Case Report

Digital gangrene due to dopamine infusion-a case report

M.M. Prabhu*, S.M. Prabhu†, P. Mishra‡, S. Palaian‡

* Department of Medicine, Manipal Teaching Hospital, Manipal College of Medical Sciences, Pokhara, Nepal.
† Department of Dermatology and Venereology, Manipal Teaching Hospital, Manipal College of Medical Sciences, Pokhara, Nepal.
‡ Department of Pharmacology, Manipal Teaching Hospital, Manipal College of Medical Sciences, Pokhara, Nepal.

Abstract
Dopamine is a vasopressor agent for the treatment of shock and hypotension in patients who do not respond to plasma volume expansion. Use of dopamine is associated with severe peripheral ischemia, particularly in patients in whom the peripheral circulation is already impaired. We describe a case of digital gangrene due to intravenous dopamine infusion in a 45-year-old man who did not have any predisposing factor. The patient developed digital gangrene within 24 hours of starting intravenous dopamine. Dopamine is one of the commonly used vasopressor agents in the treatment of shock and hypotension and measures should be taken to detect the possibility of the development of gangrene based on the predisposing factor and if gangrene occurs, immediate steps should be taken for management of the reaction.

Key words
Dopamine, gangrene, ischemia, shock

Introduction
Dopamine is an effective pressor agent for the treatment of shock and hypotension in patients who do not respond to plasma volume expansion. Adverse effects of dopamine include tachycardia, palpitations, arrhythmias (ventricular premature beats, extra systoles), nausea, vomiting, nephrotoxicity, polyuria, extravasations etc. One of the major risks during dopamine treatment is severe peripheral ischemia, particularly in patients in whom the peripheral circulation is already impaired. We hereby report a case of digital gangrene due to intravenous dopamine infusion in a male patient who did not have any predisposing factors that are documented in literature.

Case report
A 45-year-old male, non-alcoholic, non-smoker was admitted with history of high grade fever and loose stools for five days, vomiting and breathlessness for two days. He was receiving treatment for enteric fever with injection ciprofloxacin from an outside hospital. Patient was referred to our hospital for increasing breathlessness, altered
sensorium and renal failure. At admission, patient was febrile with hypoxia (oxygen saturation room air 80%) but vitals were stable. Clinical examination showed bilateral crepitations. Investigations at admission showed renal failure (serum creatinine 4.4 mg/dl, blood urea 79 mg/dl), hyperbilirubinemia with elevated liver enzymes i.e. aspartate amino transferase 100 IU/l, alanine aminotransferase 60 IU/l. The total leucocytic count was 8100 cells/mm$^3$, with neutrophilia (88%). Peripheral smear showed toxic granules. Chest X-ray revealed bilateral fluffy infiltrates consistent with acute respiratory distress syndrome (ARDS). Based on these findings, a diagnosis of sepsis with multiorgan dysfunction syndrome was made. Patient was started on injection ceftazidime, cloxacillin, and metronidazole. Blood and urine cultures were sterile. On second day of admission, patient had one episode of generalized seizure with hypotension (systolic pressure 80 mmHg). Patient was started on intravenous dopamine (10 µg/kg/minute) to maintain systolic blood pressure above 100 mmHg and was also put on anticonvulsants (phenytoin, later changed to phenobarbitone). Within 24 hours after starting intravenous dopamine, patient started having bluish discoloration in both upper limbs (Figure 1). Discoloration of limbs was thought to be due to dopamine infusion in peripheral line. Dopamine was continued through central line at a lower dose of 5 µg/kg/minute as patient had persistent hypotension. Discoloration of lower limb digits also started with gangrene formation in both fingers and toes (Figure 2). The patient deteriorated further and was discharged from our hospital against medical advice.

Discussion

Dopamine is an endogenous catecholamine and is the immediate precursor of noradrenaline. It has sympathomimetic action with prominent dopaminergic and beta-1 adrenergic effects at low to moderate doses, and alpha-adrenergic effects at high doses.$^1$ Gangrene of the extremities has occurred when high doses (infusion greater than 20 µg/kg/min) of dopamine were administered for prolonged periods and in patients with
oclusive vascular disease receiving low dose of dopamine. The suggested mechanism of dopamine induced gangrene is the conversion of dopamine in the body to adrenaline which is a known powerful vasoconstrictor. The risk category of patients includes those with history of occlusive vascular disease (e.g. atherosclerosis, arterial embolism, Raynaud’s disease, cold injury, diabetic endarteritis, Buerger’s disease etc.). These patients should be carefully monitored during dopamine therapy for decreased circulation to the extremities. If this occurs, it may be corrected by decreasing the rate of infusion or discontinuing dopamine. However, the potential benefits of continuing dopamine should be weighed against the possible risk of necrosis.

Our patient did not have any predisposing factor for development of gangrene. To reverse ischemia due to dopamine, 10 mg of chlorpromazine IV followed by chlorpromazine infusion of 0.6 mg/minute has been used. Some clinicians recommend IV administration of 5-10 mg of phentolamine (an alpha-blocking agent) if discoloration of extremities occur. Topical glyceryl trinitrate ointment is known to improve capillary blood flow in patients with dopamine-induced digital ischemia. However, in this case the patient was not given IV chlorpromazine as the patient had abnormal liver function tests.

Conclusion

Dopamine is one of the commonly used pressor agents in the treatment of shock and hypotension. Measures should be taken to detect the possibility of the development of gangrene based on the predisposing factors. If it occurs, immediate steps should be taken for management of the reaction.

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References