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RESEARCH LETTER

COULD WE PREVENT CHRONIC KIDNEY DISEASE AND ACUTE SEVERE ACUTE KIDNEY INJURY DUE TO NSAID?

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Two children aged 14 months and 18 months developed acute tubulointerstitial nephritis (AIN) after taking 3 doses of Ibuprofen (7 mg/kg/dose) and 4 doses of Mefenamic acid (5 mg/kg/dose) respectively needing peritoneal dialysis and steroids for complete recovery. They had all classical features of AIN in urine tests including urinary beta 2 microglobulin that was elevated to 2230 and 11000 ng/ml (normal 0 to 300 ng/ml) respectively. A 16-year-old girl also developed AIN secondary to Ibuprofen that she had been taking for fever for the past few years intermittently. She was asymptomatic and her serum creatinine was elevated at 3.3 mg/dl (eGFR 25 ml/min). The urinary beta 2 microglobulin was elevated at 850 ng/ml (tested on day 10 after the first blood test). The creatinine settled to 1.1 mg/dl (eGFR 65 ml/min) but remained stuck at that level. She needed Prednisolone and recovered completely. Autoimmune and other work-up for all 3 patients was normal.

AIN is a well-known entity but what is not well known is that it can be asymptomatic though it has been described before[1], noted in an asymptomatic twin. NSAIDs cause kidney damage by various methods like afferent glomerular constriction, cyclooxygenase effect that are dose dependent. But they also cause AIN that is idiosyncratic in nature.[2,3]

If asymptomatic, it will never be picked up unless a blood test is done for some other reason as described above. While it is self-limiting in nature, if it occurs secondary to NSAID that is being taken intermittently for fever (3 to 4 times a year), it is very likely that the inflammation will become chronic at some stage and the creatinine will not settle down to normal and may need steroids as in the older girl above. It is known that steroids assist recovery of NSAID induced AIN.[4] If undiagnosed, these children would then be at risk of developing CKD/ ESKD decades later if the creatinine remains mildly elevated. Similarly, it is very likely that the 2 other cases had received NSAID earlier (as it was discovered later in the first case). This would suggest that even severe AKI due to AIN secondary to NSAID may not be always seen after the first dose.

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What is the way out? Banning NSAID is self-defeating and easier said than done. They do have their uses when used appropriately. I would propose a simpler solution.

If a child has a urinary beta 2 microglobulin done 24 to 48 hours after a dose of any NSAID, it would help predict the possibility of problems. If the level is 300 or more, it is clearly AIN, but if the level is more than 200 ng/ml, it probably indicates that the NSAID has caused tubular inflammation. In such cases, NSAIDs should be avoided for life and if unavoidable, used with strict monitoring. If the level is less than 200, one can continue using NSAIDs judiciously. Ideally it should be the first dose that the child takes in life but any dose should be fine. And it would need to be done only once. A fresh urine sample that is not acidic in pH should be sent for examination. Children with UTIs and previously known renal dysfunction can be excluded.

It will be a cheap, simple and easy way to detect possibility of CKD in the long term or severe AKI in the short term. Hopefully it will help to reduce the CKD burden not only in India but the entire world as nearly 15 to 30 percent of adults with CKD have no identifiable cause and are lumped together as chronic tubulointerstitial disease. It is very likely that NSAID induced asymptomatic AIN may be playing a part in quite a few of these patients.

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