LETTER TO EDITOR

Nifedipine-Induced Hypotensive Shock

Dear Sir,

A 60-year-old lady was admitted to the hospital with the provisional diagnosis of 'acute pyelonephritis' when she presented with a 2-day history of fever with chills and rigor and left-sided colicky abdominal pain. She was found to suffer from hypertension 4 years earlier but had defaulted treatment. She was febrile (39.5°C) and toxic. Blood pressure (BP) was 180/130 mm Hg and the pulse was 120 beats per minute (bpm) and regular. The left renal punch was positive. Urinalysis revealed numerous pus cells and moderate proteinuria but the subsequent blood and urine cultures were negative. The other routine investigations as well as serial electrocardiograms (ECG) and cardiac enzymes were normal. She was treated with intravenous ceftazidime 1 gm 12-hourly and oral nifedipine 10 mg tds. An hour after taking the first dose of nifedipine, she complained of difficulty in breathing and feeling giddy. BP was 90/60 mmHg and the pulse was 130bpm. She required assisted ventilation when she failed to respond to intravenous fluids, adrenaline followed by dopamine infusion and steroids. Her limbs were moderately oedematous. BP was then unrecordable. She was given the first dose of i.v. calcium gluconate, 1 gm about 10 hours after she became hypotensive. Within 20 minutes her BP picked up to 70 mm Hg systolic. Thereafter, the calcium gluconate was repeated every 30 minutes to an hour depending on the BP and the serum calcium levels. She was also tried on i.v. glucagon but this was stopped when she developed atrial fibrillation. She returned to sinus rhythm on stopping the drug. She required a total of 34 gm of calcium gluconate over 72 hours to stabilise her BP. She was successfully weaned off the ventilator on the fifth day and discharged a week later. Her BP was stabilised with prazosin.

Initially the patient was thought to suffer from septicaemic shock, but when she failed to respond to antibiotics, fluids and inotropic support, it was felt necessary to review the diagnosis. Myocardial infarction was excluded by normal serial ECGS and cardiac enzymes. An adverse reaction to nifedipine was then felt to be a reasonable explanation. The patientís response to calcium gluconate reinforces our impression. In retrospect, the peripheral oedema the patient developed in the ward, a vasodilatory effect of nifedipine[1] as well as the patientís poor response to inotropic support[1]) should have alerted us to the real problem earlier.

Reports on patients who had ingested large doses of nifedipine either alone or in combination with other drugs are well known[1,2]. However only isolated case reports are found in the literature of patients who had developed hypotension secondary to therapeutic doses of nifedipine. Wachter earlier reported on 3 patients who developed symptomatic hypotension following the administration of 20-30 mg of nifedipine at spaced intervals, but, in only one of these patients was the drop in BP severe enough to warrant calcium gluconate and saline infusion[3]. The principles of management of our patient was based on the experience of those who had been treating patients who had taken large doses of nifedipine. Review of the literature, however, does not really explain why a single dose of nifedipine on occasion results in an adverse reaction such as this.

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References

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