Case Report

An anaesthesiologist's encounter with purple glove syndrome

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ABSTRACT

Purple glove syndrome (PGS) is a devastating complication of intravenous (IV) phenytoin administration. Anaesthetic management during the amputation of the limb for such patients is very challenging due to limited clinical experience. A 65-year-old woman developed PGS of left upper extremity after IV administration of phenytoin following generalised tonic-clonic seizures. The condition progressed rapidly leading to gangrene of left hand extending to the mid arm. Amputation was carried out under general anaesthesia with a supraglottic airway device. We discuss the prevention and alternate managements in PGS, which is a rare clinical entity with limited data in the literature.

Key words: Amputation, general anaesthesia, phenytoin, purple glove syndrome

INTRODUCTION

Intravenous (IV) administration of phenytoin can result in soft tissue injury at the site of injection leading to oedema and purplish-black discolouration of the hand. This is known as the purple glove syndrome (PGS).[1] It may occur during 1.7–5.9%[2] of all phenytoin administrations depending on the recognition as well as adverse drug event reporting. The management of PGS is mainly conservative, which includes limb elevation and physiotherapy.[3] However, some cases develop gangrenous changes warranting emergency amputation. The anaesthetic management of such a case can be very challenging as no ideal technique has been advocated.

CASE REPORT

A 65-year-old woman weighing 43 kg presented with an alleged history of generalised tonic-clonic seizures. There was no past history of seizure disorder. In the emergency room, the physician administered 600 mg of injection phenytoin sodium dissolved in 100 ml of normal saline through a 22G cannula sited into a vein on the dorsum of her left hand to flow over 20 min. The patient was later shifted to the ward for further monitoring. In the ward, maintenance dose of IV phenytoin was allowed to flow (100 mg in 500 ml of normal saline over 8 h). Four hours later, the patient complained of pain at the site of injection, which progressively became severe. The fingers, hand and forearm were swollen and within an hour had a purplish-black discolouration [Figure 1]. The radial artery and ulnar artery were not palpable under the oedema. The capillary refill under the nail bed was sluggish. The IV cannula was removed and the arm was kept elevated to reduce the oedema. The ultrasound Doppler study of the arm showed normal flow through the subclavian to digital arteries but the veins appeared collapsed. Initially, conservative management was initiated with IV non-steroidal anti-inflammatory drugs for pain relief. However, there was no respite in pain and the condition rapidly deteriorated with gangrenous changes appearing over the left hand up to the mid arm [Figure 2]. Surgical consultation was obtained and the diagnosis of PGS was suggested with the need for emergency

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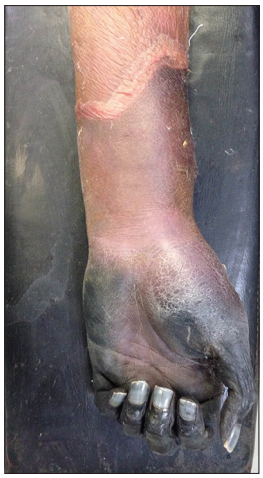

Figure 1: Swollen fingers and hand of the left upper extremity with bluish discoloration after phenytoin injection

amputation. Investigations included haemogram and serum phenytoin levels. Emergency amputation below the elbow was performed under general anaesthesia under spontaneous ventilation, and Proseal® LMA size 3 was used for airway management. Amputation was performed, and the procedure was uneventful. At the end of surgery, anaesthesia was reversed and with the return of protective airway reflexes, the Proseal® LMA was removed. The specimen was sent for biopsy. IV paracetamol was administered for postoperative pain relief. The recovery was uneventful.

DISCUSSION

Phenytoin, a broad spectrum anticonvulsant, has been widely administered parenterally for the treatment of seizures for more than 40 years. IV phenytoin is employed in emergency departments and neurological units for patients with active seizure disorders or who are unable to tolerate oral medication. Adverse reactions to phenytoin are not uncommon, due to phenytoin's narrow therapeutic index and pharmacokinetics; however, the adverse drug reaction known as PGS is a less heard of clinical entity with a prevalence of 1.7–5.9%.[2] PGS gets its name from the characteristic bluish discoloration of the skin, accompanied by pain and oedema distal to the site of IV administration of phenytoin. Generally, PGS occurs in three stages.[4] In the first stage, a pale blue or dark purple discoloration appears around the IV insertion site, 2–12 h after the administration of the drug. During the next 12–16 h (second stage), progression occurs as oedema and discolouration spread around all sides of the fingers, hand and forearm, hence the term 'purple glove'. Healing is the last stage as the

Figure 2: Gangrenous changes of the left hand extending up to mid arm

discoloration recedes, starting from the periphery and moving toward the original site of injury. The majority of reported cases resolve without complication, but a few cases resulting in necrosis have been reported.

The pathophysiology of PGS is poorly understood.[5] It has been suggested that the alkaline drug precipitates upon contact with blood and leaks out of the vein, from around the cannula and into the interstitial tissue.[6] This is likely to happen in a slow flowing stream or if the cannula is kinked, leading to stasis. Another possible mechanism could be that the highly alkaline solution induces vasoconstriction of the vein resulting in disruption of the endothelial-intercellular junctions and seepage of the drug into the interstitial space.[6] Extravasation of the highly albumin-bound (70–90%) phenytoin increases the interstitial oncotic pressure leading to oedema. Propylene glycol with its high osmolality causes necrosis of the tissue.

Women and the elderly are said to have an increased risk of PGS. Other factors associated with increased risk include peripheral vascular disease and diseases that weaken the vascular and dermal integrity, use of IV catheters smaller than 20-gauge and infusion of phenytoin at more than 25 mg/ml.[6,7] There were multiple risk factors in our patient like elderly age group, female gender, use of 22G cannula for administering phenytoin as well as the rate of infusion.

Therefore, administration of phenytoin should be into a free-flowing infusion line, through a large bore IV catheter sited into a large vein of the forearm, in a concentration of 10 mg/ml, and at a rate not exceeding 50 mg/min.[8] It should be diluted in 0.9% normal saline solution, and dextrose solutions and lactated Ringers solutions cannot be used with IV phenytoin due to the potential for precipitation. Any evidence of venous irritation such as pain, oedema and erythema warrants immediate discontinuation of the infusion and removal of the IV catheter.[6]

The diagnosis of PGS is based on the characteristic clinical findings and a high index of suspicion when it occurs after the administration of phenytoin. There was an initial differential diagnosis of compartment syndrome in our case. The other possible causes can be cellulitis and necrotising fasciitis. It can also be simply IV fluid extravasation. However, cellulitis and necrotising fasciitis produce discharge and bullous formation. Fluid extravasation has a delayed presentation and so does arterial thrombosis.

The management is mainly conservative (limb elevation, physiotherapy, control of pain, reassurance to the patient) and should be directed at minimising the degree of soft tissue damage.[3] The affected arm should not be used for venipuncture or blood pressure measurement. Medical management of PGS includes the application of nitroglycerine patch and IV heparin.

The role of the anaesthesiologist in this situation can be either for pain relief or a definitive surgical intervention like fasciotomy or amputation. Because of tissue injury and ischaemia, PGS is very painful. A low concentration of local anaesthetic would relieve the pain by preferentially blocking the Aδ and B fibres.[9] This can be achieved by brachial plexus block using local anaesthetic agents like ropivacaine. The addition of fentanyl potentiates local anaesthetic action via central opioid receptor mediated analgesia by peripheral uptake of fentanyl to the systemic circulation.

The sympathetic blockade, by blocking the α receptors, improves blood flow in vasospastic disorders.[10] Stellate ganglion blockade has the advantage of blocking the sympathetic innervation of the upper limb, thus improving the perfusion and relieving the ischaemic pain associated with vasospasm.[11]

Brachial plexus blockade has been used for this condition,[12] and the use of general anaesthesia in PGS has not been described before. We chose against brachial plexus blockade as our patient refused consent for the procedure.

CONCLUSION

IV administration of phenytoin can lead to PGS when small peripheral lines are used for drug administration. Ensuring a slow flow rate through larger lines can prevent such a complication.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Hanna DR. Purple glove syndrome: A complication of intravenous phenytoin. J Neurosci Nurs 1992;24:340-5.
2. O'Brien TJ, Cascino GD, So EL, Hanna DR. Incidence and clinical consequence of the purple glove syndrome in patients receiving intravenous phenytoin. Neurology 1998;51:1034-9.
3. Righini M, Angellillo-Scherrer A, Gueddi S, Le Gal G, Bounameaux H. Management of severe ischemia of the hand following intra-arterial injection. Thromb Haemost 2005;94:219-21.
4. Burneo JG, Anandan JV, Barkley GL. A prospective study of the incidence of the purple glove syndrome. Epilepsia 2001;42:1156-9.
5. Edwards JJ, Bosek V. Extravasation injury of the upper extremity by intravenous phenytoin. Anesth Analg 2002;94:672-3.
6. Snelson C, Dieckman B. Recognizing and managing purple glove syndrome. Crit Care Nurse 2000;20:54-61.
7. Spengler RF, Arrowsmith JB, Kilarski DJ, Buchanan C, Von Behren L, Graham DR. Severe soft-tissue injury following intravenous infusion of phenytoin. Patient and drug administration risk factors. Arch Intern Med 1988;148:1329-33.
8. Joint Formulary Committee British National Formulary. London, British Medical association and Royal Pharmaceutical Society of Great Britain, 1999.
9. Strickartz GR. Neural physiology and local anesthesia action. In: Cousins MJ, Bridenbaugh PO, editors. Neural Blockade in Clinical Anesthesia and Management of Pain. 3rd ed. Philadelphia: Lippincott; 1998. p. 35-54.
10. Breivik H, Cousins MJ, Lofstrom JB. Sympathetic neural blockade of upper and lower extremity. In: Cousins MJ, Bridenbaugh PO, editors. Neural Blockade in Clinical Anesthesia and Management of Pain. 3rd ed. Philadelphia: Lippincott; 1998. p. 411-45.
11. Elias M. Cervical sympathetic and stellate ganglion blocks. Pain Physician 2000;3:294-304.
12. Mahajan RP, Batra YK, Rajeev S. Intravenous phenytoin and percutaneous arterial cannulation: The purple-glove syndrome. Eur J Anaesthesiol 2007;24:900-1.