Complications of Overfeeding the Critically Ill Patient

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- Patients who are very small, very large, or very old are particularly vulnerable to overfeeding.

- Excessive protein leads to azotaemia, hypertonic dehydration, hyperammonaemia and metabolic acidosis.

- Excessive carbohydrate leads to hyperglycaemia, hypercapnea and fatty liver.

- Excessive fat have caused hyperlipidaemia and fat-overload syndrome.

- Enteral feeding is more frequently associated with undernutrition; a patient is more likely to be overfed with TPN, which has a greater risk than enteral overfeeding.

Though perhaps not as dangerous as refeeding syndrome, overfeeding syndrome has its own disadvantages. If the overfed patient is able to tolerate such large gastric volume (which frequently they cannot), they will develop diarrhoea as the result of high osmotic load. Malabsorption of the nutrients may result. If they absorb everything, they may develop metabolic complications of dietary protein and carbohydrate excess. In the past papers, the examiners approach this topic only once - in Question 28 from the second paper of 2007. It has not been seen since, and may never appear again.

An excellent article (Klein et al, 1998) is available which summarises the metabolic derangements which beset the grossly overnourished ICU patient. It is the primary reference for this chapter. Those with institutional access should be able to get hold of it, and share it with those without such access. I will make an attempt to summarise its most important points, to benefit the ICU trainees who don't have powerful friends with paid journal access. One should keep in mind that this article was written in 1998, and when it comes to macronutrient proportions and particularly protein supplementation, the authors say all sorts of wacky things (like “nobody benefits from more than 1.2g/kg/day of protein” for example).

Other good resources include:

- Montejo et al, 1999 (more metabolic complications)
- Smith et al, 1990 (diarrhoea)
- Griffiths et al, 2007 (excess of enteral nutrition)

In short, the problem of overfeeding arises from the fact that no mammalian organism has ever evolved in the context of continuous nutrient delivery and immobility. We simply haven't the metabolic capacity to cope with such a situation; all of our nutrient storage and fuel use mechanisms developed in the presence of the constant threat of famine. The ability to rapidly absorb, store and then gradually release nutrients has been much more useful in the environment of our ancestors.

Mechanical complications of hyperalimentation

Intolerance of nasogastric volume

This is a brutally mechanical complication of excess nasogastric feed volume. The precise maximum volume is not easy to estimate because every stomach is different and might behave differently under different circumstances.

On top of the hourly nasogastric feed volume the stomach itself also secretes a substantial amount of mucus and juices (around 100-150ml/hr) which contributes to the volume. Liquid empties faster than solid (Collins et al, 1983) and emptying in general follows an exponential pattern. If any given volume is given, approximately half of it will be emptied in the first hour. In addition to volume calorie content of the meal also plays a role, with low-calorie meals emptying faster than high-calorie meals. In the context of drinking only pure water, and on an empty stomach, the theoretical maximal rate of stomach emptying is approximately 2.5L/hr - but this is in the context of exercising athletes.

Generally speaking, in the critically ill the gastric emptying rate is reduced. Heyland et al (1996) performed an experiment where stomach emptying was measured by the rate of paracetamol absorption (for a number of reasons paracetamol is a favourite marker substance in measuring stomach emptying). These experiments demonstrated that in critically ill patients the gastric emptying rate is halved in...
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comparison to controls. Ergo, feeding these patients an excessive dose of nasogastric nutrients will result in gastric distension, vomiting, and potentially aspiration. Montejano et al (1999) found that high gastric residuals were reported in the course of 39% of NG-fed patient encounters.

Osmotic diarrhoea

Most nutrient mixtures are isonmolar. Some, intentionally, are hypertonic. For example, formulae for renal patients tend to be more concentrated so as to reduce the total fluid input. The consequence of this is an osmotic diarrhoea. Smith et al (1990) found three major contributors to diarrhoea associated with NG feeding:

- Higher rates of infusion
- Greater tube-feeding osmolality
- Change of formula

Interestingly, antibiotic use and serum albumin levels were not predictors. Though everybody knows about the association of NG feeds and diarrhoea, Montejano et al (1999) found the incidence of constipation and diarrhoea were approximately similar (15%) in their prospective cohort of NG-fed patients.

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Consequences of dietary protein excess

Azotaemia

The key issue here is protein. Already in your critical illness induced hypercatabolic state the hormones and the cytokines have mobilised metabolic fuel substrates, and amino acids are awash in the circulation. The liver and kidneys are struggling to maintain some sanity by rapidly excreting these molecules by the conventional mechanism, which turns them into urea. Urea tends to rise in critical illness as a result of this activity.

Now, into this equation, imagine adding a supraphysiological volume of supplemental protein. The amino acid content of portal blood will rise, and the urea cycle will struggle valiantly against this tide. Usually, with a normal liver, the cycle will win, and a massive increase in urea will be the result. Obviously having a ridiculously high urea is not good for anybody, and this is a strong argument against protein overfeeding even in the context of normal renal and hepatic function.

Hyperammonaemia

If one has a defective urea cycle, or one’s liver has been damaged by many years of overindulgence, one may find oneself rather more confused by the administration of a large protein load. Ammonia will be generated by the deamination of the excess amino acids, and it will accumulate, giving rise to hepatic encephalopathy.

Hypertonic dehydration

Even in the presence of a normal liver and a working urea cycle, an abnormally high protein load is a disadvantage. The uremia which results may of a low level, and thus may not have any adverse clinical effects (i.e. the patient is neither encephalopathic nor experiencing any bleeding from their platele dysfunction).

However, the urea still manages to be somehow harmful. The additional urea load demands that a certain volume of water is passed, so as to remove a sufficient volume of nitrogenous wastes.

This volume of water which undergoes this “obligate” excretion depends largely on one’s ability to concentrate water. Thus, patients who are poor water concentrators (eg. hypoosmolar patients, patients on lithium, patients on diuretics, and the elderly) will end up excreting much more water with their urea, and will become dehydrated and hypertonic. In addition to this process, there is a dehydrating contribution from the osmotic diuresis due to hyperglycaemia.

This process of gradually drying out due to uremia was well known in the 1950s and 1960s, under the name “Tube Feeding Syndrome”. The modern experience of this disturbance remains largely unchanged. The key appears to be the correct estimation of total daily water needs, and the replacement of an adequate amount of water, as well as (obviously) not feeding the patient to excess.

Metabolic acidosis

Historically, this has been associated with hydrochloric acid being a silent preservative in TPN, with lysine hydrochloride contained in early versions of TPN, or with excess casein hydrolysate.

The cause of the metabolic acidosis in these cases has been the complete metabolism of the amino acids concerned, and the complete lack of metabolism of the chloride. The consequence is a decrease of the strong ion difference, and thus a normal anion gap metabolic acidosis (this is discussed in greater detail elsewhere).

These days it is difficult to cause a metabolic acidosis due to overfeeding in the critically ill patient, because TPN no longer contains quite so much chloride. However, there has been at least one report of metabolic acidosis due to high protein enteral nutrition; a reference to it can be found in my primary source. This report is unpublished - I have no way to look at the abstract. It was presented in 1997 during a conference. It is very difficult to generate a good valid mechanism for metabolic acidosis due to enteral nutrition, and I would love to see what they came up with.
Consequences of dietary fat excess

Fat Overload Syndrome

Rather than a complication of excess enteral feeding, this tends to happen when one administers an excessively large amount of lipid-containing TPN emulsion to their patient.

- Acute respiratory failure, manifesting as pulmonary oedema
- Coagulopathy, manifesting as abnormal platelet function, as well as proper DlC
- Headache, fever and jaundice
- Hepatomegaly

These are all good reasons why you should never bolus TPN. The critical fat dose per day seems to be somewhere above 2.5g/kg/day, and somewhere below 4.0g/kg/day (though much of our data regarding this complication comes from TPN-dependent children with short gut syndromes, which are hardly a representative sample).

Hyperlipidemia, particularly high triglycerides

The overadministration of oral or NG carbohydrates has been shown to result in high serum triglyceride levels. This is thought to be the consequence of hepatic de novo synthesis of fatty acids, which are then packaged into triglycerides and VLDLs. The abovementioned saturable mechanisms of lipoprotein synthesis are responsible for keeping the liver from becoming massively swollen with stored fats. On top of this, the actual lipids in the feeds (and especially in TPN) are contributing to serum lipid level elevation.

And what, might one ask, are the acute consequences of extreme hypertriglyceridaemia?

Well,

- Hypercoagulability and thrombosis
- Pancreatitis
- Chylo micronemia syndrome:
  - Abdominal pain
  - Hepatomegaly (probably responsible for abdominal pain)
  - Nausea
  - Eruption of skin and tendon xanthoma
  - Milky appearance of blood plasma
  - Again, pancreatitis

References
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