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REVIEW ARTICLE

Natural history of pancreatitis-induced splenic vein thrombosis: a systematic review and meta-analysis of its incidence and rate of gastrointestinal bleeding

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Abstract

Background: Pancreatitis-induced splenic vein thrombosis (PISVT) is an acquired anatomic abnormality that impacts decision making in pancreatic surgery. Despite this influence, its incidence and the rate of associated gastrointestinal (GI) bleeding are imprecisely known.

Methods: The MEDLINE, EMBASE, Cochrane Central Register of Clinical Trials and Cochrane Database of Systematic Reviews databases were searched from their inception to June 2010 for abstracts documenting PISVT in acute (AP) or chronic pancreatitis (CP). Two reviewers independently graded abstracts for inclusion in this review. Heterogeneity in combining data was assumed prior to pooling. Random-effects meta-analyses were performed to estimate percentages and 95% confidence intervals.

Results: After review of 241 abstracts, 47 studies and 52 case reports were graded as relevant. These represent a cohort of 805 patients with PISVT reported in the literature. A meta-analysis of studies meeting inclusion criteria shows mean incidences of PISVT of 14.1% in all patients, 22.6% in patients with AP and 12.4% in patients with CP. The incidence of associated splenomegaly was only 51.9% in these patients. Varices were identified in 53.0% of patients and were gastric in 77.3% of cases. The overall rate of GI bleeding was 12.3%.

Conclusions: Although reported incidences of PISVT vary widely across studies, an overall incidence of 14.1% is reported. Splenomegaly is an unreliable sign of PISVT. Although the true natural history of PISVT remains unknown, the collective reported rate of associated GI bleeding is 12.3%.

Keywords

acute pancreatitis, chronic pancreatitis, splenic vein thrombosis, left-sided portal hypertension, sinistral hypertension, gastric varices, gastrointestinal bleeding

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Introduction

Pancreatitis-induced splenic vein thrombosis (PISVT) is an acquired disorder that occurs as a sequel to both acute (AP) and chronic pancreatitis (CP). Although as many as 37 different specific aetiologies for splenic vein thrombosis (SVT) have been reported,[1] the majority are related to diseases of the pancreas.[2] Although PISVT has an historical association with neoplasms,[3] recent data incriminate AP and CP as its principal causes.[4–8] Regardless of its aetiology, SVT generates a localized form of portal hypertension commonly referred to as 'sinistral', 'left-sided' or 'linear'. Collateral blood flow develops through the splenoportal or gastroepiploic systems and the resulting localized venous hypertension may produce gastric, oesophageal or colonic varices.

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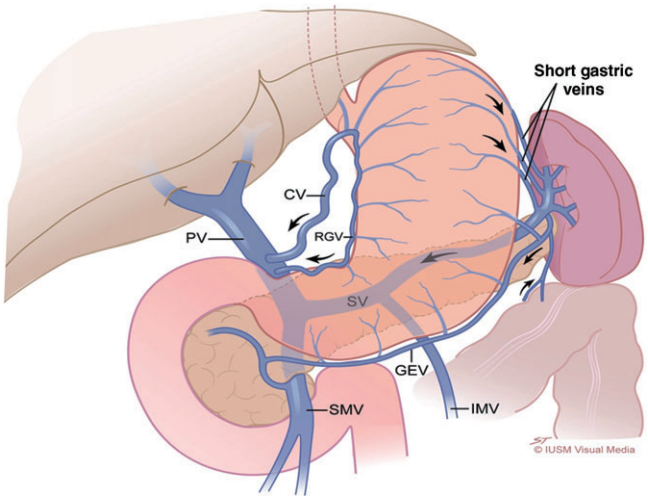

Figure 1 Normal venous anatomy. Arrows indicate the directional flow of venous blood. CV, coronary vein; PV, portal vein; RGV, right gastric vein; SV, splenic vein; GEV, gastroepiploic vein; SMV, superior mesenteric vein; IMV, inferior mesenteric vein

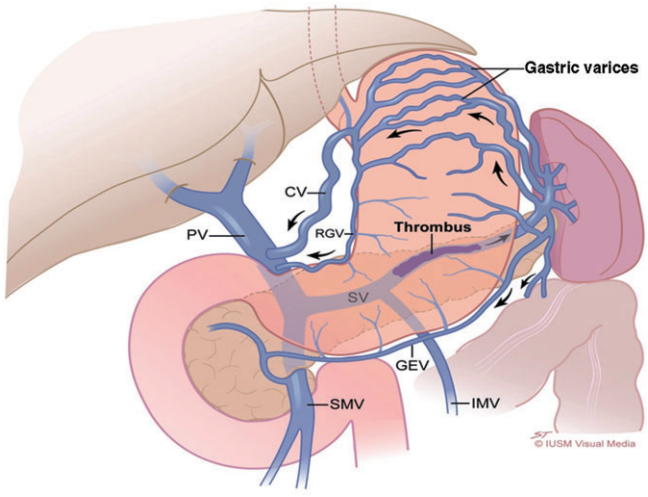

Figure 2 Splenic vein thrombosis causing left-sided portal hypertension. Note the gastric varices, dilatation of short gastric, gastroepiploic (GEV) and coronary (CV) veins. The portal vein (PV), superior mesenteric vein (SMV) and inferior mesenteric vein (IMV) are patent. RGV, right gastric vein; SV, splenic vein

These varices are a potential source of significant gastrointestinal (GI) bleeding (Figs 1 and 2).[9,10] In relation to operative management, it has been suggested that patients with PISVT and a prior history of upper GI tract bleeding or symptomatic hypersplenism may represent a high-risk subgroup in whom the risk : benefit profile is altered in favour of splenectomy.[11–13] By contrast, patients in a small cohort without this history of bleeding, in whom PISVT was identified through imaging, were found to have an incidence of bleeding of only 3.8% (two of 53 patients) over a median follow-up of 34 months.[14]

Although PISVT is an important anatomic abnormality that impacts operative decision making in pancreatic surgery, no consensus has been reached on either its incidence in patients with pancreatitis or the rate of associated GI bleeding.[1] The aim of this study was to conduct a systematic review of the published literature on PISVT, with appropriate filters and controls, to determine both its incidence and the rate of associated GI tract bleeding.

Materials and methods

Data sources

The MEDLINE, EMBASE, Cochrane Database of Systematic Reviews (CDSR) and Cochrane Central Register of Clinical Trials (CCRCT) databases were searched from their inception up to and including June 2010. Search terms were used in both isolation and combination to identify all published evidence. Reference lists from identified review articles and published studies were queried to identify articles not found through our initial database search.

Data extraction

Two investigators (JRB and TJH) independently reviewed the titles and abstracts of all returned references regardless of language or publication status to identify studies for inclusion in the analysis. All identified articles were examined using a predesigned proforma and the data collected were entered into a database for analysis. A list of gathered data is detailed in Table 1. The methodological quality of studies was assessed for a minimum Oxford Centre for Evidence-Based Medicine (CEBM) level of 2B.[15] When appropriate, studies were allocated to separate quantitative cohorts for independent meta-analyses of variables. Results of studies yielding a heterogeneous dataset of isolated and non-isolated PISVT were corrected for if results were reported in a manner that allowed the exclusion of outcomes specific for an *isolated* PISVT patient cohort. An analogous process was rarely employable for datasets confounded by malignancy. References reporting datasets with incomplete or inconsistent isolation of a non-confounded PISVT cohort were excluded (Table 2). A schematic diagram depicting reference flow through the systematic review process is shown in Fig. 3.

Statistical analysis

All included references were assigned to appropriate cohorts for individual meta-analyses of variables. A schematic diagram depicting the flow of references through the quantitative arm of this study is shown in Fig. 4. When combining data from the trials, we assumed the presence of heterogeneity existed prior to pooling and used the random effects model developed by DerSimonian.[16] This model allows for adjustment for variability between trials by providing a more conservative estimate of the range of an effect. Individualized random effects meta-analyses were performed to estimate percentages and 95% confidence intervals (CIs) for all endpoints queried.