Non cirrhotic varices: A case series

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Background

Variceal bleeding is a common associated symptoms in liver cirrhotic patient, whether in esophageal or gastric varices. However, in non cirrhotic patient with variceal bleeding had not stastistic before, especially in Eastern. We followed up 25 cases which had non-cirrhotic varices and analyzed the etiology, bleeding rate and mortality.

Aims:

To realize the etiology, bleeding rate and mortality of non-cirrhotic patient

Methods:

We had collected data from 2006-2012, by computerized hospital registration system whose CT report had key word of collateral vein or portal hypertension, and rule out who had HCC and liver cirrhosis. Enrolled CT or EGD report who had varices. About 25 patients collected from 1748 patients without liver cirrhosis but had variceal bleeding. We had analyzed the patient's back ground, etiology, bleeding rate, mortality.

Results:

Man(72%) is more then woman(28%). The etiology was pancreas cancer(28%), pancreatitis(28%), unknown(12%), cholangitis(8%), GIST with liver metasis(4%), Castleman disease with obstruction (4%), Cholangiocarcinoma with liver metastasis (4%), Epitheloid hemangioendothelioma (4%), pancreas neuroendocrine tumor (4%), trauma(4%). Most patient had initial presentation of abdomen pain(72%), Fever(12%), jaundice(8%), UGI bleeding(8%). No myeloproliferative disease or antiphosphilipid syndrome. Almost all patient (84%) had splenomegaly. Gastric varices (59%) was more then Esophageal varices (32%) alone, combined GV and EV. All cases had vessel thrombosis, splenic vein(56%) was more then both portal vein(20%), combined splenic and portal(16%). About the variceal bleeding rate was 30%, with mortality due to bleeding was 9%. We also noted that pancreas CA(50%) with EV bleeding (75%) was most, others like pancreas neuroendocrine tumor, Castleman disease,

and unknown was the other half.

About the treatment of initial esophageal variceal bleeding, most are stopped by EVL, only one was stopped by SB tube successfully. GV was stopped by Histoacryl injection. There was no surgical intervention nor TIPS done.

Conclusions:

Variceal bleeding is a common complication in cirrhotic and non-cirrhotic portal hypertension patient. The pathophysiology of variceal formation was portal hypertension, which is related to splenic vein or portal vein thrombosis. In western country, splenic vein or portal vein thrombosis was related to systemic factors such as myeloproliferative disease, antiphospholipid syndrome, protein C,S deficiency more then local factors as pancreatitis,trauma, malignancy. But in our analyze, local factors such as pancreas CA, pancreatitis was the most. Only one case was suspected to have thrombotic disease with unknown reason.

Second, the bleeding rate of variceal was 30% and only two mortality(9%). Most variceal bleeding can be stopped by EVL or Histoacryl injection. Two patients who died from variceal bleeding was case of pancrease cystadenocarcinoma with liver metastasis and Castleman disease with splenic obstruction. They had failure of EVL .Surgical intervention or SB tube was not done due to family refused.

Third, about these thrombosis patient, we had not used anticoagulation drugs. We had not noted DVT nor ischemic bowel happened.

Fourth, It is interesting that most non-cirrhotic patient who had variceal bleeding, had received EGD due to abdomen pain and coffee ground vomitus. However, there was more then two-third had noticed without bleeding. One even was gastirc ulcer bleeding.

Fifth, about the treatment of variceal bleeding, EVL for EV and Histoacryl injection for GV was still the first choice and the successful rate was high. To those refractory cases, SB tube is our second choice. We had not tried TIPS or other surgical intervention in this study.

In conclusion, in non-cirrhotic variceal bleeding,
Maybe there was different onset age, etiology, complication
from The Eastern and The Western people.