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Case Report

Accessory Right Hepatic Artery Pseudoaneurysm Resulting in Biliary Obstruction

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Keywords

Biliary intervention · Pseudoaneurysm · Stent graft · Cholangitis · Pancreatitis

Abstract

Introduction: Visceral pseudoaneurysms are prone to rupture and can cause mass effect on surrounding structures, with extrinsic compression on the biliary tree being a rare but challenging complication. ***Case Presentation:*** We report a case of a 48-year-old man with a history of alcohol excess who presented acutely unwell with jaundice. Imaging revealed a pseudoaneurysm of the accessory right hepatic artery extending into an adjacent pancreatic pseudocyst, leading to common bile duct compression. Successful management included pseudoaneurysm exclusion with a stent graft and concurrent alleviation of the biliary obstruction. ***Conclusion:*** Managing pancreatic pseudocysts with biliary compression becomes complicated when an accompanying pseudoaneurysm is present, elevating the associated risk. In this case, stent graft exclusion of the pseudoaneurysm was the chosen approach to preserve arterial flow with potential for long-term patency. Delayed pseudoaneurysm diagnosis underscores the importance of comprehensive assessment in complex presentations, such as jaundiced alcoholic patients, where the possibility of pancreatitis episodes necessitates evaluation of the visceral vasculature for pseudoaneurysms.

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Introduction

Both visceral pseudoaneurysms and pancreatic pseudocysts are known complications of pancreatitis – often secondary to alcohol excess. Pseudoaneurysms have an inherent risk of rupture which can be life-threatening and require timely treatment. The sequelae of mass effect from a pseudoaneurysm are rare, although extrinsic compression on the common bile duct has been described [1]. Surgical management was once seen to be the gold standard treatment; however, more recently, endovascular management is seen to be a safe alternative first-line treatment [2].

This case report presents a unique and previously unreported scenario involving a hepatic pseudoaneurysm that extended into an adjacent pancreatic pseudocyst, leading to biliary obstruction. The percutaneous management employed, which involved the use of a stent graft to exclude the pseudoaneurysm, as well as a temporary biliary stent for facilitating biliary drainage, represents an elegant intervention not documented in existing case reports. This singular case highlights the inherent challenges in navigating the complexities of such a clinical scenario. The CARE Checklist has been completed by the authors for this case report, attached as online supplementary material (for all online suppl. material, see https://doi.org/10.1159/000535039).

Case Report

A 48-year-old male presented to his local emergency department with a short history of jaundice, melaena, and a tense abdomen. There was no past medical, surgical, or drug history. He had a social history of alcohol excess and was a smoker.

On presentation, liver function tests were deranged with elevated bilirubin (246 umol/L) and ALP (413 U/L), as well as hypoalbuminaemia (23 g/L). The patient was not significantly coagulopathic (INR = 1.4), and his Child-Pugh score was calculated to be 10 (C). He was admitted under gastroenterology for management of a new presentation of decompensated liver disease.

An abdominal ultrasound demonstrated liver cirrhosis, splenomegaly, and a small volume of ascites. The common bile duct was obscured by overlying bowel gas; however, there was no intrahepatic biliary tree dilatation. At this point, the patient's jaundice was presumed to be attributed to decompensated liver disease.

Four days after admission, a computer tomography (CT) liver (triple phase) demonstrated a large pancreatic head pseudocyst (Fig. 1) – which seemed to have been obscured by bowel gas on the initial ultrasound examination. An adjacent accessory right hepatic pseudoaneurysm was missed by the reporting radiologist.

The pseudocyst measured 5.9 × 5 × 4 cm and was causing extrinsic compression on the common bile duct. This was confirmed on subsequent magnetic resonance cholangiopancreatography, with no intrinsic filling defect (Fig. 2, 3). The presumption was that the aetiology of both the pseudoaneurysm and pseudocyst was chronic alcoholic pancreatitis which was symptomatically silent. The initial CT abdomen/pelvis also showed a right lower lobe lung mass (Fig. 4) which was later diagnosed as a lung neuroendocrine tumour following EBUS biopsy.

The patient became septic and was treated for spontaneous bacterial peritonitis. A second CT was performed demonstrating enlargement of the accessory right hepatic artery pseudoaneurysm into the adjacent pseudocyst (now measuring 5.9 × 5.4 × 4.6 cm) (Fig. 5). Compression on the CBD was now causing intra- and extrahepatic biliary tree dilatation (Fig. 6). The patient was clinically cholangitic.

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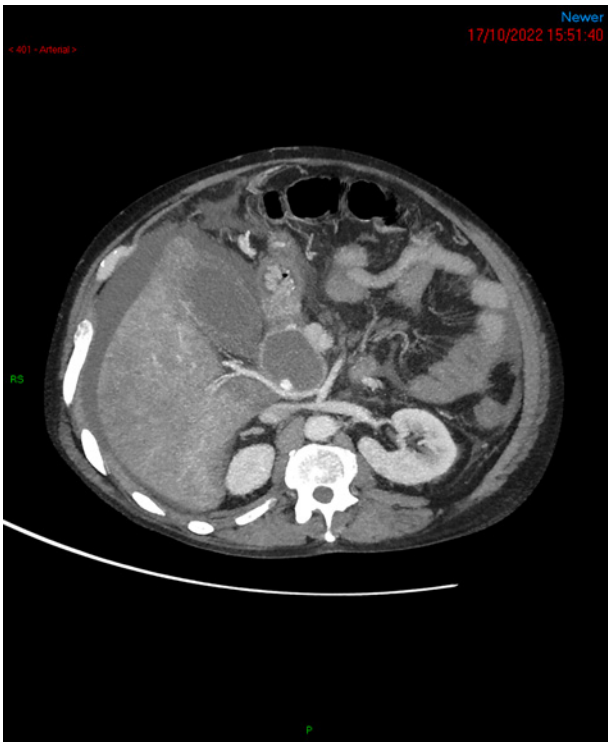

Fig. 1. CT abdomen demonstrating pseudoaneurysm of accessory right hepatic artery with surrounding pancreatic pseudocyst.

In an effort to obtain sepsis source control, as well as prevent life-threatening rupture of the aforementioned pseudoaneurysm, a decision was made to treat the pseudoaneurysm and perform biliary intervention under general anaesthesia.

Arterial access was established via the right common femoral artery. With a Merit C2 Cobra catheter, a coeliac axis angiogram was performed to demonstrate collateral flow to the right lobe of the liver if the accessory right hepatic artery were to be sacrificed.

An angled-tip 45-cm Terumo Destination catheter was used for support in accessing the superior mesenteric artery ostium. A superior mesenteric artery angiogram demonstrated the filling pseudoaneurysm (Fig. 7).

This was successfully crossed with a Terumo Glidewire and Cobra catheter then exchanged for a Rosen guide wire. A 5 mm × 29 mm Gore VBX balloon-mounted stent graft was deployed to exclude the pseudoaneurysm.

This was successful with no further filling of the pseudoaneurysm on subsequent angiogram (Fig. 8). There was minor vasospasm distally in the artery following deployment; however, satisfactory flow was demonstrated through the stent graft.

Immediately following this, a left-sided percutaneous transhepatic cholangiogram was performed. The CBD narrowing was crossed, with ease and an internal-external drain was inserted (Fig. 9).

The patient clinically improved following this with serum liver enzymes returning to normal and serum bilirubin fell precipitously. Due to high volumes through the biliary drain, there were concerns that it had been displaced, and this was confirmed on CT liver which demonstrated that there were side-holes within the peritoneal cavity and that ascites was inadvertently drained. The CT also showed that the VBX stent was patent and had successfully excluded the pseudoaneurysm (Fig. 10). This was demonstrated to still be patent 4 weeks following the procedure with resultant reduction in size of the pseudoaneurysm (Fig. 11).

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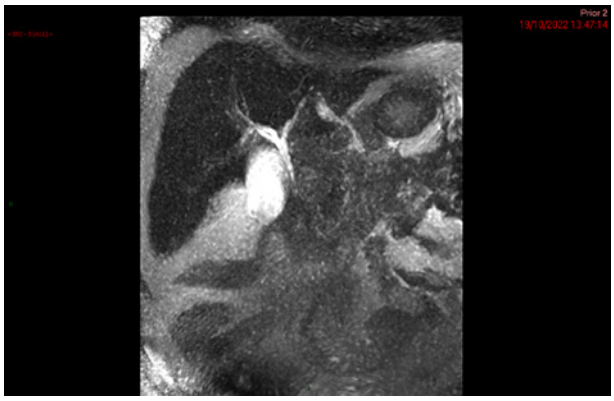

Fig. 2. Magnetic resonance cholangiopancreatography demonstrating extrinsic compression on the common bile duct.

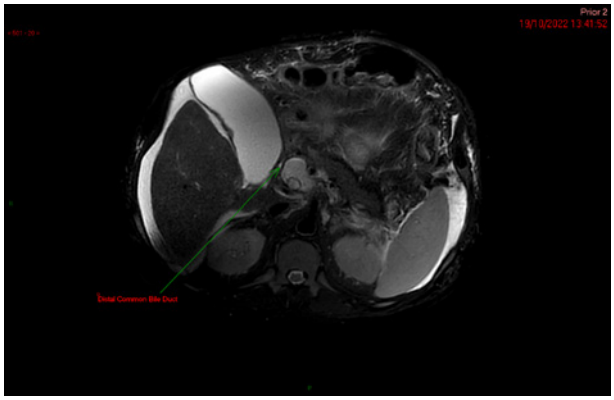

Fig. 3. Magnetic resonance cholangiopancreatography. T2w axial image demonstrating extrinsic compression on the common bile duct from the pancreatic head pseudocyst and associated right accessory hepatic pseudoaneurysm.

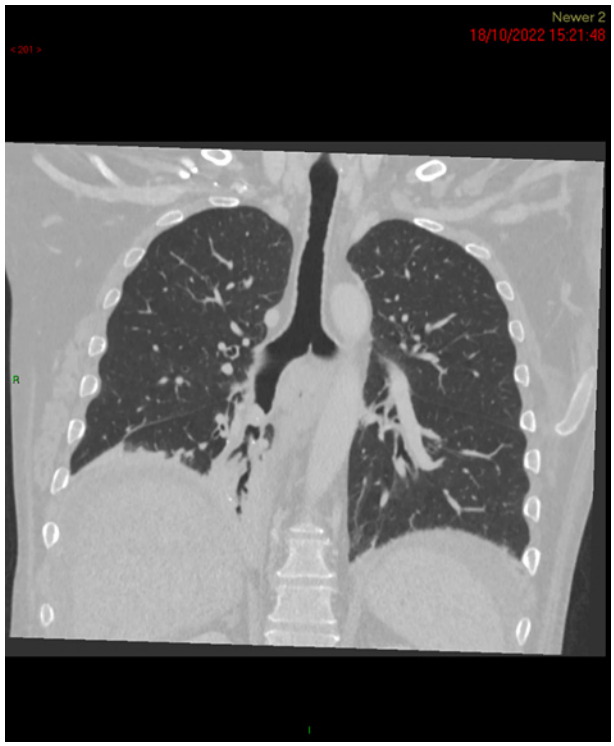

Fig. 4. CT thorax. Coronal reformat demonstrating an obstructing right lower endobronchial mass.

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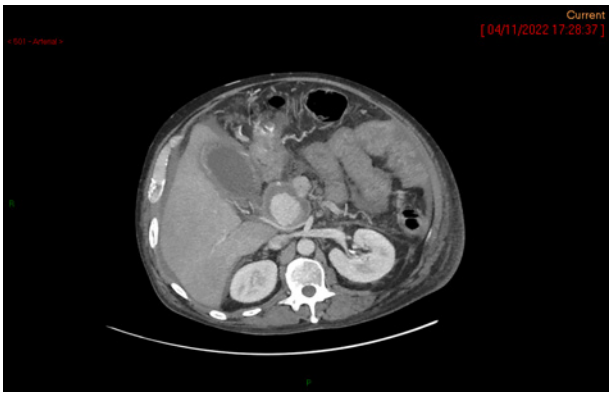

Fig. 5. Subsequent arterial phase CT demonstrating extension of accessory right hepatic artery pseudoaneurysm into the pancreatic pseudocyst.

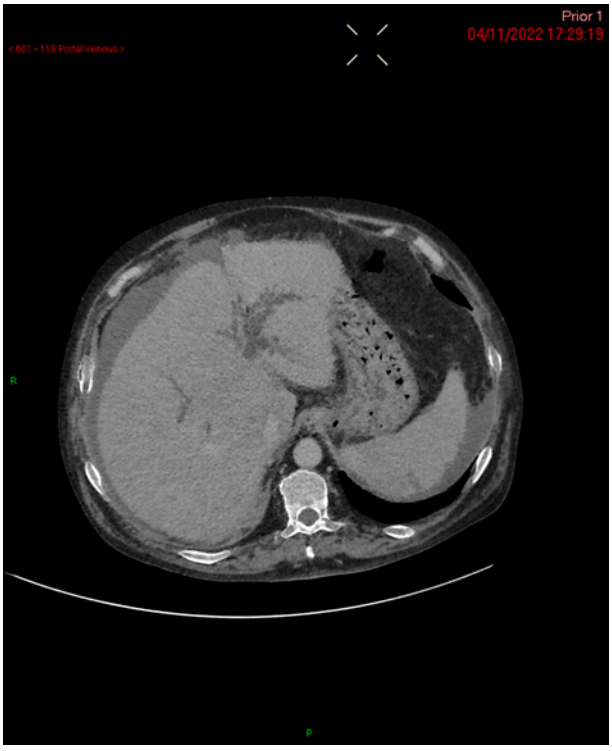

Fig. 6. Subsequent CT which demonstrating intrahepatic biliary dilatation.

The internal-external drain was removed under fluoroscopy, and a 7F plastic CBD stent was inserted (to be retrieved endoscopically in due course) (Fig. 12). A covering external biliary drain was inserted, initially capped, and removed a few days later.

Discussion

In cases involving pancreatic pseudocysts resulting in biliary compression, there exist several percutaneous, endoscopic, and surgical methods for management. However, this particular case introduces a complicating factor: the presence of an accompanying pseudoaneurysm. This factor significantly elevates the risk associated with any primary intervention for the pseudocyst. Therefore, it was necessary to address the pseudoaneurysm, which – as well as pressurising the pseudocyst – had an inherent risk of bleeding.

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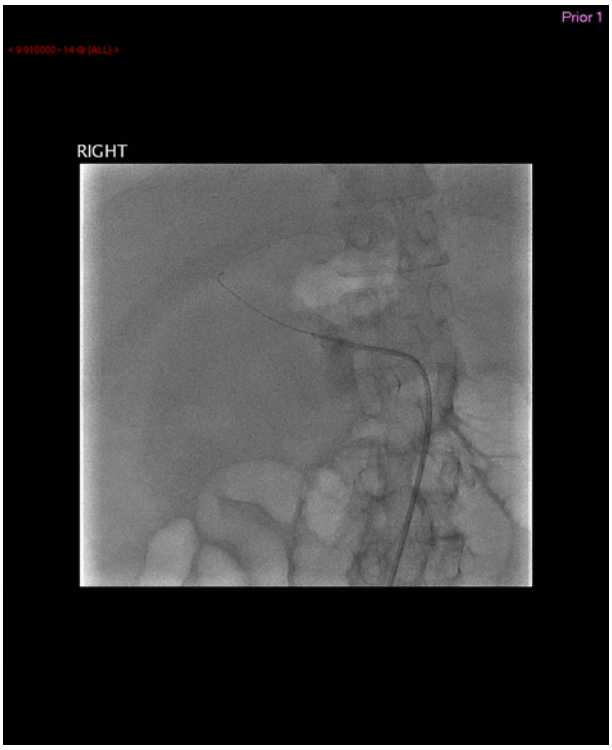

Fig. 7. Angiogram of right accessory hepatic artery with non-expanded stent in position. Contrast was injected through the sheath which demonstrated filling of the pseudoaneurysm sac.

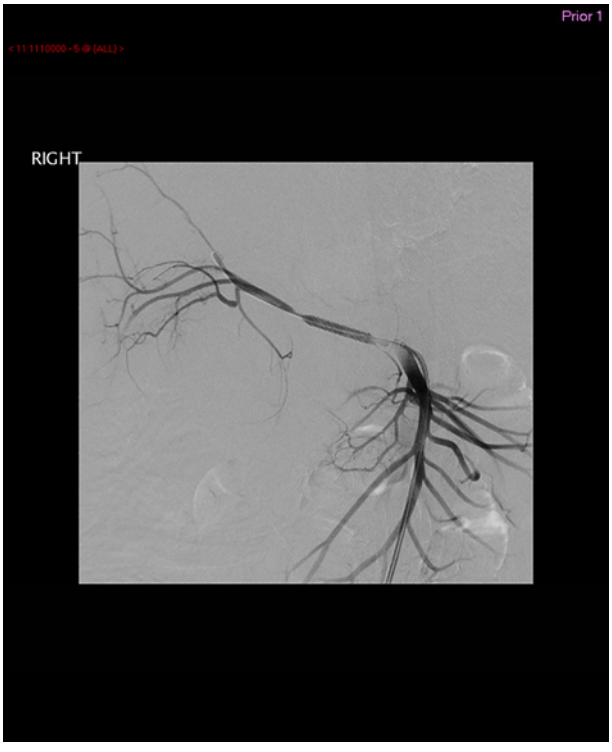

Fig. 8. Final digital subtraction angiogram following exclusion of the pseudoaneurysm with the stent graft. There is a degree of vasospasm distal to the stent graft.

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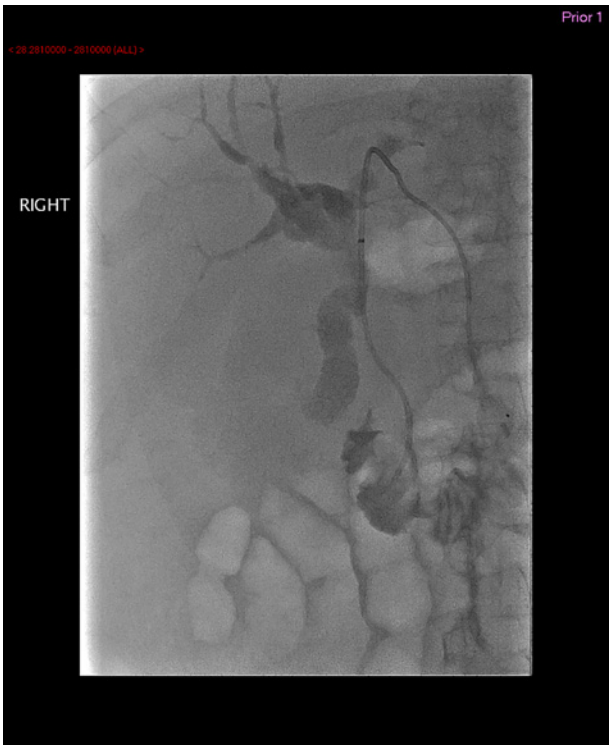

Fig. 9. Percutaneous transhepatic cholangiogram and insertion of an internal-external drain.

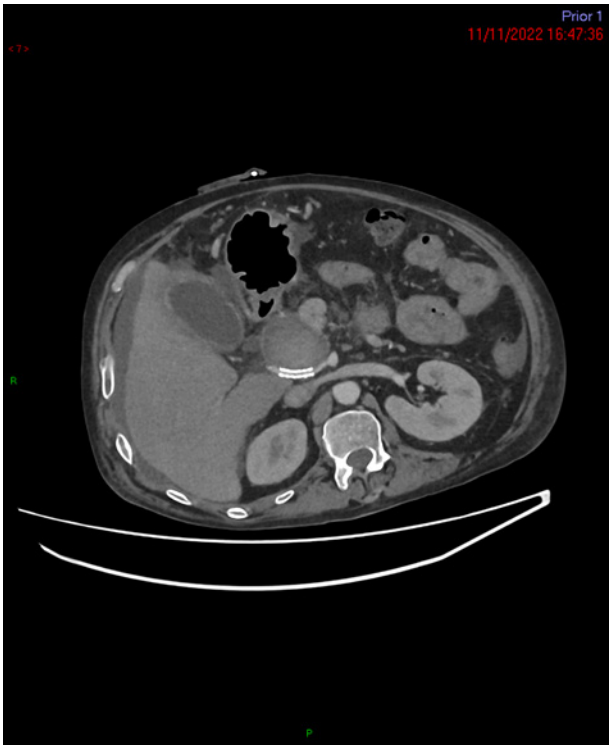

Fig. 10. Contrast-enhanced CT abdomen 5 days following the exclusion of the pseudoaneurysm.

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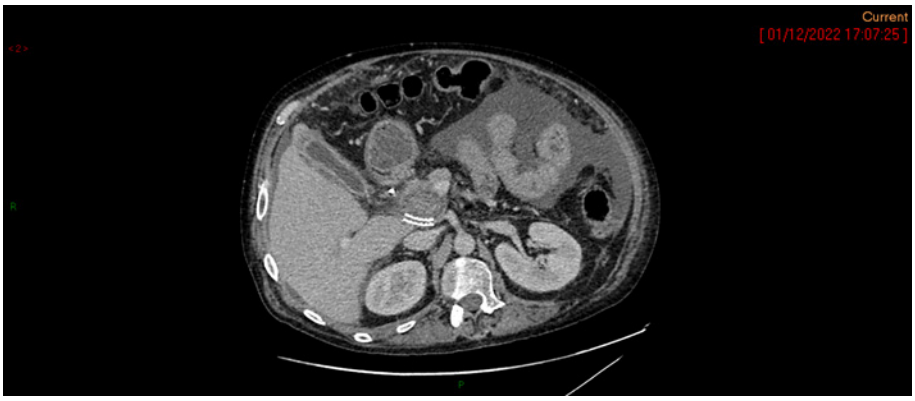

Fig. 11. Contrast-enhanced CT abdomen 4 weeks following the exclusion of the pseudoaneurysm. The stent graft remained patent, and there was an interval reduction in size of the pseudoaneurysm.

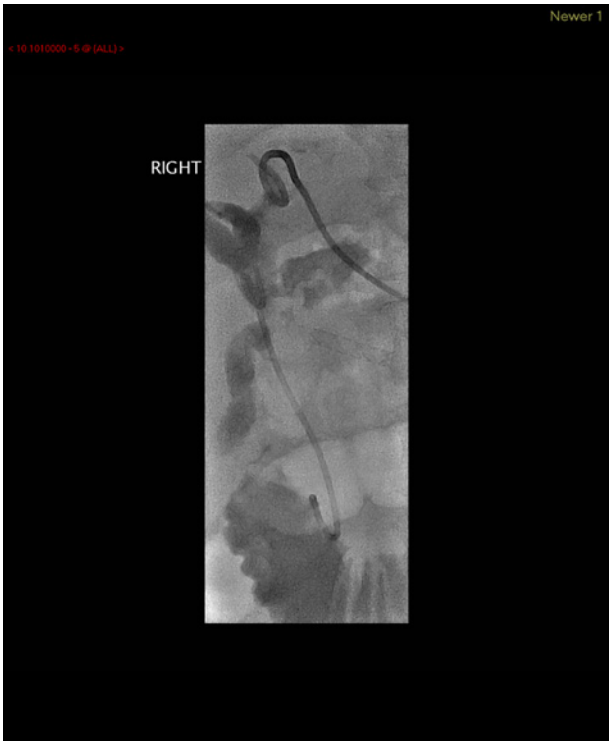

Fig. 12. Subsequent cholangiogram and internalisation with deployment of a retrievable common bile duct stent.

Pseudoaneurysms lack the three tunicae of true aneurysms and are more friable. They are the result of arterial wall disruptions (e.g., trauma, inflammation) and are at risk of rupture, resulting in haemorrhage with often lethal consequences.

Many visceral pseudoaneurysms can be treated with coil embolisation – directly into the pseudoaneurysm sac – or with "front-door/back-door" embolisation when the vessel can be sacrificed. In this instance, the pseudoaneurysm had recently expanded into the pseudocyst, and the sac was felt to be inherently friable, therefore liable to rupture if sac embolisation was to be attempted. It was important to attempt to preserve flow through the accessory right hepatic artery, especially in a patient with reduced hepatic reserve. As collateral perfusion from the coeliac axis was demonstrated, the option to sacrifice this was possible.

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Direct puncture and thrombin injection via a translumbar approach is also a potential treatment strategy but was deemed higher risk given the depth and the possibility the pseudocyst was infected.

Ultimately, exclusion of the pseudoaneurysm with a stent graft was chosen as the optimal treatment with the best chance of maintaining flow through the artery. There are data to suggest good mid- and long-term patency in these visceral arteries [3]. The main limitation is that these stent-grafts can occlude in time [4].

As an adjunct to clinical assessment of the jaundiced alcoholic patient, biochemical data and radiological examinations are usually sufficient in distinguishing between extrahepatic biliary obstruction and alcohol-induced liver disease. In this case of a new presentation of jaundice in a patient with previously undiagnosed alcoholic liver disease, it did not seem readily apparent whether his biochemical derangement was a composite of decompensated liver disease and extrahepatic biliary obstruction, or mainly secondary to the obstruction. As there was an abrupt improvement in the biochemistry following the treatment of the biliary obstruction, it is assumed that this was mainly an obstructive picture. ERCP with pseudocyst drainage was proposed; however, the patient was not fit for this initially. This was prior to the diagnosis of the adjacent pseudoaneurysm. The delay in diagnosis of the pseudoaneurysm may have delayed the definitive management of it, as well as led to the inappropriate referral for ERCP-assisted cyst drainage. This reporting discrepancy is an important learning point, as it is imperative in such patients – who may have had episodes of acute pancreatitis – to assess the visceral vasculature for pseudoaneurysms.

Statement of Ethics

The authors of this case report adhere to the principles of ethical conduct in medical research and publishing as outlined by the World Medical Association's Declaration of Helsinki. Informed consent was obtained from the patient, and all identifying information has been kept confidential. The authors declare no conflicts of interest.

In accordance with our local health board guidelines, ethical approval is not required as this case study is a retrospective review. Informed consent was obtained from the patient for the use of their information in this case report.

The patient has provided written informed consent for the publication of this case report, including the use of any associated data and images. All identifying information has been removed to protect the patient's privacy.

Conflict of Interest Statement

The authors of this case report declare that they have no conflicts of interest to disclose. This includes any financial, personal, or professional relationships that could influence or be perceived to influence the content of this report.

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Author Contributions

Colin William Primrose and Nikolas Arestis were involved in the conceptualisation and manuscript preparation of this case report. Colin William Primrose performed the initial analysis of the case, and Nikolas Arestis assisted with the interpretation of the data and final manuscript drafting. Both authors contributed to the final approval of the manuscript and agreed to be accountable for all aspects of the work.

Data Availability Statement

The data used in this case report are available upon request from the corresponding author. The data are owned by the hospital where the patient was treated, and the authors have obtained permission to use it for this case report. Any interested party may contact the corresponding author to request access to the data, subject to the approval of the Hospital's Data Access Committee.

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