Acute Effect of Dipyridamole on Urinary Prostaglandin Excretion

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Dear Sir,

We were very interested in the paper by de Jong et al. [1] in which they suggested that the acute antiproteinuric effect of dipyridamole is due to an efferent vasodilation with a fall in intraglomerular capillary pressure. Among hypothetical mechanisms of intrarenal action of dipyridamole, the increase of prostacyclin biosynthesis has been discussed.

We studied the acute effect of dipyridamole 20 mg i.v. on 1.5-hour urinary prostaglandins (PGE₂, PGF_{2α}) excretion and diuresis in 18 patients with biopsy-proven glomerulonephritis. The patients had proteinuria of 1.1 ± 0.75 g/day (mean $\pm$ SD), a mean blood pressure of 117.6 ± 12.6 mm Hg, and a plasma creatinine concentration of 127 ± 29 μg/l. They were on a diet containing 85–120 mmol Na/ day and did not receive any drugs at least 5 days before investigation. At the beginning of the first and second periods of urine collection the patients drank 200 ml of water.

Urinary PGE₂ and PGF_{2α} were measured using 'clinical assays' PGF_{2α} RIA kits after amberlite XAD-2 column concentration, ethylacetate extraction and silicic acid column chromatography with registration of the percentage of the substance at the output. Standard and unknown samples containing PGE₂ were processed with natriumborhydrid for conversion of PGE₂ into PGF_{2α}. After dipyridamole infusion PG excretion increased in 15 out of 18 patients (table 1).

The rate of the increase of PG excretion after dipyridamole was positively correlated with the increase of diuresis ($r = 0.68$ and 0.48 ; $p < 0.05$) and did not correlate with blood pressure, plasma creatinine concentration and glomerular filtration rate which were measured before the investigation.

Table 1. Effect of dipyridamole infusion 20 mg on PG excretion and diuresis in 18 patients with chronic glomerulonephritis (mean $\pm$ SD)

We suggest that dipyridamole had a direct stimulating action on production of PG in the kidneys. The stimulation of renal PG synthesis enhances the diuresis; it may also determine, at least partly, the changes in renal hemodynamics responsible for antiproteinuric effect of dipyridamole in patients with chronic glomerulonephritis. But, it is impossible to exclude that the increase in PG excretion will be the consequence of urine flow rise and simple washing out of PG from the kidneys.

The direct stimulating effect of dipyridamole on renal prostaglandin synthesis merits further investigation.

References

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