



Contents lists available at ScienceDirect

International Journal of Surgery Case Reports

journal homepage: www.casereports.com

Common hepatic artery thrombosis after iatrogenic injury in pancreaticoduodenectomy operation, unexpected course

Ramy Hassan¹, Tameem Ibraheem, Ahmed Taha, Basher Fadel, Ahmed Zidan^{*,1}

Department of HPB and Liver Transplantation, Rajhy Liver Hospital, Assiut University, Assiut, Egypt

ARTICLE INFO

Article history:

Received 29 February 2016

Received in revised form 24 March 2016

Accepted 30 April 2016

Available online 6 May 2016

Keywords:

Hepatic artery thrombosis

Liver failure

Pancreatic resection

ABSTRACT

INTRODUCTION: Pancreatic resection is the only effective treatment with prolonged survival in operable pancreatic cancer and *peri*-ampullary cancer; it is also a procedure of significant morbidity and complications. Vascular injury is one of the most serious intraoperative complications.

PRESENTATION OF CASE: We report here a case of common hepatic artery injury during pancreaticoduodenectomy followed by complete thrombosis after its repair. As common hepatic artery is the only arterial blood supply of the liver, acute liver failure and necrosis were the usual course. Surprisingly liver enzymes and bilirubin start to improve in post operatively due to small hardly detected accessory left and middle hepatic artery.

DISCUSSION AND CONCLUSION: Although hepatic artery is the only arterial supply of the liver, occasionally small accessory arteries may give significant arterial blood supply. In such a situation liver enzymes act as surrogate markers to assess the sufficiency of this flow to the liver.

© 2016 Published by Elsevier Ltd on behalf of IJS Publishing Group Ltd. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

1. Introduction

While pancreatic resection is the only effective treatment with prolonged survival in operable pancreatic cancer and *peri*-ampullary cancer, it is also a procedure of significant morbidity and complications. The most commonly reported postoperative complications after pancreatic resection are pancreatic fistula, Delayed gastric emptying, intra-abdominal sepsis and postoperative hemorrhage. Although vascular injury and intra operative bleeding are the most serious intraoperative complications [1].

Many vessels are encountered during pancreatic resection in particular pancreaticoduodenectomy (PD), especially superior mesenteric and portal veins, superior mesenteric and hepatic arteries, and celiac trunk. Hepatic artery is usually the only arterial supply of the liver, any injury of common hepatic artery (CHA) carries the hazard of deprivation of the liver of arterial blood which may lead to acute liver failure, sepsis or liver abscess, and biliary complications in form of stricture or leak at site of anastomosis or bile duct vanishing syndrome in the most devastating scenario [2,3].

2. Case report

We describe a case of 70-year-old male, with eight weeks history of jaundice and repeated vomiting. No other relevant history was available. The significantly altered lab parameters showed a total serum bilirubin 5.7 mg/dl, direct of 4.7 mg/dl, INR 1.2, with normal ALT, AST, serum electrolyte and serum creatinine. An ultrasound of abdomen was done which showed mild hepatomegaly with dilated intra and extra hepatic biliary channels, no features of cirrhosis of liver or portal hypertension and identified a hypo-echoic lesion in the head of pancreas. Based on these data we ordered contrast enhanced computed tomography (CECT) scan for the abdomen, which revealed soft tissue mass arising from head of pancreas with 3 × 5 cm size with slight haziness of fat planes between it and stomach, superior mesenteric artery, portal and superior mesenteric vein are free from any invasion, with mildly dilated stomach with enlarged supra-pancreatic and *peri*-portal lymph nodes 2 × 3 cm with no distant metastasis. Patient was diagnosed as cancer head of pancreas with possibility of gastric wall invasion and decision was taken for surgical intervention and preoperative plan was for pancreaticoduodenectomy. He was evaluated by cardiac, pulmonary and anesthesia physician and was found fit for surgery. Intraoperative finding was similar to preoperative imaging and planning. During dissection of supra-pancreatic lymph nodes, common hepatic artery was accidentally injured and repair with Proline 8/0 using surgical lumps 3.5 × was done. Intraoperative Doppler on hepatic artery shows normal flow with RI 0.6 and PSV 100 cm/s pre anastomotic and 125 cm/s post anastomotic and SAT 71 ms. Pyloric

* Corresponding author at: Rajhy Liver Hospital, Assiut University, 71516 Assiut, Egypt.

E-mail address: ahmed.zidan@med.au.edu.eg (A. Zidan).

¹ Authors contributed equally to this work.

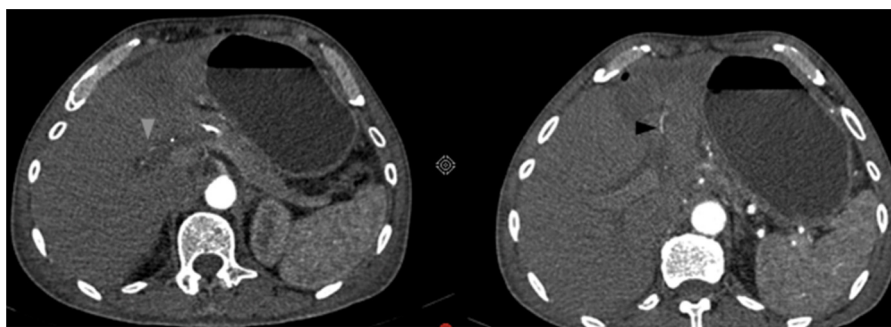


Fig. 1. CECT in POD 1 showing attenuated accessory left hepatic artery (black arrow) and middle hepatic artery (grey arrow).

preserving pancreaticoduodenectomy was done and patient admitted to Intensive care. Postoperative day (POD) one, patient was stable, but Doppler on hepatic artery revealed no detectable flow. Liver enzymes were highly elevated in POD1 with ALT 1278 U/L, AST 973 U/L. (normal range; ALT <41 U/L, AST <38 U/L) CECT was done and revealed completely thrombosed common hepatic artery with no detected contrast beyond site of repair. Interestingly intra-hepatic small accessory left hepatic artery with hardly detectable middle hepatic artery was found which followed to be arised from Left gastric artery (Fig. 1).

Decision was taken to follow the patient for few days to detect the trend of laboratory findings. Surprisingly ALT, AST, and bilirubin start to decrease significantly from POD2 with gradual improvement of all liver function parameters. Patient was shifted from ICU to ward on POD3; another Doppler on hepatic artery did not detect any flow in the hepatic artery after the site of repair. On POD 10, ALT was 137 U/L; AST 34 U/L and total bilirubin was 1.3 mg/dl (Fig. 2).

Patient was discharged. Follow up two weeks later in out patient clinic revealed normalization of liver function tests with non-detectable hepatic artery flow by liver Doppler and no relevant significant complaint. Follow up six months later revealed normal lab parameters, CECT scan was done again and revealed completely thrombosed hepatic artery with small accessory left hepatic artery with hardly detectable middle hepatic artery (Fig. 3).

3. Discussion

Hepatic artery thrombosis following PD in not a commonly encountered complication. Although thrombosis of common hepatic artery may pass without significant morbidity due to compensatory flow of arterial blood to the hepatic artery proper through gastroduodenal artery (GDA), but this is not the situation in PD as GDA is ligated as a step of the procedure, keeping the hazards any thrombosis of CHA will lead to complete deprivation of the liver from its arterial blood supply. The typical course of such

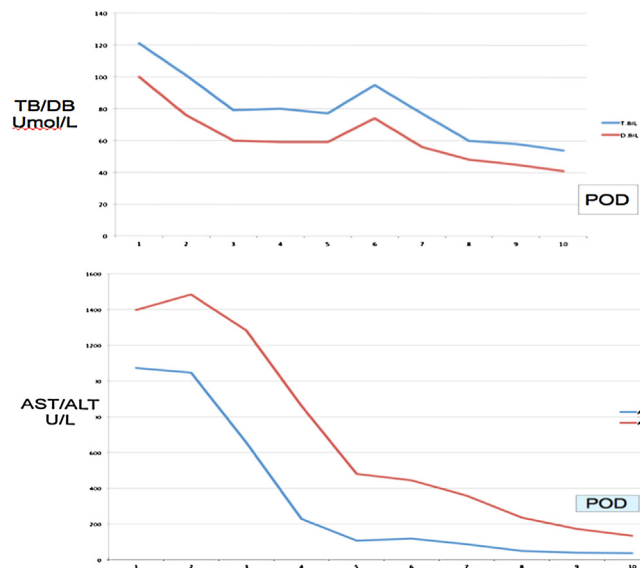


Fig. 2. Trends of decline of AST, ALT, direct and total bilirubin in postoperative period.

a situation is hepatic infarction followed by acute liver failure and septicemia [4].

Impairment of hepatic blood supply was termed as ischemic hepatitis (IH) which indicates inadequate hepatic perfusion, one of the possible causes of ischemic hepatitis is common hepatic artery thrombosis, It is characterized by significant rise in liver enzymes and bilirubin with undetectable Doppler signals on common hepatic artery, CECT is a confirmatory imaging modality is such a situation showing hypo perfusion of the liver, abrupt of the contrast at site of obstruction and hypo dense filling defect in the arterial lumen. Clinical presentations of IH ranges from devastating

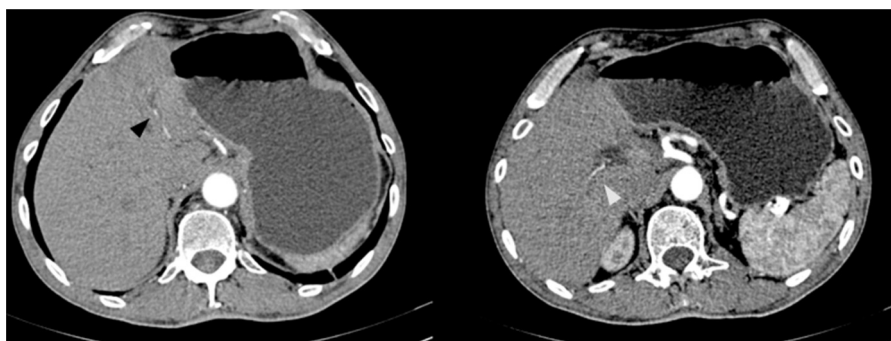


Fig. 3. CECT after 6 months showing accessory left hepatic artery (black arrow) and middle hepatic artery (white arrow).

course which is fulminant acute liver failure due to ischemic hepatic necrosis and bile duct vanishing syndrome to less catastrophic courses as biliary stricture or liver abscess. All these undesirable sequel encourage early intervention and trial to revascularize the obstructed CHA to avoid these morbidities [2,5,6].

Here in our case we noticed small intra hepatic arterial vessels in spite of absence of any contrast in hepatic artery in CECT scan with apparent thrombus at site of repair. A hardly detectable small accessory left hepatic artery with middle hepatic artery mostly arised from left gastric artery are noticed. Also faint hepatic contrast was noticed in the arterial phase in CECT, This encourage our team to take the decision of not to operate the patient again to re-vascularize the artery as we believed, these small vessels were sufficient to give enhancement to the liver so they may be sufficient to supply the liver itself. Surprisingly liver enzymes and liver functions improve significantly starting from POD 2 which supported our believe regarding sufficiency of these small vessels to give arterial blood supply to the liver, six months later follow up CECT scan showed a completely thrombosed CHA with no detected liver infarction, abscess or biliary complications.

4. Conclusion

Although hepatic artery is the only arterial supply of the liver, occasionally small accessory arteries may give significant arterial blood supply, which may compensate any blockage or thrombosis of hepatic artery, especially after ligation of GDA as in PD. In such a situation liver enzymes and liver enhancement in CECT scan act as surrogate markers to assess the sufficiency of this flow to the liver.

Conflict of interest

Ahmed Zidan and other co-authors have no conflict of interest.

Source of funding

There are no funding sources of any type.

Ethical approval

Local ethical committee in Rajhy liver hospital.

Consent

There is no patient data involved in this report.

Author contribution

Ramy Hassan and Ahmed Zidan were involved in writing manuscript. Ahmed Taha and Bashir Fadel were involved in patient management, and Tameem Ibraheem revise and approve manuscript for submission.

Guarantor

Ahmed Zidan is the Guarantor of this work.

References

- [1] C.K. Ho, J. Kleeff, H. Friess, M.W. Buchler, Complications of pancreatic surgery, *HPB (Oxford)* 7 (2) (2005) 99–108.
- [2] A.G. Tzakis, R.D. Gordon, B.W. Shaw Jr., S. Iwatsuki, T.E. Starzl, Clinical presentation of hepatic artery thrombosis after liver transplantation in the cyclosporine era, *Transplantation* 40 (6) (1985) 667–671.
- [3] D. Pandey, P.K. Garg, A. Jakhetiya, J. Sharma, S.H. Chandrashekhara, Markedly elevated liver transaminases following pancreaticoduodenectomy: celiac artery thrombosis in disguise, *Hepatobiliary Surg. Nutr.* 4 (5) (2015) 367–368.
- [4] M.A. Silva, P.S. Jambulingam, B.K. Gunson, D. Mayer, J.A. Buckels, D.F. Mirza, et al., Hepatic artery thrombosis following orthotopic liver transplantation: a 10-year experience from a single centre in the United Kingdom, *Liver Transpl.* 12 (1) (2006) 146–151.
- [5] B.J. Stange, M. Glanemann, N.C. Nuessler, U. Settmacher, T. Steinmuller, P. Neuhaus, Hepatic artery thrombosis after adult liver transplantation, *Liver Transpl.* 9 (6) (2003) 612–620.
- [6] M. Grodzicki, A. Anysz-Grodzicka, P. Remiszewski, B. Cieslak, M. Kotulski, P. Kalinowski, et al., Treatment of early hepatic artery thrombosis after liver transplantation, *Transplant. Proc.* 43 (8) (2011) 3039–3042.

Open Access

This article is published Open Access at [sciencedirect.com](https://www.sciencedirect.com). It is distributed under the [IJSCR Supplemental terms and conditions](#), which permits unrestricted non commercial use, distribution, and reproduction in any medium, provided the original authors and source are credited.