



PIC QUESTION OF THE WEEK: 02/28/05

Q: A nurse practitioner called regarding a 50-year old man with hypertension, non-insulin dependent diabetes mellitus, hypertriglyceridemia, and GERD who recently developed his second attack of gouty arthritis. Current medications consist of furosemide, Toprol XL, Cozaar, metformin, omeprazole, niacin, and ASA (325 mg daily). The caller questioned whether any of the medications could be contributing to these gouty attacks.

A: Gouty arthritis is a disorder of purine metabolism usually characterized by elevated uric acid levels and severe acute arthritis resulting from deposition of monosodium urate crystals in synovial fluid. The subsequent inflammatory process results in severe pain, swelling, redness, and tenderness of the affected joint. Although the first metatarsophalangeal joint (“great toe”) is involved in 50% of cases, the process may also affect the knee, ankle, wrist, Achilles tendon sites, etc. This patient’s uric acid value was 10 mg/dL (normal plasma saturation is ~ 7 mg/dL). Many drugs decrease renal elimination of uric acid and can result in hyperuricemia. These include thiazide and loop diuretics, low-dose aspirin (≤ 2 g /day), cyclosporine, niacin, levodopa, and some anti-tuberculars. The prescriber deemed it necessary that furosemide be maintained. It would be important to determine this patient’s fluid status because dehydration can predispose to acute gouty arthritis. Although the quantitative effect of low-dose aspirin on this patient’s uric acid level is difficult to determine, it could be a causal factor. Substitution with clopidogrel may be warranted. Lastly, niacin may produce hyperuricemia and hyperglycemia. A fibrate such as Tricor may be preferable to niacin. The other currently prescribed medications do not result in hyperuricemia. If urate lowering is begun with allopurinol, treatment should be withheld until a few days after resolution of the acute attack. Low-dose colchicine should probably be prescribed to reduce the potential for another gouty attack that could be induced upon addition of allopurinol.

References:

- Lee M. Basic skills in interpreting laboratory data. 3rd ed. Bethesda: American Society of Health-System Pharmacists; 2004.
- Root KT, Agudelo CA. Gout. JAMA 2003; 289: 2857-60.

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