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Catecholamine refractory, vasopressin responsive, anaphylactic shock - a close differential diagnosis for septic shock in a neutropenic patient

Oncology

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ABSTRACT

We describe a case of catecholamine refractory anaphylactic shock, secondary to the glycopeptide antibiotic teicoplanin mimicking septic shock, which responded to vasopressin in a patient with acute myeloid leukemia (AML) on induction chemotherapy. Teicoplanin injection for suspected gram-positive infection precipitated profound anaphylactic shock in this patient who continued to be hypotensive for 48 hours and did not respond to adrenaline, nor adrenaline or dopamine infusions. Subsequently she was started on vasopressin infusion at low doses which was effective in promptly restoring hemodynamic stability.

We conclude that anaphylaxis can rarely be a cause of hypotension in neutropenic patients. Most episodes of anaphylaxis respond to treatment with single doses of epinephrine, however severe anaphylaxis can be associated with cardiovascular collapse that is difficult to manage. Use of vasopressin in this setting can be life-saving.

KEYWORDS

refractory shock, neutropenia, vasopressin

Introduction:

In a neutropenic patient, the commonest cause for hypotension is sepsis. However even in this patient population, anaphylaxis due to drugs including intravenous antibiotics can be a differential diagnosis for hypotension. Severe anaphylaxis can be associated with cardiovascular collapse that is difficult to manage and does not respond to epinephrine. Recent reports have documented the successful use of vasopressin to treat catecholamine refractory septic and anaphylactic shock.

In this report, we describe a case of catecholamine refractory anaphylactic shock, secondary to the glycopeptide antibiotic teicoplanin mimicking septic shock, which responded to vasopressin, in a patient with AML on induction chemotherapy. We obtained consent from our patient for publishing this report.

Report:

Our patient was a 17 year old girl, with AML M5 admitted for induction chemotherapy. She had a history of allergy to sea-foods. She received chemotherapy with cytosine arabinoside for 7 days and daunorubicin for 3 days. She had received 5 days of teicoplanin for suspected gram positive infection while on chemotherapy which was tolerated well. In the post chemotherapy neutropenic period as her neutrophil counts started recovering, she developed fever while on imipenem-cilastatin. She was re-started on teicoplanin for suspected gram positive infection.

Prior to teicoplanin infusion, a test dose with 0.1 ml of the antibiotic was given intra-dermally which did not evoke any reaction. The patient complained of pruritus and nausea, two minutes after start of infusion and collapsed suddenly. There was an abrupt fall in BP, systolic BP was not recordable and extremities were cold. Peripheral pulses were not felt, but the carotid pulse was palpable. The patient was immediately given i.v. hydrocortisone, chlorpheniramine, i.m. epinephrine and rapid crystalloid infusion. Systolic B.P picked up to 60-70 mm Hg and dopamine and nor-adrenaline infusions were also started. As she was neutropenic, keeping in mind the possibility of septic shock, she also received Inj. Colistin, Inj. Vancomycin and Inj.Voriconazole to cover possible microbes implicated in septic shock. However, her absolute neutrophil count recovered to more than 1000/mm^3 on the day following the acute episode.

Crystalloids were continued and pressor doses were escalated to maximal levels for more than 36 hours but further improvement in B.P. was not obtained. She was then given Inj. Vasopressin 20 U bolus followed by low dose infusion (0.2-0.3 units per min). After starting vasopressin infusion hemodynamic function stabilised quickly and she was off pressor support by the next 24 hours.

Discussion:

In a neutropenic patient, the commonest cause for hypotension is sepsis. However even in this patient population, anaphylaxis due to drugs including intravenous antibiotics can be a differential diagnosis for severe/life-threatening hypotension (1). In our patient the ANC was more than 500/mm^3 at the time of development of shock and since the hemodynamic collapse in this patient was closely temporally related to the administration of the drug teicoplanin, the possibility of septic shock seems unlikely.

Teicoplanin is an infrequent cause of anaphyactoid/allergic reactions unlike vancomycin (the other glycopeptide antibiotic used for the same indication) which frequently causes reactions due to histamine release from basophils and mast cells (2,3). However, Asero et al reported a 41 year old lady who experienced urticaria, angioedema and vomiting approximately 2 minutes after i.v teicoplanin infusion. This was successfully treated with iv corticosteroids and antihistamines without requiring adrenaline (4). This patient had received a ten day course of teicoplanin four months prior, uneventfully. Our patient had also been exposed to teicoplanin, ten days prior without any reactions, showing that hypersensitivity can develop on repeated exposure in patients who have received the drug earlier. In view of the severity of the adverse drug reaction in our patient, due to ethical reasons, skin testing with teicoplanin was not repeated to confirm causality.

Severe anaphylaxis can be associated with cardiovascular collapse that is difficult to manage and does not respond to treatment with epinephrine. Because anaphylaxis is uncommon, unpredictable and may be fatal, a prospective, randomized, controlled trial in humans on the best management is difficult and guidelines are based on theory and anecdotes only. Epinephrine has been widely accepted to be the standard of care to reverse cardiovascular collapse in anaphylaxis. However recent reports have documented the successful use of vasopressin to treat catecholamine refractory septic and anaphylactic shock (5).

Hussain et al (6) reported a 24-year-old woman who developed severe anaphylactic shock at induction of anaesthesia while undergoing laparoscopic cholecystectomy. Circulatory shock was refractory to epinephrine and high doses of the pure alpha-agonist phenylephrine and norepinephrine. Single intravenous dose of two units of vasopressin re-established normal circulation and blood pressure in this patient.

Similarly, Schummer et al (7) reported 6 cases with anaphylactic shock in which epinephrine, nor-epinephrine and high dose steroids were ineffective in restoring circulatory pressures, but low dose vasopressin (2 units bolus and/or low dose infusion) was successful in bringing about hemodynamic stability.

In our patient also, low dose vasopressin infusion was successful in promptly restoring hemodynamic stability. Several mechanisms have been postulated for the role of vasopressin in such situations. In

vasoplegic shock states vasopressin restores vascular tone by (a) activation of V1 receptors that mediates vaso-constriction via Gq protein activation of phospholipase (b) closure of ATP sensitive K+ channels causing cellular hyper-polarisation and resulting in vasodilatation (c) modulation of nitric oxide release and (d) enhancement of adrenergic and other vasoconstrictor drugs. In addition, vasopressin also acts as an anti-inflammatory drug by antagonising the effect of nitric oxide.

Conclusion:

Anaphylaxis due to drugs can rarely cause hypotension in neutropenic patients and be confused with septic shock. Most episodes of anaphylaxis respond to treatment with single doses of epinephrine, however severe anaphylaxis can be associated with cardiovascular collapse that is refractory to catecholamines. In such situations, vasopressin may help to promptly and effectively restore hemodynamic stability before the effects of massive distributive shock lead to severe organ hypo-perfusion and therefore, it should be considered early in the course of treatment for patients with catecholamine refractory shock.

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