

## Case Report

# Colorectal tubular adenoma bleeding in patients with decompensated cirrhosis-a case report

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Received May 3, 2017; Accepted October 3, 2017; Epub November 15, 2017; Published November 30, 2017

**Abstract:** Background: Esophageal variceal bleeding (EVB) is one of the most common complications in patients with decompensated cirrhosis and there is rare of patients with decompensated cirrhosis complicated with intestinal bleeding of benign tumor, which is prone to misdiagnosis. Case presentation: A 65-year old male with a history of hepatitis B decompensated cirrhosis with EVB was admitted due to repeated haematemesis more than 1 years and blood in the stool for 4 hours. The patient was underwent endoscopic variceal ligation and was discharged with regular medication (propranolol 10 mg, 2 times/day; entecavir 0.5 mg, 1 time/day; esomeprazole 20 mg, 2 times/day), and 6 preventive treatments of esophageal variceal ligation in previous hospitalization. Emergency gastroscopy after hemostasis, fluid replacement treatment in our hospital showed that esophageal mild varicose veins without the red sign and thrombosis. Esophagus, stomach, duodenum showed no bloody fluid. Colonoscopy after saline enema showed, 45 cm from the anus, a large amount of yellow stool retention mixed with dark red bloody fluid. Sigmoid colon tumor (size 6×10 mm) was seen with surface blood scab formation, which was verified as tubular adenoma by using pathological biopsy. The tumor was treated by sigmoid colon tumor root ligation. Postoperative colonoscopy showed no active bleeding after ligation of sigmoid colon tumor and the HGB up to 110 g/L. Colonoscopy review showed that there was no residual tumor in sigmoid colon and no obvious abnormality in the terminal ileum and large intestine mucosa. Conclusion: In this case, through clinical observation and analysis, EVB was excluded by gastroscopy and lower gastrointestinal bleeding due to sigmoid colon tumor was diagnosed by colonoscopy. The patient achieved excellent therapeutic effect because of positive endoscopic treatment of suspicious lesions.

**Keywords:** Esophageal variceal bleeding, decompensated cirrhosis, sigmoid colon tumor, gastroscopy, colonoscopy

### Background

Esophageal variceal bleeding (EVB) is one of the most common complications in patients with decompensated cirrhosis. The common manifestations include hematemesis and melena or bloody stool. Hematemesis and melena often appear sequentially, but there are some patients who only have melena or bloody stools, and no hematemesis. However, there is rare of patients with decompensated cirrhosis complicated with intestinal bleeding of benign tumors, which is prone to misdiagnosis. In November 2016, our hospital diagnosed and treated 1 case of liver cirrhosis patient with colorectal tubular adenoma bleeding.

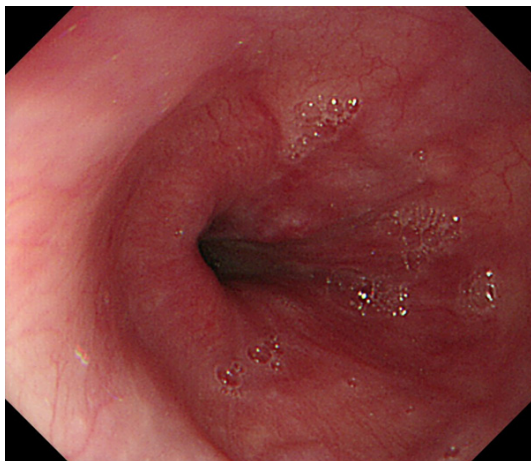
### Case presentation

A 65-year old male was admitted to our hospital in November third, 2016, due to repeated

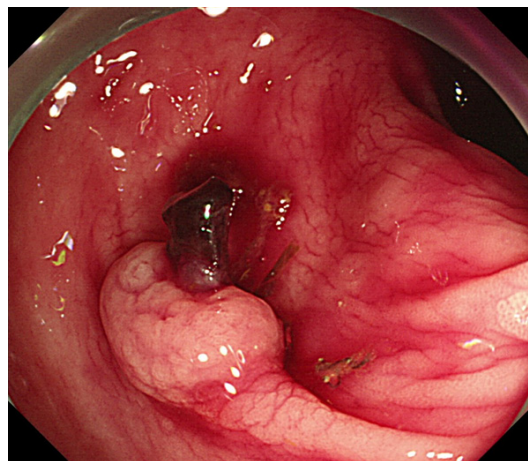
haematemesis more than 1 year and blood in the stool for 4 hours before admission. One years ago, he was hospitalized in our hospital diagnosed as hepatitis B decompensated cirrhosis with EVB, due to vomiting blood and discharging black pulpy stool.

The patient was underwent endoscopic variceal ligation in our hospital and postoperative recovery was good. The patient was discharged with regular medication (propranolol 10 mg, 2 times/day; entecavir 0.5 mg, 1 time/day; esomeprazole 20 mg, 2 times/day), and 6 preventive treatments of esophageal variceal ligation. The last time was March 1st, 2016. Three months ago, the patient was treated in our hospital due to cerebral infarction and improved after treatment. After oral administration of aspirin "100 mg QD" treatment, the patient was discharged with haemoglobin (HGB) examination 122 g/L.

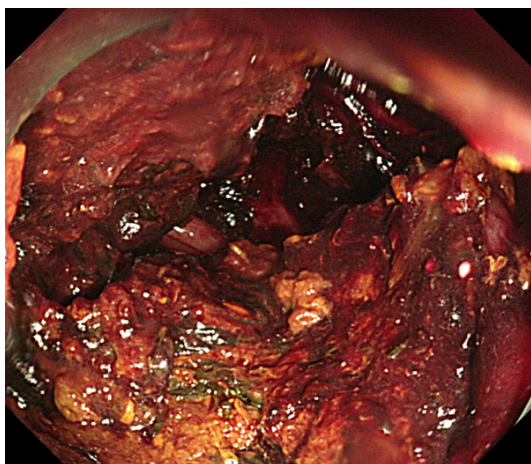
## Colorectal adenoma with decompensated cirrhosis



**Figure 1.** Emergency gastroscopy for diagnose EVB. The results showed that esophageal varicose veins without red signs and thrombosis.



**Figure 3.** The colonoscopy diagnosis for bleeding. The formation of the surface scab of the sigmoid colon tumor was seen (size 6×10 mm).



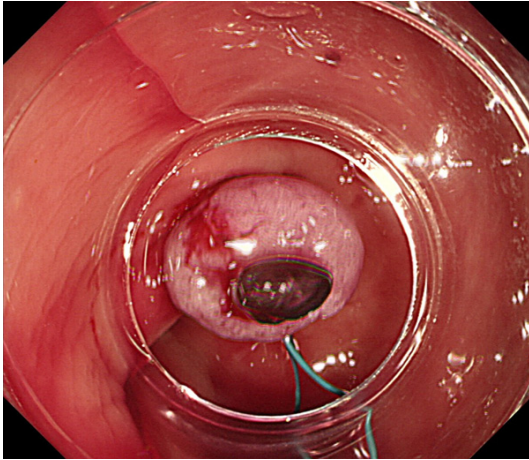
**Figure 2.** The colonoscopy diagnosis for bleeding. The results showed that a large amount of yellow stool mixed with dark red bloody fluid were found in the colon 45 cm from anus.

Four hours before admission, the patient began to discharge dark red blood stool, a total of 5 times about 800 ml, not mixed with the stool, no hematemesis, no abdominal pain, and no syncope. In another hospital, the patient showed no improvement by stopping taking aspirin after examination of HGB (72 g/L) and treating with esomeprazole (8 mg/h intravenous injection), octreotide (50 ug/h, 24 h intravenous injection with micro pump maintenance) and other drugs, and blood transfusion (O type Rh positive red suspension 4U). Therefore, the patient was transferred to our hospital to continue treatment.

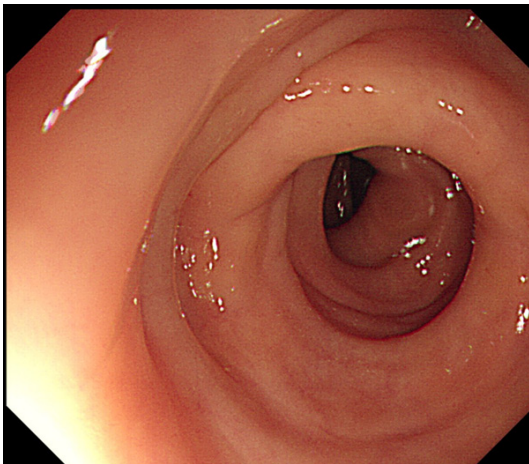
Physical examination after admission showed that temperature was 36°C; pulse was 115 times/min; breathing was 21 times/min; blood pressure was 95/62 mmHg; conjunctiva and lips were slightly pale; there were no yellow sclera and conjunctival edema; lungs was clear without dry and wet rales; heart rate was 115 beats/min; each heart valve auscultation area was no noise. The abdomen was soft without tenderness and rebound tenderness, and liver and spleen were not palpable under ribs. Bowel sounds was active with 12 times/min and much dark red bloody fluid was visible in anus. Finger examination of anus was negative and no edema was visible in both lower extremities.

Blood routine examination after admission showed that white blood cell count was  $5.26 \times 10^9/L$ ; HGB was 85 g/L; platelet count was  $112 \times 10^9/L$ . Prothrombin time (PT) were 11.4 seconds, the international normalized ratio (INR) was 0.96, activated partial thromboplastin (APTT) was 34.4 seconds and the total amount of fibrinogen was 3.42 g/L. After admission, the patient discharged 2 times of dark red blood stool about a total volume of 200 ml. Patients admitted to hospital was diagnosed as gastrointestinal bleeding.

Patients had a history of cirrhosis with EVB, but the patient discharged dark red stool without hematemesis performance. Esophagogastroduodenoscopy (EGD) is a reliable method for diagnosis of EVB within 12 to 24 hours of bleeding [1]. Emergency gastroscopy after hemosta-



**Figure 4.** The treatment of sigmoid colon tumor root ligation.



**Figure 5.** The colonoscopy review after discharging. The results of colonoscopy showed no residual tumor in the sigmoid colon.

sis, fluid replacement treatment showed esophageal mild varicose veins without the red sign and thrombosis. Esophagus, stomach, duodenum showed no bloody fluid (**Figure 1**), so the upper gastrointestinal bleeding was excluded. Colonoscopy after saline enemas showed, 45 cm from the anus, a large amount of yellow stool retention mixed with dark red bloody fluid (**Figure 2**). Descending colon, sigmoid colon and rectum were seen with small amount of dark red bloody fluid retention. Sigmoid colon tumor (size 6×10 mm) was seen with surface blood scab formation (**Figure 3**). After repeatedly washing the descending colon, sigmoid colon, rectum, there was no active bleeding.

The tumor was treated by sigmoid colon tumor root ligation treatment (**Figure 4**) and biopsy.

Postoperative daily monitoring of blood showed that HGB increased progressively (HGB 95 g/L after 3 days). Pulse was 85 beats/min and blood pressure was 135/70 mmHg. Colonoscopy after oral purgative bowel preparation showed that terminal ileum, ileocecal, ascending colon, transverse colon, descending colon and rectal mucosa showed no abnormality. Sigmoid colon tumor after root ligation showed no active bleeding. Pathological biopsy showed tubular adenoma (sigmoid colon). One week after admission, the HGB was recovered to 110 g/L. The occult blood of stool was negative. The patient was discharged. One month after discharge, colonoscopy review showed that there was no residual tumor in sigmoid colon (**Figure 5**), and there was no obvious abnormality in the terminal ileum and large intestine mucosa.

#### Discussion

Hepatitis B virus is the main cause of cirrhosis in China. Esophageal varices (EV) are a common complication of cirrhosis. The main performances are hematemesis and melena. Patients are at a high risk of bleeding and death after stop of the acute variceal bleeding. For patients without preventive treatment, the average recurrence rate in 1 to 2 years was 60% and the mortality rate was 33% [2]. So the prevention is very important including drug therapy, endoscopic therapy, surgery or radiation therapy [3-5]. Non selective beta receptor blockers (commonly used drugs propranolol) can reduce the risk of bleeding and improve the survival rate [6, 7]. Endoscopic variceal ligation (EVL) was used for the prevention of EV rebleeding [8, 9]. A number of clinical studies demonstrated that non selective beta receptor blockers combined with endoscopic therapy is the first choice for prevention of EVB [10, 11]. The patient was given propranolol combined with endoscopic variceal ligation (a total of 7 times) after initial esophageal varices. According to our clinical experience, the esophageal varices should be controlled. But it must be given clear diagnosis as soon as possible to determine whether it was EBV, because misdiagnosis might be life-threatening. With gastroscopy, we proved that our initial diagnosis at admission was not EVB.

Polyps of colon include adenoma, hyperplastic polyps and hamartoma [12]. Adenomas can be divided into tubular, tubular villous and villous adenoma. The tubular adenoma, with a tubular structure and the villi component less than 20%, is one of the most common gastrointestinal polyps composed of hyperplastic mucosa epithelium. Endoscopic manifestations show multiple or single nodular surface. Most are pedunculated and the size is generally not more than 2 cm. They are dark red and easy to bleed. Under a microscope, they are hyperplasia of glandular tissue. Glandular epithelium was arranged regularly and well differentiated, with atypia, much mitosis, but not invading the muscle membrane. In tubular adenoma, large volume and wide base tumors with nodular or lobulated surface and surface erosion and hemorrhage are easy for canceration. A report showed that [13], the canceration rate of adenomatous polyps with the diameter of 1~2 cm is about 10% and the diameter more than 2 cm is close to 50%. Therefore, the adenomatous polyps should be treated as soon as possible. Endoscopic high-frequency electroablation resection [14] is the preferred method of treatment of colon polyps, but has the higher incidence of hemorrhage and perforation to remove wide base and thick pedicle polyps [15]. Bleeding is the most common and most serious complication with the incidence rate is about 0.3%-6.1% [16, 17] and the perforation rate is about 0.085%-0.18% [18, 19]. Nylon rope ligation can reduce the risk of bleeding and perforation [20].

At present, low-dose aspirin (75~150 mg/D) is widely used in the treatment of coronary atherosclerotic heart disease (CHD), cerebrovascular disease and peripheral arterial disease [21]. Studies have shown that aspirin can increase the risk of gastrointestinal injury 2 to 4 times [22]. Meta analysis showed that the absolute risk of severe gastrointestinal bleeding caused by aspirin was 0.12% per year and was associated with doses [23]. Primary prevention meta-analysis showed that aspirin increased the incidence of gastrointestinal bleeding by 1.37 times [24]. The mechanism of gastrointestinal bleeding because of [25, 26]. 1) local effects: aspirin has a direct stimulating effect on the phospholipid layer on the gastric mucosa, destroying hydrophobic protective ba-

rier of gastric mucosa; the disintegration of aspirin in stomach induced a large amount of release of leukotrienes, damaging gastric intestinal mucosa and 2) systemic effects: aspirin can induce serine acetylation in the active site of cyclooxygenase (COX), inhibiting the activities of COX-1 and COX-2 in gastric mucosa, and decreasing the production of prostaglandin. PG regulates gastrointestinal blood flow and mucosal function and its reduction is the main cause of gastrointestinal mucosal injury caused by aspirin. In recent years, the incidence of lower gastrointestinal bleeding was significantly higher in patients receiving dual antiplatelet therapy and most patients combined with PPI [27]. It usually occurs within 12 months after taking the drug and the peak reaches [28] at 3 months. The patient was characterized by a clear history of decompensated cirrhosis with EVB and was given 3 months of medication of aspirin. But he was admitted because of hematochezia without vomiting and hematemesis. Lowering portal pressure and acid suppression therapy in another hospital failed to effectively stop bleeding and EVB diagnosis was not clear. Under the premise of maintaining the stable life signs of the patient, emergency gastroscopy was performed to exclude the possibility of EVB and upper gastrointestinal bleeding. The first colonoscopy found suspicious bleeding lesions (sigmoid colon tumor: tubular adenoma), and treated by endoscopic snare ligation; second underwent colonoscopy identified the cause of the bleeding and third colonoscopy identified the complete removal of sigmoid colon tubular adenoma.

### Conclusions

In this case, through clinical observation and analysis, EVB was excluded by gastroscopy and lower gastrointestinal bleeding due to sigmoid colon tumor was diagnosed by colonoscopy. The patient achieved excellent therapeutic effect because of positive endoscopic treatment of suspicious lesions.

### Acknowledgements

This study was supported by grants (JCYJ-20160422164132993, JCYJ2016042217025-1848, JCYJ20150403102020231, JCYJ20130-402092657774, 201201013) from Science and technology plan project of Shenzhen.

**Disclosure of conflict of interest**

None.

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**References**

- [1] de Franchis R. Expanding consensus in portal hypertension: report of the Baveno VI consensus workshop: stratifying risk and individualizing care for portal hypertension. *J Hepatol* 2015; 63: 743-752.
- [2] Groszmann RJ, Garcia-Tsao G, Bosch J, Grace ND, Burroughs AK, Planas R, Escorsell A, Garcia-Pagan JC, Patch D, Matloff DS, Gao H, Makuch R; Portal Hypertension Collaborative Group. Beta-blockers to prevent gastroesophageal varices in patients with cirrhosis. *N Engl J Med* 2005; 353: 2254-2261.
- [3] Shi KQ, Liu WY, Pan ZZ, Ling XF, Chen SL, Chen YP, Fan YC, Zheng MH. Secondary prophylaxis of variceal bleeding for cirrhotic patients: a multiple-treatments metaanalysis. *Eur J Clin Invest* 2013; 43: 844-854.
- [4] Garbuzenko DV. Contemporary concepts of the medical therapy of portal hypertension under liver cirrhosis. *World J Gastroenterol* 2015; 21: 6117-6126.
- [5] Albillos A, Tejedor M. Secondary prophylaxis for esophageal variceal bleeding. *Clin Liver Dis* 2014; 18: 359-370.
- [6] D'Amico G, Pagliaro L, Bosch J. Pharmacological treatment of portal hypertension: an evidence-based approach. *Semin Liver Dis* 1999; 19: 475-505.
- [7] Gluud LL, Krag A. Banding ligation versus beta-blockers for primary prevention in oesophageal varices in adults. *Cochrane Database Syst Rev* 2012; 8: CD004544.
- [8] Garcia-Tsao G, Bosch J. Management of varices and variceal hemorrhage in cirrhosis. *N Engl J Med* 2010; 362: 823-832.
- [9] Lo GH. The optimal interval of endoscopic variceal ligation: an issue of controversy. *Gastrointest Endosc* 2015; 81: 774.
- [10] Funakoshi N, Segalas-Largey F, Dony Y, Oberti F, Valats JC, Bismuth M, Daurès JP, Blanc P. Benefit of combination B-blocker and endoscopic treatment to prevent variceal bleeding: a meta-analysis. *World J Gastroenterol* 2010; 16: 5982-5992.
- [11] Puente A, Hernandez-Gea V, Graupera I, Roque M, Colomo A, Poca M, Aracil C, Gich I, Guarner C, Villanueva C. Drugs plus ligation to prevent bleeding in cirrhosis: an updated systematic review. *Liver Int* 2014; 34: 823-833.
- [12] Wallace MB, Kiesslich R. Advances in endoscopic imaging of colorectal neoplasia. *Gastroenterology* 2010; 138: 2140-2150.
- [13] Coe SG, Wallace MB. Colonoscopy: new approaches to better outcomes. *Curr Opin Gastroenterol* 2012; 28: 70-75.
- [14] Di Giorgio P, De Luca L, Calcagno G, Rivellini G, Mandato M, De Luca B. Detachable snare versus epinephrine injection in the prevention of postpolypectomy bleeding: randomized and controlled study. *Endoscopy* 2004; 36: 860-863.
- [15] Walsh RM, Ackroyd FW, Shellito PC. Endoscopic resection of large sessile colorectal polyps. *Gastrointest Endosc* 1992; 38: 303-309.
- [16] Baillie J. Postpolypectomy bleeding. *Am J Gastroenterol* 2007; 102: 1151-1153.
- [17] Dobrowolski S, Dobosz M, Babicki A, Głowacki J, Nałecz A. Blood supply of colorectal polyps correlates with risk of bleeding after colonoscopic polypectomy. *Gastrointest Endosc* 2006; 63: 1004-1009.
- [18] Rabeneck L, Paszat LF, Hilsden RJ, Saskin R, Leddin D, Grunfeld E, Wai E, Goldwasser M, Sutradhar R, Stukel TA. Bleeding and perforation after outpatient colonoscopy and their risk factors in usual clinical practice. *Gastroenterology* 2008; 135: 1899-1906.
- [19] Panteris V, Haringsma J, Kuipers EJ. Colonoscopy perforation rate, mechanisms and outcome: from diagnostic to therapeutic colonoscopy. *Endoscopy* 2009; 41: 941-951.
- [20] Koklu S, Basar O, Akbal E, Ibiş M. Gastric serrated adenoma polyp treated with endoscopic band ligation (with video). *Surg Laparosc Endosc Percutan Tech* 2010; 20: e204-e205.
- [21] Antithrombotic Trialists' Collaboration. Collaborative meta-analysis of randomized trials of antiplatelet therapy for prevention of death, myocardial infarction, and stroke in high risk patients. *BMJ* 2002; 324: 71-86.
- [22] Yeomans ND, Lanis AI, Talley NJ, Thomson AB, Daneshjoo R, Eriksson B, Appelman-Eszczuk S, Långström G, Naesdal J, Serrano P, Singh M, Skelly MM, Hawkey CJ. Prevalence and incidence of gastroduodenal ulcers during treatment with vascular protective doses of aspirin. *Aliment Pharmacol Ther* 2005; 22: 795-801.
- [23] McQuaid KR, Laine L. Systematic review and meta-analysis of adverse events of low-dose aspirin and clopidogrel in randomized controlled trials. *Am J Med* 2006; 119: 624-638.
- [24] Raju N, Sobieraj-Teague M, Hirsh J, O'Donnell M, Eikelboom J. Effect of aspirin on mortality in the primary prevention of cardiovascular disease. *Am J Med* 2011; 124: 621-629.

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- [25] Takeuehi K. Pathogenesis of NSAID-induced gastric damage: importance of cyclooxygenase inhibition and gastric hypermotility. *World J Gastroenterol* 2012; 18: 2147-2160.
- [26] Higuchi K, Umegaki E, Watanabe T, Yoda Y, Morita E, Murano M, Tokioka S, Arakawa T. Present status and strategy of NSAIDs-induced small bowel injury. *J Gastroenterol* 2009; 44: 879-888.
- [27] Casado Arroyo R, Polo-Tomas M, Roncales MP, Scheiman J, Lanas A. Lower GI bleeding is more common than upper among patients on dual antiplatelet therapy: long-term follow-up of a cohort of patients commonly using PPI co-therapy. *Heart* 2012; 98: 718-723.
- [28] Lanas A, García-Rodríguez LA, Arroyo MT, Gómollón F, Feu F, González-Pérez A, Zapata E, Bástida G, Rodrigo L, Santolaria S, Güell M, de Argila CM, Quintero E, Borda F, Piqué JM; Asociación Española de Gastroenterología. Risk of upper gastrointestinal ulcer bleeding associated with selective cyclo-oxygenase-2 inhibitors, traditional non-aspirin non-steroidal anti-inflammatory drugs, aspirin and combinations. *Gut* 2006; 55: 1731-1738.