Dopamine-Furosemide in Oliguric Acute Renal Failure

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Dear Sir,

Lindner [1] has recently stressed the effectiveness of a combined dopamine and furosemide treatment in early oliguric acute renal failure. The question raised whether, as previously suggested [2,3], dopamine alone too may induce a diuretic response in oliguric states. We would like, therefore, to report 1 case in which the time relationship between therapeutic approach and the diuresis seems to support the synergistic effect of dopamine with furosemide.

The patient, a 54-year-old man, was hospitalized for cholecystectomy. The nephrologist was consulted on the 4th day following surgery, 36 h after the onset of oliguria. Urine flow rate was 10–15 ml/h; serum creatinine was 3.2 mg/dl. Prerenal azotemia was excluded: the patient appeared well hydrated and, moreover, failed to respond to ECV expansion (normal saline, 1,500 ml over 2 h); no hypotension, congestive heart failure, cardiac arrhythmias, or other known causes of renal hypoperfusion, were present. The diagnosis of acute tubular necrosis was made; urinary indices were compatible with it [4]. Accordingly to Henderson et al. [2], low-dose dopamine intravenous infusion (1 µg/kg/min) was started. No change in urinary output was observed within the following 12-hour period. Serum creatinine rose to 3.7 mg/dl. An intravenous bolus of furosemide (200 mg) was then given and repeated every 8 h, while dopamine infusion was going on. Urine flow rate rose immediately to 70 ml/h at 1 h, 95 ml/h at 6 h, and 105 ml/h 24 h after the onset of combined dopamine-furosemide treatment. When both agents were discontinued, 48 h later, daily urine volume was 2,800 ml. As far as renal function is concerned, it remained stable throughout the treatment, at the end of which serum creatinine was 4.0 mg/dl. Final outcome was a complete uneventful recovery.

Dopamine alone, therefore, was ineffective in inducing a diuretic response in this case; on the other hand, a combined dopamine-furosemide treatment promptly converted oliguric acute renal failure to a nonoliguric state. Obviously, we cannot rule out a diuretic response to furosemide per se in our patient; this possibility, however, is unlikely [5]. In conclusion we agree fully with the suggestive hypothesis proposed by Lindner [1]: subpressor, low-dose dopamine is effective in inducing renal vasodilatation and enhances, through this mechanism, delivery of furosemide to a critical intrarenal locus where the drug exerts diuretic effect.

References

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