

degree of perilobular or ($p=0.655$) intralobular fibrosis ($p=0.587$) or acinar atrophy ($p=0.584$).

Conclusions: Our results indicate that the pancreatic stroma is associated with PDAC patients' DSS. Additionally, the more severe the fibrosis, acinar atrophy and chronic inflammation, the worse the impact on DSS, thereby warranting further studies investigating stroma-targeted therapies.

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EARLY DIAGNOSIS OF POSTPANCREATECTOMY ACUTE PANCREATITIS AFTER PANCREATICODUODENECTOMY: A STUDY USING BIOMARKERS AND DIFFUSION-WEIGHTED MAGNETIC RESONANCE IMAGING

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Introduction: Early phases of postpancreatectomy acute pancreatitis (PPAP) are poorly characterized but might represent novel therapeutic opportunities. This study aims to investigate the radiologic, biochemical, and clinical spectrum associated with PPAP.

Method: 65 patients undergoing pancreaticoduodenectomy prospectively collected. IVIM magnetic resonance imaging (MRI) at 1.5T scanner was performed on a post-operative day (POD) 3. Serum pancreatic amylase and lipase were assessed after surgery and daily until POD 5. PPAP was defined based on radiologic findings.

Results: PPAP incidence was 9.2%. All patients with PPAP had a sustained increased serum enzyme activity persisting at least 48 hours (POH—postoperative serum hyper-amylasemia, -lipasemia) and lead to the worst post-operative outcome with (all $p<0.05$) high incidence of local (postoperative pancreatic fistula: 83%) and systemic morbidity (Clavien Dindo \geq grade IIIb: 67%). Still, 21.5% and 24.6% of cases with no radiologic features of PPAP showed POH, without significant differences in median serum activity. Patients with POH-PPAP had significantly lower values of true diffusion coefficient (D) and higher heterogeneity of perfusion parameters: D* and F on IVIM MRI. No macroscopic changes consistent with PPAP were identified at any MRI. 21 patients subsequently underwent computer tomography imaging for clinical worsening, and 6 have PPAP confirmed.

Conclusions: PPAP is characterized by early biochemical alteration, without correlation between severity and enzymes elevation. Macroscopic radiologic changes appear later and correlate with the most severe clinical pictures. This paper paves the ground for future prospective series validating correlations between biochemical and radiological features, eventually leading to changes in clinical practice management.

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SPLANCHNIC VEIN THROMBOSIS IN ACUTE PANCREATITIS: INCIDENCE, RISK FACTORS AND LONG TERM OUTCOMES

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Introduction: There is paucity of data on the incidence, risk factors and role of anticoagulation for splanchnic vein thrombosis (SVT) in acute pancreatitis (AP).

Methods: A retrospective review of AP admissions between 2018-2021 across the North East of England was undertaken. Data on demographics, etiology, severity of AP and SVT was collected. In addition, a selective anticoagulation policy for portal vein thrombosis (PVT) and progressive splenic vein thrombosis was explored.

Results: 401 patients were included with a mean age of 57.0 and M:F ratio of 1.6:1. 152 patients developed intestinal oedematous pancreatitis and 249 developed necrotising pancreatitis based on Revised Atlanta criteria (RAC). 109 patients (27.2%) developed SVT of which 27 developed a PVT and splenic vein thrombus, 36 PVT only and 46 splenic vein thrombus only.

On univariate analysis, alcoholic aetiology, severe pancreatitis, necrotising pancreatitis with $>50\%$ necrosis and elevated CRP at 2 weeks were risk factors for developing SVT. On multivariable analysis, alcohol aetiology (OR 2.6, $p = 0.002$), and $>50\%$ pancreatic necrosis (OR 14.6, $p = 0.048$) increased the risk of developing SVT.

58 patients received anticoagulation for SVT, with a median duration of 90 days of anticoagulation. Recanalization rates were higher for PVT when compared to splenic vein thrombosis. 6 patients developing bleeding complications whilst on anticoagulation therapy.

Conclusion: A third of patients with AP develop SVT, particularly those with severe AP secondary to alcohol and with extensive pancreatic necrosis. A selective anticoagulation policy was associated with improved recanalization rates and fewer bleeding complications.

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DIAGNOSIS AND TREATMENT OF EXOCRINE PANCREATIC INSUFFICIENCY IN CHRONIC PANCREATITIS: AN INTERNATIONAL EXPERT SURVEY AND CASE VIGNETTE STUDY

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Introduction: Despite evidence-based guidelines, exocrine pancreatic insufficiency is frequently underdiagnosed and undertreated in patients with chronic pancreatitis. Therefore, the aim of this study is to provide insight into the current opinion and clinical decision-making of international pancreatologists regarding the management of exocrine pancreatic insufficiency.

Methods: An online survey and case-vignette study was sent to experts in chronic pancreatitis and members of various pancreatic associations: EPC, E-AHPBA and DPSG. Experts were selected based on publication record from the past 5 years.

Results: Overall, 252 pancreatologists participated of whom 44% had ≥ 15 years of experience and 35% treated ≥ 50 patients with chronic pancreatitis per year. Screening for exocrine pancreatic insufficiency as part of the diagnostic work-up for chronic pancreatitis is performed by 69% and repeated annually by 21%. About 74% considers nutritional assessment to be part of the standard work-up. Patients are most frequently screened for deficiencies of calcium (47%), iron (42%), vitamin D (61%) and albumin (59%). In case of clinically steatorrhea, 71% prescribes enzyme supplementation. Of all pancreatologists, 40% refers more than half of their patients to a dietician. Despite existing guidelines, 97% supports the need for more specific and tailored instructions regarding the management of exocrine pancreatic insufficiency.

Conclusion: This survey identified a lack of consensus and substantial practice variation among international pancreatologists regarding guidelines pertaining the management of exocrine pancreatic insufficiency. These results highlight the need for further development and adaptation of these guidelines according to current expert opinion and the level of available scientific evidence.

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UTILITY OF PRESEPSIN FOR PROGNOSIS OF INFECTED COMPLICATIONS OF ACUTE NECROTIZING PANCREATITIS

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Introduction: Early determination of the patients with high risk for infected complications of acute necrotizing pancreatitis (ANP) is crucial for their effective treatment. So purpose of our research was to study utility of presepsin determination for prognosis of subsequent development of infected complications of ANP.

Method: Prospective study of 115 patients with suspected infected ANP admitted to single intensive care department has been performed. Level of presepsin in patients with system inflammatory response syndrome (SIRS) lasted longer than 1 week was estimated every 3rd–5th day until invasive treatment was applied. Bacteriological investigation of acquired material served as criteria for diagnosis of infected necrotic collections.

Results: Bacteriological investigation of necrotic collections confirmed infection presence in 80 (69.5%) patients. The rate of presepsin was significantly higher in the patients with infected complications than those in the individuals with sterile pancreatic necrosis with cut-of level above 632 pg/ml of at time of invasive procedure (0.956, $p < 0.001$), 457 pg/ml – within week before decision of its application (0.916, $p < 0.001$) and 403 pg/ml (0.876, $p < 0.05$) – in case of prolonged SIRS.

Conclusions: Presepsin level over 403 pg/ml in patients with prolonged SIRS is highly associated with subsequent development of infected complications of ANP and may serve as guide for antibacterial therapy.

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CHANGES IN ACTIVITY OF HSPA1A AND HSPA1L IS ASSOCIATED WITH EARLY ACUTE PANCREATITIS SEVERITY

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Introduction: Genome assessment shows promise as a potential method for predicting acute pancreatitis (AP) severity. We hypothesized that Heat Shock Protein 70 (HSP70) single nucleotide polymorphisms (SNPs) and expression changes may play a role in early detection of acute pancreatitis severity.

Methods: A total of 57 AP patients and 52 age- and sex-matched healthy controls were studied. Peripheral blood samples from pancreatitis patients were collected upon admission. Two SNPs of the HSP70-gene family were selected. RNA was extracted parallel to genomic DNA from AP patients (N=12) and healthy controls (N=21). Gene expression of two HSP70 family members HSPA1A and HSPA1L was measured using TaqMan gene expression assays via reverse transcription quantitative polymerase chain reaction (RT-qPCR).