Editorial

The Mechanism of Analgesic Nephropathy

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INTRODUCTION

Analgesic nephropathy is kidney damage caused by pain relievers including aspirin, bucetin, phenacetin, and paracetamol. Excessive use of these drugs, especially combinations that include phenacetin, can cause harm.

Renal papillary necrosis and chronic interstitial nephritis are two kidney injuries caused by analgesics. They tend to be caused by reduced blood flow to the kidney, rapid antioxidant intake, and oxidative damage to the kidney. Chronic kidney failure, irregular urinalysis results, high blood pressure, and anaemia may all be symptoms of kidney injury. Analgesic nephropathy can lead to end-stage kidney disease in a small percentage of people.

In parts of Europe, Australia, and the United States, analgesic nephropathy was once a common cause of kidney injury and end-stage kidney disease. Since the use of phenacetin fell in the 1970s and 1980s, its incidence has dropped dramatically in most areas. Headache, anaemia, elevated blood pressure (hypertension), and white blood cells in the urine are all common symptoms of analgesic nephropathy (leucocyturia, pyuria). Protein can be found in the urine of people who have analgesic nephropathy.

Pyelonephritis and end-stage kidney failure are two complications of analgesic nephropathy. Recurrent urinary tract infection and persistently elevated blood pressure are also risk factors for a poor prognosis. Analgesic nephropathy tends to raise the risk of developing urinary system cancers. Capillary sclerosis, or the scarring of tiny blood vessels, is the first symptom of analgesic nephropathy. Capillary sclerosis, which affects the renal pelvis, ureter, and capillaries supplying the nephrons, is thought to cause renal papillary necrosis and, as a result, chronic interstitial nephritis.

The exact mechanism by which phenacetin and other analgesics do this damage is unknown. Analgesic nephropathy is believed to be caused by a combination of the kidney toxicity of NSAIDs and the antipyretics phenacetin and paracetamol.

Analgesic Nephropathy Study

In 2000, a committee of researchers concluded that there was insufficient evidence to indicate that non-phenacetin analgesics are associated with analgesic nephropathy on their own. Adequate blood flow to the kidney is needed for proper kidney function. Kidney blood flow is a dynamic, closely regulated mechanism that is influenced by a variety of hormones and other small molecules including prostaglandins. Under normal circumstances, the kidney's production of prostaglandin E2 (PGE2) is needed to maintain sufficient blood flow to the kidney.

Cyclooxygenases are inhibited by aspirin and other nonsteroidal anti-inflammatory drugs (NSAIDs). This inhibition causes a drop of PGE2 concentration in the kidney, resulting in a reduction in blood flow. The deeper structures of the kidney are most vulnerable to reduced blood flow because blood flow to the kidney first enters the renal cortex (outside) and then the renal medulla (inside).

As a result, prostaglandin synthesis is particularly important for maintaining sufficient blood flow in the kidney's innermost structures, known as the renal papillae. As a result, inhibiting cyclooxygenases damages the renal papillae more selectively, raising the risk of renal papillary necrosis. In healthy dogs given an aesthesia, NSAIDs had no negative effects on renal function. Most healthy kidneys have enough physiologic reserve to compensate for the blood flow reduction caused by NSAIDs. Those who are exposed to additional injury from phenacetin or paracetamol, on the other hand, can develop analgesic nephropathy.

The above clinical results, in tandem with inappropriate analgesic use, have historically been used to make a diagnosis. Before clinical proof of analgesic nephropathy appears, it is estimated that between 2 and 3 kg of phenacetin or aspirin must be ingested. Analgesic nephropathy can be verified with relative accuracy using computed tomography (CT) imaging without contrast after it has been suspected. The presence of papillary calcifications on CT imaging was found to be 92 percent sensitive and 100 percent specific for the diagnosis of analgesic nephropathy in one study.

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