

Does autoimmune pancreatitis type III exist?

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Presentation

Hepatobiliary

Immunology

Pancreas

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The speaker questions whether immune checkpoint inhibitor-associated pancreatic injury warrants classification as a distinct type 3 autoimmune pancreatitis, emphasizing the need for awareness and interdisciplinary discussion rather than definitive disease labeling.

KEY POINTS

- ICI-associated pancreatic injury is characterized by elevated lipase levels (often three times the upper limit of normal) that are frequently asymptomatic, CD8-positive T-cell infiltration, and progressive pancreatic volume loss with increased diabetes risk in follow-up, as reported in cohort studies screening thousands of ICI-treated cancer patients.
- The speaker presents a clinical dilemma: the condition is often asymptomatic, yet removing potentially life-saving cancer immunotherapy for asymptomatic lipase elevation or pancreatic changes may cause greater patient harm than the pancreatic injury itself.
- No clear treatment recommendations exist; limited evidence suggests that adding steroids may worsen pancreatic atrophy, and combining multiple checkpoint inhibitors simultaneously increases injury risk according to predictive scoring.
- Pancreatic volume loss occurs in both this ICI-associated injury and in type 1 autoimmune pancreatitis and appears to be permanent, distinguishing it from reversible acute pancreatitis.
- The speaker argues this entity is most likely an immune-related adverse event variant rather than a true pancreas-specific disease, noting that two of the three major cohort studies originate from a single research group and that histological criteria remain unclear.
- The speaker recommends that clinical symptoms and frank pancreatitis, not isolated lipase elevation, should guide decisions about ICI continuation, consistent with traditional pancreatology teaching that asymptomatic lipase elevation is not clinically significant.

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