Journal Scan

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de Rijk F, Sissingh NJ, Boel TT, et al. Development of pancreatic diseases during long-term follow-up after acute pancreatitis: a post-hoc analysis of a prospective multicenter cohort. J Gastroenterol Hepatol 2024;39(4):674–684. DOI: 10.1111/jgh.16453

It is now known that acute pancreatitis (AP), recurrent acute pancreatitis (RAP), and chronic pancreatitis (CP) represent a disease continuum, though the mechanism and risk factors underlying disease progression are still not very clear. In this multicentric cohort analysis, a total of 1,184 adult patients who presented with AP were prospectively registered and followed up from 2008 to 2015, with the primary endpoints being RAP, CP, or pancreatic cancer. A few secondary endpoints were also taken into consideration as new onset of diabetes mellitus and/or exocrine pancreatic insufficiency (EPI), medication for pancreatic pain, endoscopic or surgical pancreatic interventions, quality of life (QoL), pain severity, and mortality. This study observed that 25% of patients developed RAP in 9 months, 6% of patients progressed to CP in 31 months, and 1% of patients were diagnosed with pancreatic cancer in 24 months. For the development of RAP, the independent risk factors were alcoholic and idiopathic pancreatitis and lack of any pancreatic intervention in the initial AP episode. Endoscopic retrograde cholangiopancreatography (ERCP) and cholecystectomy performed within 3 months after the onset of AP were identified as independent protective factors. For CP, factors independently associated were alcoholic and idiopathic AP, RAP, pancreatic intervention performed during the initial episode, and male sex. Only 14 patients developed pancreatic cancer, and this number was insufficient for analysis. However, out of 14 patients, 6 patients (43%) had RAP before the development of cancer. QoL was not significantly different between patients with and without progression to RAP and CP. Both RAP and CP patients reported significantly higher pain scores. The drawback of this study was that many common factors for the development of primary endpoints were not taken into account.

Comment: In the pediatric population, pancreaticobiliary abnormalities or genetic risk factors have a higher rate of progression to RAP or CP, with a progression rate of 30 and 27%, respectively. However, this study may give some insight into the protective factors and may be useful in the pediatric population.

Jarasvaraparn C, Hartley C, Karnsakul W. Updated clinical guidelines on the management of hepatitis C infection in children. Pathogens 2024;13(2):180. DOI: 10.3390/pathogens13020180

Though children present only a small proportion of hepatitis C virus (HCV) infection, the prevalence is rising among young adults mainly due to illicit substance abuse. These young adults would raise the prevalence of HCV in adulthood. Women of childbearing age who are HCV positive can transmit it vertically in 5% of infants as perinatal transmission is the most common mode of HCV transmission in children. In 20–45% of infants, HCV may clear spontaneously within the first 2 years of life. Hepatitis C virus acquired later in life may get cleared spontaneously in only 15–20% of cases. Hepatitis C virus is a slowly progressive disease, and the development of advanced liver conditions is not generally seen in children but in adults. It may progress to chronic infection, including cirrhosis, portal hypertension, hepatic decompensation with hepatic encephalopathy, and hepatocellular carcinoma. For identification and early management of perinatally infected children, screening is very important. Centers for Disease Control and Prevention (CDC) recommends universal HCV testing of adults, especially pregnant women, and all perinatally exposed infants with a nucleic acid test (NAT) to detect HCV ribonucleic acid (RNA) at 2–6 months of age. If there is an undetectable HCV RNA result at or after 2 months of age, no further follow-up is required. However, NAT is recommended for perinatally exposed infants and children aged 7–17 months who previously have not been tested, followed by anti-HCV antibody test for HCV RNA for perinatally exposed children aged ≥18 months who previously have not been tested. Apart from exposed infants, children with a high risk of developing HCV infection should be screened, for example, children with hepatitis or human immunodeficiency virus (HIV), international adoptees or refugees, victims of a sexual assault, and adolescents with history of multiple sexual partners or illicit drug use. For the definite diagnosis of HCV infection in children aged ≥18 months old, the initial evaluation is anti-HCV antibody testing. A reactive or indeterminate antibody test should be checked by HCV RNA testing. Detection of HCV RNA suggests viremia/active disease, and such children should be further evaluated to determine the genotype and monitor for disease progression or spontaneous clearance. In case anti-HCV antibody test is positive and HCV RNA is not detected, then either the patient has cleared the virus spontaneously or there was a false-positive antibody test. In children aged <18 months old, initially, HCV RNA should be done at 2 and 6 months of age. Nondetection suggests unlikely infection. If HCV RNA is detected, it only suggests that

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the infant has acquired the infection, but it does not suggest chronicity and may clear spontaneously. In such children, anti-HCV antibodies should be checked at 18 months of age, and those with positive anti-HCV antibodies should be tested for HCV RNA. For the management of chronic infection in children above 3 years of age, direct-acting antivirals (DAAs) that have activity against all known HCV genotypes can be used for 12 weeks. In case of nonresponse or relapse, sofosbuvir/velpatasvir/voxilaprevir can be offered. There are no data showing treatment of acute HCV infection in children.

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