

confuse the clinical picture unless the associated electrocardiographic changes and other evidence of systemic gas embolism are recognized.

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Analgesic Nephropathy, A Form of Chronic Renal Disease

To the Editor:—I read with great interest the case report by Sivaran concerning a 38-yr-old who underwent lumbar discectomy and subsequently developed acute tubular necrosis (ANESTHESIOLOGY, 1997; 86:1390-2). The authors imply that preoperative intake of nonsteroidal antiinflammatory drugs (NSAIDs) was the culprit in this otherwise healthy patient's perioperative renal dysfunction.

However, the authors have failed to notice the patients admitted intake of acetaminophen (300 mg-30 mg) tablets every 6 h as another possibility. A form of chronic renal disease called *analgesic nephropathy* can occur in patients who ingest large quantities of acetaminophen and prostaglandin inhibitors. It is believed the minimal requirements for the development of renal damage are 2-3 kg of acetaminophen taken over a 3-yr period.

Pathologically, the acetaminophen injures cells by covalent binding and oxidative damage. The NSAIDs may then potentiate the effect of the acetaminophen by inhibiting the vasodilatory effects of prostaglandin, thus predisposing the papilla to ischemia. Thus, the papillary necrosis may be a result of the combination of the direct toxic effects of acetaminophen and of the ischemic injury to tubular cells and vessels.¹

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In Reply:—We were aware of analgesic nephropathy occurring in patients ingesting large quantities of acetaminophen alone or in combination with NSAIDs. The first reference by Clive and Stoff in our case report has a detailed description of analgesic nephropathy. Analgesic nephropathy is a *chronic* disease that causes tubulointerstitial damage and dysfunction that presents as leukocyturia, hematuria, and inability to concentrate urine.¹ As mentioned in the case report, our patient exhibited none of these, and result of preoperative urinalysis was normal. Our patient suffered acute renal failure, and postoperative urinalysis was characteristic of acute tubular necrosis from

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Reference

1. Dullye K, et al: Laser treatment of endobronchial lesions. ANESTHESIOLOGY 1997; 86:1387-90

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Luckily for this particular patient, after appropriate treatment and cessation of the offending drugs, his blood urea nitrogen and serum creatinine levels returned to normal. However, if his renal failure actually was a result of analgesic nephropathy, it is believed that he is at increased risk for the development of transitional papillary carcinoma of the renal pelvis.²

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References

1. Robbins Pathologic Basis of Disease, 4th ed, Edited by Cotran, Kumar, Robbins, p 1054-5
2. Bergtsson V, et al: Malignancies of the Urinary Tract and Their Relation to Analgesic Abuse. Kidney Int. 13:107 1978

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which full recovery took place. Therefore, we did not consider analgesic nephropathy as a possibility.

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