Nutritional therapy in acute pancreatitis—Time to take stock

Acute pancreatitis (AP) remains a formidable disease with significant mortality and major morbidity. The nutritional issues are at least as complex and controversial as other facets of therapy. AP is diabetogenic and catabolic and affects all major organ systems including the intestine. As early as 1973, it became one of the first non-surgical indications for parenteral nutrition (PN) [1]. The use of enteral nutrition (EN), initially universally spurned (“rest the pancreas, rest the gut”), was pioneered by McClave and others in the mid 1990s [2]. To reduce pancreatic secretions, nutritional administration was performed using a nasojejunal rather than a nasogastric tube. Patients with severe AP could be fed enterally with similar if not better outcomes compared with PN administration [3].

Placement of nasojejunal feeding tubes can be difficult, particularly in hospitals without the necessary radiologic or gastroenterologic expertise. Possibly as a reaction to this, nasogastric feeding was then somewhat daringly embraced and it was shown to give similar clinical outcomes as nasojejunal feeding in two trials [4,5]. Notwithstanding the criticism that the “nasojunal” groups in both these studies were in fact really nasoduodenal, the results in the nasogastric groups were quite acceptable, leaving many observers aghast at how badly shaken one of the great medical axioms of pancreatology had become.

Some of the nuances of EN in AP have now also been investigated. Elemental and semi-elemental formulae do not lead to better results compared with polymeric formulae. The addition of antioxidants, ω-3 fatty acids, arginine, and glutamine has all been trialed without a great deal of success [6]. However, diet therapy with stringent dietary fat restriction and ω-3 fatty acids supplementation was successful in preventing recurrent triglyceridemia-related pancreatitis during pregnancy [7]. Various regimens in terms of timing of administration and energy content of substrate have also been used without a clear consensus emerging with respect to these parameters.

Two recent studies have contributed to thinking in this area—the Dutch probiotic (“PROPATRIA”) trial [8] and the Australian and New Zealand multicenter observational study [9]. In the Dutch trial, probiotics were added to EN in patients with predicted severe AP. Nine of the 152 patients in the probiotic group developed intestinal infarction with eight deaths compared with none of the 144 patients in the placebo (standard EN) group. The possible explanations for these worrying results have been numerous and inconclusive. One wonders whether patients already on probiotics which are now widely available to consumers would also have fared so badly if they had developed severe AP. In view of the promising earlier study of probiotic use in AP, the lack of standardization in manufacture and administration of these substances merits scrutiny. AP per se impairs intestinal function and this study, if nothing else, highlights the importance of meticulous observation of gut function in AP patients. To the extent that the study was conducted in leading teaching hospitals, it also shows how quickly intestinal catastrophes become irretrievable and how inadequate is the monitoring of intestinal function in the critically ill.

In the multicenter observational Australasian study of 117 patients with severe AP, remarkably a majority were fed parenterally in the first instance [9]. The leading reason given for the use of PN rather than EN was “physician preference.” This widespread transgression of current guidelines was somewhat surprisingly no less prevalent in teaching hospitals. One wonders how the decision regarding nutritional route was taken. In how many of the study patients was there a full and frank discussion about the issues of nutritional support? Were patients commenced on PN more commonly during the weekend, when there may have been less nutritional expertise available? Interestingly over the course of the study, there were more patient days of EN compared with PN, indicating that several patients started on PN were changed to EN. Again, this is at variance with the usual approach in which EN is introduced early and PN is seen as salvage therapy, the benefit of which may not occur until the systemic inflammatory reaction is beginning to resolve.

Nasojejunal feeding in severe acute pancreatitis should be the standard of care and is seen by some as “proactive primary therapy” rather than as simply supportive or adjunctive [10]. Nonetheless, there is significant on-going controversy. Possibly due to this, physicians’ responses to the nutritional issues of this challenging disease are inconsistent and at times disturbingly illogical. The medical misadventure associated with probiotic use is also salutary and shows how inadequate monitoring is in this group of patients. If nutritional therapy in AP is to advance, better means of contemporaneous physiologic assessment are needed together with more strenuous physician education.

References


Andrew Thomson, M.B.B.S., M.R.C.P.(UK), F.R.A.C.P.
Gastroenterology and Hepatology Unit
The Australian National University
The Woden Hospital, Woden, Australia

Kavitha Subramaniam, M.B.B.S., M.P.H.
The Canberra Hospital
Woden, Australia

Andrew Davies, M.B.B.S., F.R.A.C.P.
The Intensive Care Unit, Alfred Hospital and
Monash University, Melbourne, Australia