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CASE REPORT

A case report on red man syndrome

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ABSTRACT

Vancomycin is a glycopeptide antibiotic used for the treatment of serious infections caused by Gram-positive organism. Rapid infusion-related adverse drug reaction (ADR) of vancomycin is known as red man syndrome (RMS) or red neck syndrome. The manifestations of RMS are erythema, flushing, pruritis of the face, upper torso, and in severe cases dyspnea, chest pain, and hypotension. RMS occurs due to the release of histamine from degranulated mast cells and basophils. This histamine release is also associated with ciprofloxacin, amphotericin B, rifampicin, and teicoplanin. It can be prevented by slowing the infusion rate or pre-treatment with an H1- or H2-receptor antagonist.

KEY WORDS: Vancomycin; Glycopeptide; Red Man Syndrome

INTRODUCTION

Vancomycin is a tricyclic glycopeptide antibiotic derived from *Amycolatopsis orientalis* (formerly *Nocardia orientalis*) used for serious gram positive infections. It acts by inhibiting the cell wall synthesis, and it is indicated for the treatment of infections caused by methicillin resistant *Staphylococcus aureus*,[1] coagulase negative staphylococci, multidrug resistant *Staphylococcus epidermidis*, resistant strains of *Streptococcus pneumoniae*.[2] Vancomycin is associated with hypersensitive reactions, red man syndrome (RMS), and anaphylaxis. RMS is an infusion-related adverse drug reaction (ADR) of vancomycin also known as red neck syndrome. The direct release of histamine from the degranulation of mast cells and basophils causes RMS. The amount and rate at which vancomycin is infused affect the extent of histamine release.[2-4] Patients develop RMS when vancomycin infusion is given over <1 h. RMS is characterised by pruritis, and erythematous rash involving face, neck and upper torso, upper body flushing, hypotension, and cardiovascular collapse.[4] Patients develop these signs and symptoms 4-10 min after starting infusion or may begin after its completion.[1] Slowing intravenous (IV) administration or by pre-administration of H1 or H2 blockers can prevent the ADR.[4] Here is a case report of vancomycin induced RMS.

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CASE REPORT

A 20-year-old male patient with a known case of decompressive craniotomy following road transport accident and seizure disorder. The patient was met with an accident and had decompressive craniotomy done outside hospital and developed right hemiplagia with on and off seizure activity. Post injury presented with complaints of deformity of the left-sided skull associated with unhealthy wound. The patient was admitted with unhealthy scar tissue on left calvaria with underlying bone with discharging sinus. Diagnosed with right-sided bone flap osteomyelitis and underwent exploration, debridement, and bone flap removal. The patient was shifted to intensive care unit for neurological monitoring. Culture pus from scalp had growth *Pseudomonas aeruginosa* (moderate growth) sensitive to gentamycin, ciprofloxacin, ceftazidime, amikacin, tobramycin, piperacillin and tazobactam and

levofloxacin and *Enterococcus faecalis* (moderate growth) sensitive to vancomycin, chloramphenicol, and linezolid. IV antibiotics were started as per culture sensitivity. Vancomycin 1 g Q12 H over half an hour infusion was prescribed. The patient developed redness on chest, face, right, and left forearm after the first dose of vancomycin therapy. Vancomycin was stopped immediately. On examination, fall in blood pressure (110/80 mm/Hg) was observed. Vancomycin infusion was stopped, and diphenhydramine 50 mg was given intravenously for the management. The patient recovered thereafter.

DISCUSSION

The rapid intravenous infusion of vancomycin is associated with RMS, an anaphylactoid reaction. Several case reports are there. RMS can be prevented by reducing the infusion rate or by administration of H1 or H2 antagonists before vancomycin therapy.[5] Vancomycin should be administered over a period of not <60 min to avoid RMS.[6,7]

RMS is a common ADR of vancomycin. The reaction is characterised by flushing, erythema, and pruritis of face, neck, and upper torso.[8-10] A drop in blood pressure, chest pain, and back pain are also its symptoms. The mechanism behind RMS is the direct release of histamine by activation of mast cells and basophils. Other agents are also involved in pre-disposing patients to RMS such as opoids, muscle relaxants, and radiocontrast media. In severe cases, it is a life-threatening condition. Topical steroids and antihistamines can relieve most of its symptoms related to skin. RMS can be prevented by administering the vancomycin at rates ≤10 mg/min. In patients requiring more rapid infusions pre-medication with an H1 antihistamine or a combination of H1 and H2 antihistamine also prevent this reaction. Treatment is based on the severity of the reaction requiring prolonged hospitalization. Besides stopping the infusion, patient's medication list is also reviewed to discontinue other predisposing medications.[11-14]

CONCLUSION

In summary, infusion-related adverse effects of vancomycin are a serious problem. Clinicians should be aware about the rapid infusion-related ADR of vancomycin. The patient is informed about the reaction to avoid future complication. Studies have shown that vancomycin is much tolerated when it is given in smaller and more frequent doses. Pre-treatment with an H1-receptor antagonist or prolonged infusion can prevent the ADR.

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