Case Report

Massive increase in proteinuria after the introduction of midodrine in an elderly patient—a case report

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Midodrine is an alpha adrenergic agonist advocated for the treatment of orthostatic hypotension [1] including patients with diabetes [2] and renal failure [3], but as it also increases intraglomerular pressures one might expect an increase in proteinuria. We report such a case.

Case report

An 82-year-old woman with a serum creatinine of 1.3 mg/dl and 2.3 g of proteinuria and a creatinine clearance of 27 cc/min was evaluated by the nephrology team. She had a history of congestive heart failure with severe orthostatic hypotension secondary to autonomic dysfunction. Because of her advanced age and severe left ventricular dysfunction, the patient and her family refused a biopsy. She continued to return in 3-month-intervals and was found to have stable renal function with ~2 g of proteinuria a day for 9 months. She then sought consultation with a gerontologist, who unfortunately placed her on midodrine as an attempt to prevent her severe hypotension. On return 3 months later, her blood pressure had increased to 140/80 mmHg with no further orthostatic symptoms, but her proteinuria had increased to 13.3 g/24 h with a resulting drop in her creatinine clearance to 17 cc/min. Withdrawal of the midodrine resulted in the reduction of her proteinuria to 11.094 g/24 h at 1 month while her blood pressure remained 150/80 mmHg and to near baseline proteinuria of 3.7 gm/24 h when her pressure decreased to 90/38 mmHg.

Midodrine is a prodrug, which is converted to desglymidodrine that acts identically to other \(\alpha_1\)-adrenoceptor stimulants, such as phenylephrine or methoxamine. The increase in the generated blood pressure is almost entirely due to an increase in peripheral resistance. Midodrine is known to increase glomerular pressures and has been used therapeutically to reverse endogenous vasodilators that result in low glomerular pressures in hepatorenal syndrome [4]. Although we do not have a tissue diagnosis, an increase in intraglomerular pressures will often worsen proteinuria and renal function regardless of the underlying glomerular pathology [5]. Although the proteinuria of many glomerulopathies can be quite variable, the massive increase and subsequent decrease in proteinuria seemed to correlate with the introduction and then withdrawal of adrenergic stimulation. Increased proteinuria and subsequent loss of function from midodrine has not yet been reported; however, vasoconstriction from all other drugs has been shown to be harmful. Since midodrine and its major metabolite, desglymidodrine are eliminated through the kidney, the effect may be even more pronounced in renal dysfunction. In our patient, the blood pressure and proteinuria remained well over baseline levels for over a month after discontinuing the midodrine. Clinicians should be aware of this potential complication.

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References


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