Bowel cleansing in patients with chronic renal failure – an often overlooked hazard

Sir,

Bowel cleansing is a common almost routine procedure that is used in ambulatory as well as in hospitalized patients in preparation for abdominal surgery, radiological examination of the lower gastrointestinal tract and colonoscopy. Bowel cleansing is also commonly used as a ‘relieving’ measure in elderly patients with constipation and faecal impaction.

Many of the preparations used for bowel cleansing, whether oral or per enema, contain large amounts of phosphate. When ingested orally, two thirds of the phosphate that is ingested is normally absorbed in the gastrointestinal tract, whereas one third is excreted in the faeces. Bowel cleansing thus constitutes an acute phosphate load. This phosphate load is normally dealt with predominantly by the kidneys. In patients with normal renal function, the kidneys excrete the excess phosphate and maintain normophosphataemia. When renal function is impaired, however, the phosphate load becomes clinically significant and life-threatening hyperphosphataemia can develop.

A number of reports have appeared during the past decade describing severe life-threatening complications as result of the use of phosphate containing bowel cleansers in patients with renal insufficiency [1–5]. Unfortunately, these reports have failed to achieve the necessary awareness of health-care providers insofar as the danger involved in bowel cleansing. One of the reasons may be that bowel cleansing is usually carried out by nursing or ancillary medical staff who are unaware of the hazards involved when renal function is impaired.

We recently came across three additional cases of patients with impaired renal function in whom an oral phosphate containing bowel cleansing preparation was used in preparation for colonoscopy in a hospital setting. Severe life-threatening hyperphosphataemia developed, resulting in the death of two of the three patients.

Case 1

A 74-year-old female patient was suspected to have gastrointestinal blood loss. Colonoscopy was scheduled. In preparation for the examination, the patient was admitted to the hospital and underwent bowel cleansing. A 45 ml dose of an oral phosphate containing solution (Soffodex) was administered on the evening prior to and 45 ml on the morning of the examination. Colonoscopy was uneventful. The patient felt well during and after the procedure and was discharged from the hospital in good general condition one day later. A routine blood test taken prior to her discharge, the results of which were received by the medical staff only one day after discharge, indicated that serum phosphate level had risen to 13 mg/dl. The patient was recalled but returned only 3 days later at which time serum phosphate levels had come down to 6.3 mg/dl. She reported feeling well and not having developed any symptoms over the period between discharge and follow-up. A review of the patient’s records revealed that she had been diagnosed earlier with tubulo-interstitial nephritis and had a creatinine clearance of 25 ml/min (stage 4 CKD).

Case 2

An 84-year-old female was admitted for investigation of anaemia with a haemoglobin level of 4.1 g/dl. Anaemia was partially corrected with blood transfusions. The patient was scheduled for colonoscopy for which she was prepared with a one time oral dose of 90 ml Soffodex at noontime. Colonoscopy, which had been scheduled for the next day, was postponed for technical reasons. Two days after administration of Soffodex, the patient became confused and began vomiting. She became tachypneic and started having generalized seizures. Blood tests revealed severe hyperphosphataemia of 14 mg/dl. Additional abnormal biochemistry included hypocalcaemia of 3.6 mg/dl, hypokalaemia of 3.2 meq/l, hypomagnesaemia 1.4 mg/dl and metabolic acidosis with pH 7.15. Treatment with intravenous saline, calcium gluconate, and bicarbonate was initiated. The patient developed bradycardia and hypotension. Body temperature rose to 39°C. The patient died 60 h after administration of the bowel cleansing solution. A review of her medical records revealed that prior to admission she had been diagnosed as having chronic renal failure with a plasma creatinine level of 2.4 mg% (stage 4 CKD).

Case 3

A 73-year-old male patient was admitted because of severe constipation for five days and signs of paralytic ileus. Colonoscopy was scheduled to rule out a tumour in the colon. Initial bowel cleansing with 45 ml Soffodex was ineffective. A second dose of 90 ml Soffodex was administered, still with unsatisfactory results. A third 45 ml dose was given. During nighttime, the patient became confused. Blood pressure dropped from 168/95 to 80/50 mmHg, pulmonary oedema developed and urine output diminished. Blood tests revealed serum phosphate 13.5 mg%. The patient was transferred to the intensive care unit with a diagnosis of Soffodex-induced acute hyperphosphataemia. Continuous veno-venous haemodiafiltration was initiated and plasma phosphate levels gradually returned to normal. The patient went nevertheless into deep shock, developed apnoea and died. Post mortem revealed multiple foci of myocardial necrosis. Prior to admission, the patients had had a creatinine clearance of 12 ml/min (stage 5 CKD).

The three cases described demonstrate the potential life-threatening hazards of phosphate-containing bowel cleansers in patients with impaired renal function. Although these are not the first reports of such cases, the impact of previous reports has apparently not been sufficient to increase the level of awareness amongst medical personnel to this important side effect of the so commonly used bowel cleansing procedure. This report comes to increase further the awareness of health-care givers to the hazard of bowel cleansing in patients with impaired renal function.

Why is bowel cleansing hazardous? Sodium phosphate is an important component of the commonly used bowel cleansers. Bowel cleansing increases the phosphate load nearly 50-fold of the recommended daily intake. The kidney, which can normally deal successfully with this load, is unable to excrete the excess load in face of renal insufficiency. As a result, hyperphosphataemia develops. Depending on the degree of hyperphosphataemia and of kidney function, it can be transient and clinically insignificant and go unnoticed as occurred in one of our patients, or it can
be catastrophic, as demonstrated by two of the three cases presented in this report in which the outcome was fatal.

What is the magnitude of the problem? How often does hyperphosphataemia develop after bowel cleansing with a phosphate-containing preparation? Hyperphosphataemia can be only transient [6], with no concomitant decrease in serum calcium and thus go unnoticed. It apparently becomes more common and clinically significant when kidney function decreases [7], when gut motility is poor [8] and with advancing age [9]. Once clinically significant hyperphosphataemia has developed, the only therapeutic measure that is available beyond resuscitative measures and cardiovascular support is mechanical removal of the excess phosphate with dialysis.

In one of our cases which was treated with haemofiltration, normalization of serum phosphorus levels was achieved, but the patient did not recover.

What conclusions can be drawn and what recommendations can be derived from the three reported cases? The most important conclusion is that phosphate-containing bowel cleansers can be hazardous in patients with impaired kidney function and must be used judiciously and with extreme caution while closely monitoring serum phosphate levels when creatinine clearance is below 60 ml/min. Such compounds are best avoided altogether when creatinine clearance is below 30 ml/min. They are certainly contraindicated when creatinine clearance is below 15 ml/min. Beyond the level of renal function, additional factors that must be considered when deciding on the composition of a bowel-cleansing preparation include gut motility and age. If severe hyperphosphataemia develops in spite of all the necessary precautionary measures, the patients are best treated in an intensive care setup with continuous veno-venous haemofiltration.

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In vivo quantification of central venous catheter leak

Sir,

In the June issue of Nephrology Dialysis Transplantation, we reported the results of an in vitro experiment aimed at quantifying the heparin leak that occurs as a result of central venous catheter heparin lock [1]. Our results showed an early catheter leak of ~0.7 ml during the first 30 s after performing a catheter lock. Continuous late catheter leak was substantial in this experiment (75% within 30 min); however, its relevance to in vivo conditions was unknown. We would like to report now briefly the results of an in vivo experiment in a single patient who agreed to have a modified lung scan to confirm a possible catheter leak.

A routine nuclear medicine lung perfusion scan is usually performed following intravenous injection of Tc99m-labelled albumin macro-aggregates (MAAs). These MAA particles become ‘trapped’ in the terminal alveolar arterioles of the lungs since the particles are in the range of 15 μm in diameter and capillaries are only in the range of 7 μm in diameter. Therefore, the radioactivity distribution recorded by a gamma camera over the chest area represents lung perfusion.

With the patient positioned under the camera, as if for an anterior view of a lung scan, we filled the dialysis catheter lumens with MAAs diluted in a 4.5 ml solution (2.2 ml in the arterial lumen and 2.3 ml in the venous lumen). Images were taken every minute for 30 min (Figure 1). Once this process was completed, the MAAs in both catheter lumens were flushed and a final imaging of the lungs was performed. The rationale for this procedure was that any leak from the catheter would be detected by an accumulation of radioactivity in the lungs. In addition, quantification of the degree of lung uptake would allow for a relative quantification of any leak. A time/activity curve (Figure 2) of the lung area was used to quantify the relative catheter leak over time. The value at the end of the curve, following a rapid rise obtained after complete flushing of the catheter, represents the entire lock volume (100% value) from which the percentage of the leak can be calculated at any time point.

The results show an almost 20% leak that occurred initially without any detectable continuous leakage over the following 30 min (Figure 2). The slight continuous decrease of the curve, seen prior to the flushing, is probably due to the physical radioactive decay of the tracer (half-life of 6 h) as well as slow disintegration of MAAs. In our case, the 20% leak represents 0.9 ml. The results of the present experiment confirm the magnitude of initial leak that we quantified in our previous in vitro study. However, in contrast to our in vitro experiment, this single in vivo experiment showed no evidence of