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Timing and Route of Enteral Nutrition in Severe Acute Pancreatitis?

To the Editor:

We read with interest the recent *Pancreas* article by Wu et al[1] entitled "When to initialize enteral nutrition in patients with severe acute pancreatitis?" The timing for enteral nutrition initiation in severe pancreatitis is a relevant issue, but we believe that the study design does not allow answering the question. Indeed, this study evaluates essentially the impact of enteral nutrition in comparison to parenteral nutrition because 83% of the patients in "delayed enteral nutrition" group received exclusive parenteral nutrition. However, it is already well established that enteral nutrition was superior to parenteral nutrition to decrease infectious and surgical complications, as well as mortality in severe pancreatitis.[2] On the other hand, the authors used the presence of necrosis to define the expected severity of acute pancreatitis. To date, it is accepted that the systemic inflammatory response syndrome is the better parameter to predict the expected severity of an acute pancreatitis.[3] To assess comparability between the groups, SIRS and organ failure on admission are missing.

The main objective of an early enteral nutrition during severe acute pancreatitis is to maintain the integrity of the gut barrier and then to prevent bacterial translocation. The choice made by the authors to use nasojejunal feeding tube placed by endoscopy could be debated. Authors give no information about energy supply within the first 48 hours, but we can assume that some days are necessary to reach the goal of 25 to 30 kcal/kg per day because of delay for the tube placement and the progressive feeding speed (initiated at 20 mL/h). In a recent randomized trial comparing the early nasojejunal tube feeding with an oral diet, nasojejunal feeding was started at a median of 23 hours after admission, and the calories target was reached only on the fourth day.[4] Moreover, we previously observed that success rate of tube placement in the jejunum decreased with severity of pancreatitis.[5] Anyway, the concept of pancreatic rest to avoid pancreatic secretion is now outdated: a meta-analysis showed that nasogastric route would be as effective and safe as nasojejunal route.[6] So, the use of a nasogastric tube that can be placed at the bedside could allow starting enteral nutrition earlier.

In daily practice, the feasibility of a really early enteral nutrition remains uncertain because of potential paralytic ileus observed in severe acute pancreatitis. Most studies about nutritional management of severe acute pancreatitis do not report the failure rate of enteral nutrition initiation. It would be very informative to obtain data about intestinal tolerance to enteral nutrition and prevalence of ileus in the Wu et al study population. Despite of this large sample study, the feasibility of early enteral nutrition in severe acute pancreatitis is not established, and other studies are necessary to conclude.

The authors declare no conflict of interest.

Benoît Dupont, MD
Marietta Musikas, MD
Manh-Thông Dao, MD
Marie-Astrid Piquet, MD, PhD
Department of Hepato-Gastroenterology and Nutrition
Caen University Hospital
Caen, France
benoit-dupont@outlook.com

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A Case of Pancreatic Small Cell Neuroendocrine Carcinoma Associated With SIADH

To the Editor:

Pancreatic neuroendocrine neoplasms (PNENs) account for only 1% to 2% of pancreatic tumors.[1] Depending on the tumour grading, which is measured by Ki67, the World Health Organization 2010 classification distinguishes 3 subgroups of PNENs: PNET G1 (Ki67, ≤2%), PNET G2 (Ki67, 3% to 20%), and PNEC (pancreatic neuroendocrine carcinoma) G3 (Ki67, >20%). From a morphological viewpoint, PNECs are further classified as large cell and small cell types.[2,3] As far as functional status is concerned, PNENs can be associated with a specific syndrome (functioning tumors) due to secretion of peptides (insulinoma, gastrinoma, glucagonoma, and others); otherwise, they are considered nonfunctioning. A quote of PNENs can also be associated to paraneoplastic syndromes caused by substances not related to the specific cells of origin, such as parathyroid hormone, corticotropin, somatotropin, human chorionic gonadotropin, or vasopressin.[4] The syndrome of inappropriate antidiuretic hormone secretion (SIADH) is diagnosed in the presence of hyponatremia with retention of free water due to dysregulated release of ADH.[5]

CASE REPORT

We report the case of a 30-year-old man with a metastatic, poorly differentiated small cell NEC of the pancreas associated with SIADH. The patient was admitted in December 2012 to a local hospital because of jaundice. A contrast-enhanced computed tomography (CECT) scan showed a nonhomogeneous mass of the pancreatic head and uncinate process, measuring 40 mm, involving the retroperitoneal space and the horizontal part of the duodenum; no cleavage plan with the inferior vena cava was present. A rounded, hypodense right lobe hepatic lesion, measuring 50 mm, associated with biliary tree dilatation as well as many enlarged peripancreatic lymph nodes were also demonstrated (Fig. 1A, B). To palliate the biliary obstruction, an endoscopic retrograde cholangiopancreatography was performed, and a plastic stent (9 cm, 10 F) was positioned.

The patient was then referred to our center to complete diagnosis and disease