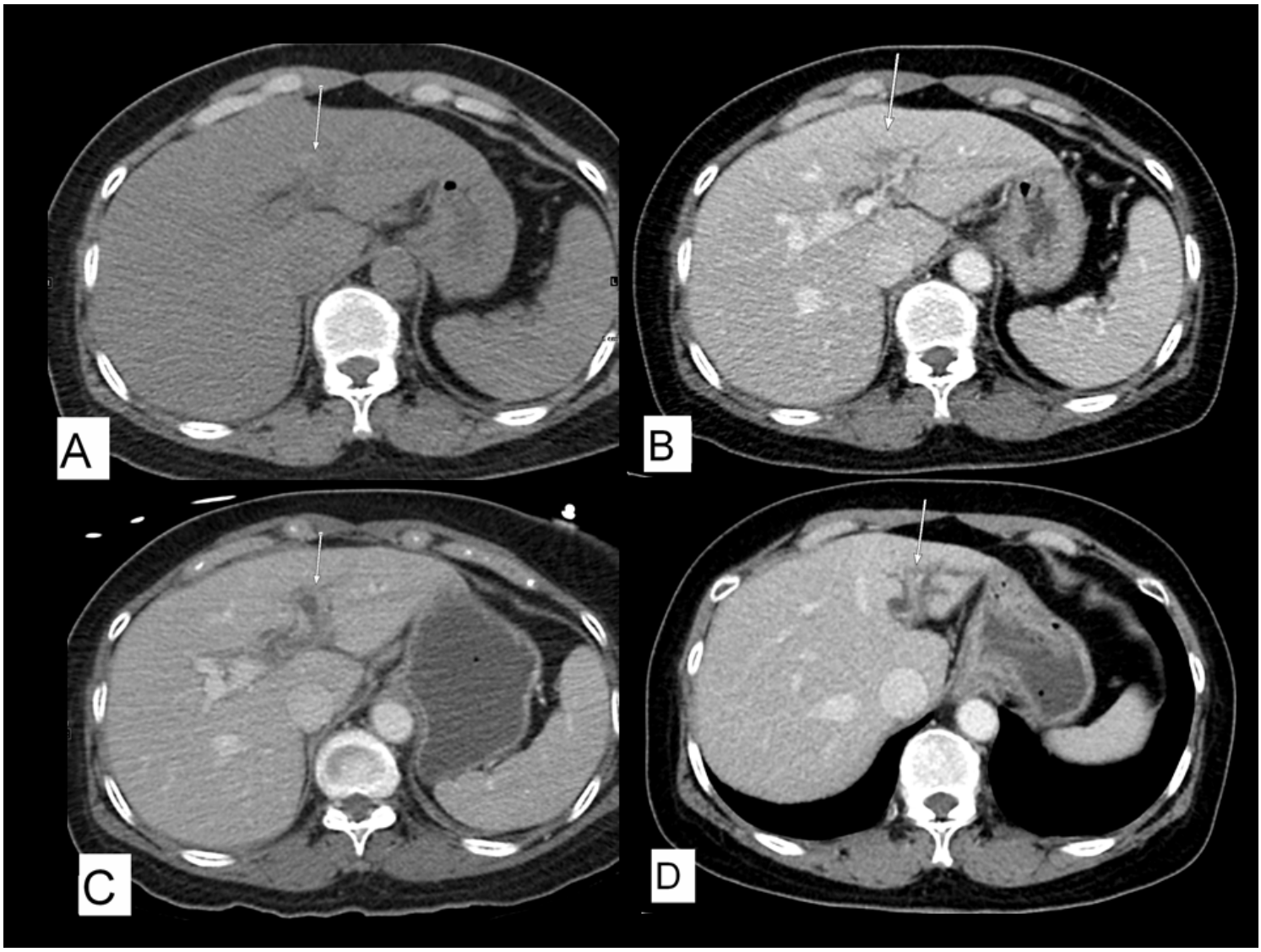

Figure 3

Pre-operative CT in un-enhanced phase reveals the CT density of the left portal vein increasing (A), and post-enhanced phase discloses portal vein thrombosis (B). Interval progression of the portal vein thrombosis is seen on CT ten days later (C). A consequence of left lobe atrophy is revealed on CT four years later (D).