

World Journal of *Clinical Cases*

World J Clin Cases 2021 January 16; 9(2): 291-520



OPINION REVIEW

- 291 Continuity of cancer care in the era of COVID-19 pandemic: Role of social media in low- and middle-income countries
Yadav SK, Yadav N

REVIEW

- 296 Effect of a fever in viral infections – the ‘Goldilocks’ phenomenon?
Belon L, Skidmore P, Mehra R, Walter E
- 308 Overview of bile acid signaling in the cardiovascular system
Zhang R, Ma WQ, Fu MJ, Li J, Hu CH, Chen Y, Zhou MM, Gao ZJ, He YL

MINIREVIEWS

- 321 Gut microbiota and inflammatory bowel disease: The current status and perspectives
Zheng L, Wen XL

ORIGINAL ARTICLE**Retrospective Cohort Study**

- 334 Effective immune-inflammation index for ulcerative colitis and activity assessments
Zhang MH, Wang H, Wang HG, Wen X, Yang XZ

Retrospective Study

- 344 Risk factors associated with acute respiratory distress syndrome in COVID-19 patients outside Wuhan: A double-center retrospective cohort study of 197 cases in Hunan, China
Hu XS, Hu CH, Zhong P, Wen YJ, Chen XY

META-ANALYSIS

- 357 Limb length discrepancy after total knee arthroplasty: A systematic review and meta-analysis
Tripathy SK, Pradhan SS, Varghese P, Purudappa PP, Velagada S, Goyal T, Panda BB, Vanyambadi J

CASE REPORT

- 372 Lateral position intubation followed by endoscopic ultrasound-guided angiotherapy in acute esophageal variceal rupture: A case report
Wen TT, Liu ZL, Zeng M, Zhang Y, Cheng BL, Fang XM
- 379 Perioperative mortality of metastatic spinal disease with unknown primary: A case report and review of literature
Li XM, Jin LB

- 389** Massive gastric bleeding - perforation of pancreatic pseudocyst into the stomach: A case report and review of literature
Jin Z, Xiang YW, Liao QS, Yang XX, Wu HC, Tuo BG, Xie R
- 396** Natural history of inferior mesenteric arteriovenous malformation that led to ischemic colitis: A case report
Kimura Y, Hara T, Nagao R, Nakanishi T, Kawaguchi J, Tagami A, Ikeda T, Araki H, Tsurumi H
- 403** Coil embolization of arterioportal fistula complicated by gastrointestinal bleeding after Caesarian section: A case report
Stepanyan SA, Poghosyan T, Manukyan K, Hakobyan G, Hovhannisyanyan H, Safaryan H, Baghdasaryan E, Gemilyan M
- 410** Cholecystoduodenal fistula presenting with upper gastrointestinal bleeding: A case report
Park JM, Kang CD, Kim JH, Lee SH, Nam SJ, Park SC, Lee SJ, Lee S
- 416** Rare case of fecal impaction caused by a fecalith originating in a large colonic diverticulum: A case report
Tanabe H, Tanaka K, Goto M, Sato T, Sato K, Fujiya M, Okumura T
- 422** Intravitreal dexamethasone implant – a new treatment for idiopathic posterior scleritis: A case report
Zhao YJ, Zou YL, Lu Y, Tu MJ, You ZP
- 429** Inflammatory myofibroblastic tumor successfully treated with metformin: A case report and review of literature
Liang Y, Gao HX, Tian RC, Wang J, Shan YH, Zhang L, Xie CJ, Li JJ, Xu M, Gu S
- 436** Neonatal isovaleric acidemia in China: A case report and review of literature
Wu F, Fan SJ, Zhou XH
- 445** Malignant solitary fibrous tumor of the greater omentum: A case report and review of literature
Guo YC, Yao LY, Tian ZS, Shi B, Liu Y, Wang YY
- 457** Paratesticular liposarcoma: Two case reports
Zheng QG, Sun ZH, Chen JJ, Li JC, Huang XJ
- 463** Sinistral portal hypertension associated with pancreatic pseudocysts - ultrasonography findings: A case report
Chen BB, Mu PY, Lu JT, Wang G, Zhang R, Huang DD, Shen DH, Jiang TT
- 469** Epstein-Barr virus-associated monomorphic post-transplant lymphoproliferative disorder after pediatric kidney transplantation: A case report
Wang Z, Xu Y, Zhao J, Fu YX
- 476** Postoperative complications of concomitant fat embolism syndrome, pulmonary embolism and tympanic membrane perforation after tibiofibular fracture: A case report
Shao J, Kong DC, Zheng XH, Chen TN, Yang TY
- 482** Double-hit lymphoma (rearrangements of MYC, BCL-2) during pregnancy: A case report
Xie F, Zhang LH, Yue YQ, Gu LL, Wu F

- 489** Is sinusoidal obstructive syndrome a recurrent disease after liver transplantation? A case report
Liu Y, Sun LY, Zhu ZJ, Wei L, Qu W, Zeng ZG
- 496** Portal hypertension exacerbates intrahepatic portosystemic venous shunt and further induces refractory hepatic encephalopathy: A case report
Chang YH, Zhou XL, Jing D, Ni Z, Tang SH
- 502** Repair of a severe palm injury with anterolateral thigh and ilioinguinal flaps: A case report
Gong HY, Sun XG, Lu LJ, Liu PC, Yu X
- 509** Indirect inguinal hernia containing portosystemic shunt vessel: A case report
Yura M, Yo K, Hara A, Hayashi K, Tajima Y, Kaneko Y, Fujisaki H, Hirata A, Takano K, Hongo K, Yoneyama K, Nakagawa M
- 516** Recurrent inverted papilloma coexisted with skull base lymphoma: A case report
Hsu HJ, Huang CC, Chuang MT, Tien CH, Lee JS, Lee PH

ABOUT COVER

Editorial Board Member of *World Journal of Clinical Cases*, Dr. Mukul Vij is Senior Consultant Pathologist and Lab Director at Dr Rela Institute and Medical Center in Chennai, India (since 2018). Having received his MBBS degree from King George Medical College in 2004, Dr. Vij undertook postgraduate training at Sanjay Gandhi Postgraduate Institute of Medical Sciences, receiving his Master's degree in Pathology in 2008 and his PDCC certificate in Renal Pathology in 2009. After 2 years as senior resident, he became Assistant Professor in the Department of Pathology at Christian Medical College, Vellore (2011), moving on to Global Health City as Consultant Pathologist and then Head of the Pathology Department (2013). (L-Editor: Filipodia)

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Portal hypertension exacerbates intrahepatic portosystemic venous shunt and further induces refractory hepatic encephalopathy: A case report

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Abstract

BACKGROUND

Intrahepatic portosystemic venous shunt (IPSVS) is a rare hepatic disease with different clinical manifestations. Most IPSVS patients with mild shunts are asymptomatic, while the patients with severe shunts present complications such as hepatic encephalopathy. For patients with portal hypertension accompanied by intrahepatic shunt, portal hypertension may lead to hemodynamic changes that may result in exacerbated portal shunt and increased shunt flow.

CASE SUMMARY

A 57-year-old man, with the medical history of chronic hepatitis B and liver cirrhosis, was admitted to our hospital with abnormal behavior for 10 mo. He had received the esophageal varices ligation and entecavir therapy 1 year ago. Comparing with former examination results, the degree of esophageal varices was significantly reduced, while the right branch of the portal vein was significantly expanded and tortuous. Meanwhile, abdominal ultrasound presented the right posterior branch of portal vein connected with the retrohepatic inferior vena cava. The imaging findings indicated the diagnosis of IPSVS and hepatic encephalopathy. Instead of radiologic interventions or surgical therapies, this patient had only accepted symptomatic treatment. No recurrence of hepatic encephalopathy was observed during 1-year follow-up.

CONCLUSION

Hemodynamic changes may exacerbate intrahepatic portosystemic shunt. The intervention or surgery should be carefully applied to patients with severe portal hypertension due to the risk of hemorrhage.

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Core Tip: Intrahepatic portosystemic venous shunt (IPSVS) is a rare hepatic disease. Here we have reported a case that portal hypertension exacerbated IPSVS and resulted in hepatic encephalopathy. The decreased liver stiffness and the portal hypertension expanded IPSVS and significantly increased shunt flow. Then increased shunt ratio relieved portal hypertension, but it resulted in hyperammonia and eventually precipitated hepatic encephalopathy. This case highlights hemodynamic changes may exacerbate intrahepatic portosystemic shunt. Intervention or surgery should be carefully applied to patients with severe portal hypertension due to the risk of hemorrhage.

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INTRODUCTION

Intrahepatic portosystemic venous shunt (IPSVS) is a relatively rare hepatic disease and is more frequent in patients with liver-related diseases. IPSVS shows a wide variety of clinical manifestations, from asymptomatic to fatal complications. Previous research illustrates that intrahepatic shunts are commonly presented as multiple small shunts in patients with liver cirrhosis^[1], which can be asymptomatic. Herein, we have presented that portal hypertension exacerbated asymptomatic IPSVSs and induced refractory overt hepatic encephalopathy in a patient with chronic hepatitis B and cirrhosis.

CASE PRESENTATION

Chief complaints

A 57-year-old man was admitted to our hospital with abnormal behavior for 10 mo, accompanied with unconsciousness, dizziness, vomiting or headache.

History of present illness

The above symptoms started 10 mo previous and occurred once or twice per month. The abnormal behavior, vomit and unconsciousness recurred 1 d earlier and gradually improved in 7-8 h.

History of past illness

The patient had been diagnosed with chronic hepatitis B 3 years ago. He was admitted to our hospital for the first time with melena and was diagnosed with chronic hepatitis B, decompensated liver cirrhosis and esophageal varices 1 year ago. Our patient was assessed as Child-Pugh C, model for end-stage liver disease (MELD) score of 7.87 and MELD-Na score of 12.37. Then he started entecavir therapy and received the esophageal varices ligation. He had constipation for several months.

Personal and family history

There is not anything special in personal and family history.

Physical examination

Splenomegaly was noticed in physical examination, while no signs of jaundice,

anemia, bloating and abdominalgia were found.

Laboratory examinations

Laboratory examination indicated the increased level of blood ammonia (97 $\mu\text{mol/L}$ $1.35 \times$ upper limit of normal) and decreased level of platelets ($84 \times 10^9/\text{L}$ $0.84 \times$ lower limit of normal). Meanwhile, leukocytosis, hemoglobin, liver function, coagulation, alpha-fetoprotein and hepatitis B virus DNA quantification had normal results. The result of urobilinogen was positive, while other urine indices were negative. After 1-year of entecavir therapy, the Child-Pugh of our patient changed from C to B. The MELD score decreased from 7.87 to 7.85, and the MELD-Na score decreased from 12.37 to 9.64.

Imaging examinations

The gastroscope examination illustrated mild esophageal varices and negative red color sign (Figure 1A). Comparing with the former result, the degree of esophageal varices was significantly reduced (Figure 1B). Cranial magnetic resonance imaging indicated that there was no sign of new infarction or ischemia. Moreover, abdominal computed tomography (CT) showed cirrhosis, splenomegaly and portal hypertension with collateral circulation, while the right branch of the portal vein was expanded and tortuous (Figure 2A). The internal diameter of IPSVS was significantly enlarged from 0.3 cm to 1.3 cm when compared with previous CT results (Figure 2B). Meanwhile, abdominal ultrasound presented the right posterior branch of portal vein connected with the retrohepatic inferior vena cava (Figure 3).

FINAL DIAGNOSIS

These findings led us to the diagnosis of IPSVS and hepatic encephalopathy, which resulted in the abnormal behavior.

TREATMENT

Radiologic interventions and surgical therapies are conventional treatments of IPSVS. However, considering that the patient has a history of esophageal varices, shunt-occlusion therapy may increase the risk of gastrointestinal hemorrhage. Therefore, our patient did not receive radiologic interventions or surgical therapies and only accepted symptomatic treatment for hepatic encephalopathy. This patient was treated with a high fiber diet, branched chain amino acid, lactulose (10 mL tid oral) and rifaximin (0.2 g bid oral) combination therapy for 2 wk.

OUTCOME AND FOLLOW-UP

Hepatic encephalopathy did not occur after treatment, while the level of blood ammonia returned to normal and other laboratory indicators had no significant changes. No recurrence of hepatic encephalopathy was observed during 1-year follow-up.

DISCUSSION

IPSVS has various phenotypes and clinical manifestations. Previous research has categorized IPSVS into four types as follows^[2]: Type 1 corresponds to the right branch of the portal vein connecting with the inferior vena cava through a shunt and is commonly encountered in portal hypertension; types II and III include the shunts between the portal branch and the hepatic vein. According to the classification, the shunts between liver segments correspond to type II, while type III includes aneurysmal communication; and type IV corresponds to the unique or multiple tubular communications between the right portal branch and the inferior vena cava. As the most common type, this patient developed a type I intrahepatic shunt. Meanwhile, IPSVS can be either asymptomatic or accompanied with complications. The existence of IPSVS may aggravate impaired liver function and result in hepatic

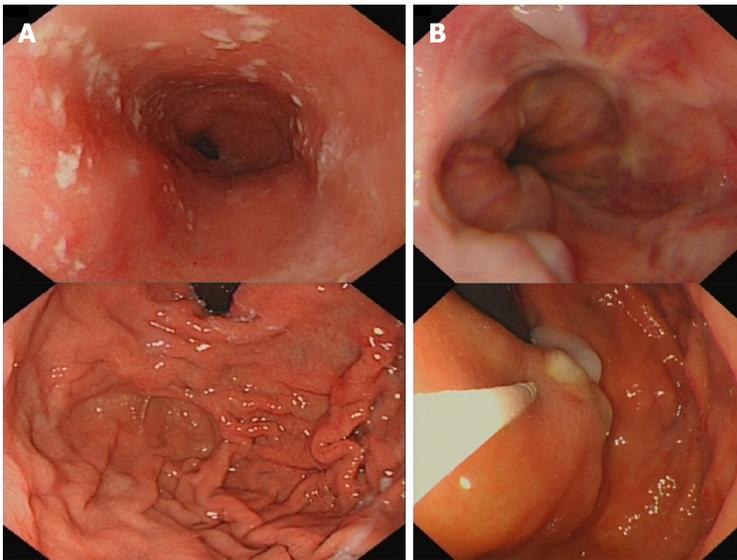


Figure 1 Gastroscopy showing the changes of esophageal varices. (A and B).

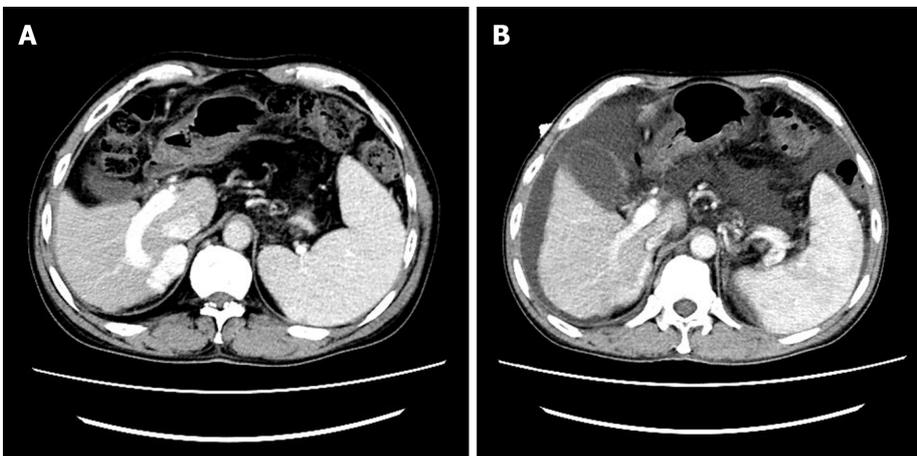


Figure 2 Appearance of intrahepatic portosystemic venous shunt in computed tomography-angiography. (A and B).

encephalopathy or hyperammonemia^[5]. A former study claimed that age and shunt ratio are risk factors of the complications. Hepatic encephalopathy is likely to occur in patients with a shunt ratio over 60%. Meanwhile, the shunt will likely remain asymptomatic in patients with a shunt ratio less than 30%^[4]. For older patients, an increased risk of encephalopathy is attributed to a decreased tolerance of the brain to toxic metabolites.

In consideration of asymptomatic IPSVS, clinical examinations for intrahepatic communications are not only important for the diagnosis, but also significant for the prevention and treatment. For patients with IPSVS, ultrasound and CT are invariably required for the diagnosis of intrahepatic shunt. Meanwhile, color doppler ultrasound is important to recognize asymptomatic IPSVS, which may be misdiagnosed as hypervascular lesions on CT or sonography^[4]. Moreover, the measurement of the shunt ratio by color doppler ultrasound is useful to determine the therapeutic options^[6]. Although the ultrasound and CT have increased the detection rate of intrahepatic shunt, IPSVS is still a relatively rare disorder. There are only 0.0235% adults who were found with spontaneous IPSVS by color doppler ultrasound^[6].

For IPSVS patients, an increased shunt ratio may induce complications. As for this patient, the brain had adapted to the small amount of toxic blood ammonia brought by the intrahepatic shunt at the beginning. IPSVS was asymptomatic before medical treatment. Then our patient received esophageal varices ligation and antiviral treatment. According to previous studies, entecavir antiviral therapy reduced liver stiffness in patients with chronic hepatitis B^[7,8]. Meanwhile, the esophageal varices ligation could only treat gastrointestinal hemorrhage but not relieve portal

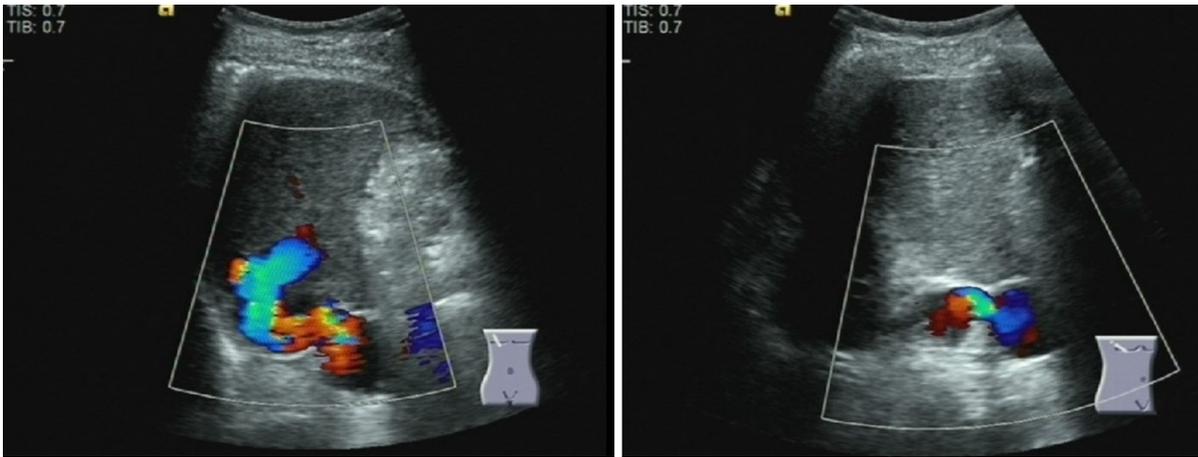


Figure 3 Color Doppler ultrasound showing the existence of intrahepatic portosystemic shunt.

hypertension. In consideration of the decreased liver stiffness, the portal hypertension expanded the shunt channel leading to the significant increased shunt ratio. Then increased intrahepatic shunt relieved the portal hypertension. However, the significantly increased shunt flow broke the brain's tolerance to toxic insult, leading to the elevated risk of complications such as hepatic encephalopathy. Moreover, constipation provided the toxic blood ammonia and eventually precipitated hepatic encephalopathy. Therefore, portal hypertension and reduction of liver stiffness were the reasons of aggravated IPSVS and hepatic encephalopathy.

It is believed that radiological or surgical therapies, such as occlusion, reverse symptoms and prevent long-term complications^[9]. Radiologic interventions are preferred therapies for IPSVS, and surgery is reserved for patients who are not suitable for radiologic intervention therapy or require liver transplantation. The endovascular treatment is recommended for patients presenting with symptomatic IPSVS^[10,11]. However, for patients with severe portal hypertension, occlusion may lead to an increased risk of esophageal varices and hemorrhage. Considering that our patient had portal hypertension and the history of esophageal varices hemorrhage, symptomatic treatment for hepatic encephalopathy^[12] was the optimal therapy for him.

CONCLUSION

In conclusion, hemodynamic changes may exacerbate intrahepatic portosystemic shunt and make it more complicated. Portal hypertension can increase shunt flow and break the brain's tolerance to toxic substances, leading to complications such as hepatic encephalopathy. For patients with liver cirrhosis and IPSVS, continuous monitoring of intrahepatic shunt is necessary for the prevention of hepatic encephalopathy and other complications. Surgical or radiological occlusions, while effective for IPSVS, may pose challenges to management of portal hypertension. The risk of portal hypertension and hemorrhage should be fully considered before occlusion, especially for patients with severe portal hypertension.

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