RECENT STUDIES INMANAGEMENT OF PORTAL VEIN THROMBOSIS

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Abstract

Portal vein thrombosis is a life threatening vascular disorder of the liver. I will review the recent advance regarding the epidemiology, etiology, management, and prognosis of portal vein thrombosis.

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Abberviations

PVT: portal vein thrombosis

HCC: hepatocellular carcinoma

MTHFR: methylene tetrahydrofolate reductase

PAI:plasminogen activator inhibitor

SMVT: superior mesenteric vein thrombosis

EXPVO: extra hepatic portal vein obstruction

EUS: endoscopic ultra sonar

CUES: contrast enhanced ultra sonar

NCCT: non contrasted computed tomography

CE-MRI: contrast enhanced magnetic resonance imaging

ERCP: endoscopic retrograde cholangio-pancreatography

MRCP: magnetic resonance cholangio-pancreatography

APPT: activated thromboplastin time

HIT: heparin induced thrombocytopenia

UFH : unfraction heparin

LMWH:low molecular weight heparin

LBW: lean body weight

AJBW: adjusted body weight

NOACs : new oral anticoagulants

PPC:prothrombin complex concenterate

APCC: activated prothrombin complex

EST: endoscopic sclerotherapy

TIPS: trans jugular intrahepatic Porto systemic anastomosis

MELD: model for end stage liver disease

TPA: tissue plasminogen activator

TGF-B: transforming growth factor beta

PAR:protease activated receptors

SMA-SMV: superior mesenteric artery and vein

PH: portal hypertension

VB: variceal bleeding

VTE:venous thromboembolism

keywords

Portal vein thrombosis

Virchow's triad

Portosystemic anastomosis

Doppler and ultrasonar

Cavenoma

Variceal bleeding

Endoscopic ligation and sclerotherapy

Heparin and new oral anticoagulants

Transjugular intrahepatic portosystemic anastomosis

Surgical shunts

Liver transplantation

INTRODUCTION

The term portal vein thrombosis (PVT) refers to thecomplete or partial obstruction of blood flow in the trunk of portal vein, due to the presence of a thrombus in the vessel lumen(**Bayraktar and Harmanci ,**2006).This includes its right and left intrahepatic branches. It may evenextend to the splenic or superior mesenteric veins or towards the liver involving intra hepatic portal branches **(Chawla,** et al. 2009).

PVTis considered a rare event. It can occur either in childhood or in adulthood, with the same incidence. the reported lifetime risk of developing PVT in the general population is approximately 1%.its prevalence among cirrhoticpatients ranges between 4.4%-15%, and is responsible forabout 5%-10% of overall cases of portal hypertension (**Amitrano,**et al,2004**)** Moreover, inpatients with a hepatocellular carcinoma, the incidence of PVT rises to 10%-40%..the first case of PVT was reported in 1868 by Balfourand Stewart, describing a patient presenting splenomegaly,ascites, and variceal dilation (**Wang,**et al,2005).

Several causes can be involved in the pathogenesis of PVT and, frequently, more than one coexist. A simple classification distinguishes between local (70%) and systemic (30%) risk factors, Inflammatory abdominal foci (such as appendicitis, diverticulitis,

inflammatory bowel disease pancreatitis,cholecystitis, hepatic abscesses, and cholangitis), livercirrhosis or tumors, represent the most common localthrombotic risk factors. Malignancies, frequently of hepatic or pancreatic origin, are responsible for 21%-24% of overall cases of PVT.Direct vascular invasion, compression by tumor mass, or ahypercoagulable state are the mechanisms involved in neoplastic PVT development; hormonal factors might alsoplay a role in this process, especially in men.PVT is common in patients affected by liver cirrhosis, with a risk related to the severity of the disease(**Ponziani,**et al,2010). PVT can be classified also into four categories, depending on the extension: confined to the portal vein beyond the confluence of the splenic vein; extended to the superior mesenteric vein, but with patent mesenteric vessels; extended to the whole splanchnic venous system, but with large collaterals; or with only fine collaterals. This classification is useful to evaluate a patient's operability and clinical outcome. In fact, when thrombosis is extended to both portal and mesenteric veins, the risk of bowel ischemia is considerable and mortality high, despite a lower risk of variceal bleeding (**Riva,**et al, 2012).

Clinicalpresentationis different in the context of acute or chronic onset and depends on the development and the extent of a collateral circulation. Intestinal congestion and ischemia, with abdominal pain, diarrhea, rectal bleeding, abdominal distention,