Case Report

Acute Gout Precipitated by Total Parenteral Nutrition

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ABSTRACT. Hypouricemia is seen in a variety of clinical situations. Although precipitation of gout is well known following initiation of uricosuric therapy, it has been reported rarely following the institution of total parenteral nutrition (TPN), despite its known uricosuric effect. We describe a patient who developed polyarticular gout on 2 occasions after a sudden decline in serum uric acid after initiation of purine-free TPN. Potential etiologies include increased urate clearance due to the infusion of glycine or amino acids. Monitoring of serum uric acid concentrations in patients with a history of gout may help predict a gout attack. Prophylactic treatment or alternative TPN formulations may be indicated.

DISCUSSION

TPN associated hypouricemia has been reported by a variety of investigators. Koretz noted in a small retrospective study that the serum uric acid decreased by a mean of 3 mg/dl and postulated a lower rate of uric acid production as a likely mechanism. Al-Jurf et al described a marked reduction in serum uric acid of 92% of 26 patients during the course of TPN, with the maximum reduction occurring during the first 3 days. Al-Jurf et al described a 3- to 4-fold increase in uric acid clearance. Morichau-Beauchant, et al cited similar marked declines in serum uric acid due to increased urate clearance despite substituting fructose for glucose in some patients and switching to purine-free elemental enteral nutrition. In 1987, Derus, et al described a case of crystal-proven podagra that developed after 3 days of TPN. Urate reabsorption was felt to be inhibited, either by the infusion of glycine or amino acids. In the references cited above, as in our case, serum uric acid normalized within several days of discontinuation of TPN.

A literature review disclosed a few potential mechanisms of TPN related hypouricemia. The most accepted include increases in urinary urate excretion, likely secondary to decreased tubular reabsorption or decreased production of uric acid due to initiation of a purine-free TPN solution. Protein infusions of 0.1 g/kg/h have been noted to be potentially uricosuric, while solutions rich in...
Dextrose may increase urate excretion due to an osmotic effect. Other potential causes of uricosuria such as glycine-rich amino acid solutions (17–25 g/day)\(^6\), high dose ascorbic acid (4 g/day), and elevated blood sugars\(^6\) have been reported as a possible etiology, but not substantiated. Extracellular fluid expansion possibly due to stimulation of antidiuretic hormone has been postulated\(^5-7\).

Evaluation of the TPN formula in this case found the patient was receiving an amino acid infusion of 0.75–1.5 g/kg/day and dextrose infusion rate up to 2.85 mg/kg/min. In each course of TPN, the patient was started at a lower rate and titrated to the maximum rates noted. The glycine content of each day’s TPN solution was roughly 0.5 g/day, much lower than the amounts noted to be uricosuric. Unfortunately, we did not measure urinary uric acid clearance or fractional excretion of urate to assess for increased urinary clearance. Standard injectable multivitamins were used daily and contained only 100 mg ascorbic acid.

For patients with or at risk for gout, feeding with TPN becomes a difficult issue. There are no current guidelines for formulating TPN solutions in this population and very few data exist. There is no indication that using lower doses of amino acid or dextrose will prevent hypouricemia. Widhalm, et al\(^8\) showed that a lipid emulsion based on soy oil (20%) had no effect on uric acid concentrations, and therefore it may be feasible to use lipid-rich TPN solutions in this population.

Routine daily monitoring of uric acid concentrations in addition to standard biochemical measurements in patients with gout may be helpful to alert clinicians to declining serum levels and potential exacerbations of gout in susceptible patients. It is feasible that this patient’s serum uric acid diminished as a result of an acute episode of gout, although no other definite risk factors were temporally identified. The role of prophylactic or therapeutic corticosteroids, colchicine, or nonsteroidal antiinflammatory drugs and of their route of administration is less clear. The potential toxicities of preventive therapy need to be considered carefully.

REFERENCES