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Case Report

A Cyanotic Clue: Severe Methemoglobinemia Following Benzocaine Use for TEE Leading to Hypoxic Crisis

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ABSTRACT

We present a case of a 70-year-old man who developed life-threatening methemoglobinemia following topical benzocaine administration for transesophageal echocardiography (TEE). Prompt recognition and treatment with methylene blue led to rapid reversal of hypoxia. This case highlights the need for heightened awareness of this rare but serious complication.

Keywords: Methemoglobinemia, benzocaine, transesophageal echocardiography, methylene blue, Vitamin C

Introduction

Methemoglobinemia is a rare but potentially fatal condition caused by oxidation of hemoglobin's iron moiety from ferrous (Fe^{2+}) to ferric (Fe^{3+}), impairing oxygen delivery. It can be congenital or acquired, with the latter often triggered by oxidizing agents such as local anesthetics, nitrites and certain antibiotics[1]. In a large retrospective study at Mayo Clinic, out of 28,478 transesophageal echocardiograms (TEEs) performed over 90 months, 19 cases of clinically significant methemoglobinemia were identified. This corresponds to an incidence of approximately 0.067%.

Among local anesthetics, benzocaine has been prominently linked to methemoglobinemia, especially when used topically in mucosal procedure[2]. The clinical diagnosis is often challenging due to the nonspecific presentation of hypoxia unresponsive to oxygen therapy, with "chocolate-colored blood" being a classical but often missed sign[3].

TEE procedures frequently employ topical anesthetics and though rare, multiple case reports have documented benzocaine-induced methemoglobinemia following TEE, highlighting a preventable risk in routine diagnostics[4].

Case Presentation

A 70-year-old male with a history of hypertension, COPD, hyperlipidemia, bilateral lower extremity peripheral arterial disease, bladder cancer under surveillance and chronic benzodiazepine use presented with acute encephalopathy. MRI brain revealed multiple small, scattered acute to subacute infarcts across both supra- and infratentorial regions, suggestive of embolic etiology.

Neurology and cardiology consults were obtained. Given the suspicion for embolic source, he underwent TEE after receiving topical 20% benzocaine spray (HurriCaine). Shortly after the procedure, he developed peripheral cyanosis and hypoxia refractory to oxygen supplementation, requiring escalation from 0 to 10 L/min via non-rebreather mask. Despite increasing FiO_2, SpO_2 remained under 88%. Arterial blood gas revealed a PaO_2 of 371 mmHg with an O_2 saturation of 100%, but a fractional oxyhemoglobin (FO2Hb) of only 53% and a critically elevated methemoglobin level >30%. Characteristic appearance of chocolate-brown arterial blood is seen in **(Figure 1)**.

Figure 1: Distinct chocolate-brown discoloration of arterial blood observed in a patient who developed methemoglobinemia secondary to topical benzocaine administration during transesophageal echocardiography. Despite a markedly elevated PaO_2 (>300 mmHg), oxygen saturation remained low due to the oxidized (Fe^{3+}) form of hemoglobin, which cannot effectively bind or deliver oxygen. This abnormal blood color serves as a classic visual clue for diagnosis

The patient was promptly treated with IV methylene blue 1 mg/kg over 5 minutes. A second dose was administered one hour later due to persistent hypoxia. Oral vitamin C (ascorbic acid 1500 mg every 4 hours) was started due to unavailability of IV formulation. His oxygen requirement decreased significantly and by day 3 he was stable on room air. No further recurrence was noted.

Discussion

Benzocaine-induced methemoglobinemia occurs due to oxidative stress that overwhelms the enzymatic systems (chiefly NADH-cytochrome b5 reductase) responsible for reducing methaemoglobin back to haemoglobin[5]. TEE-related cases arise because benzocaine is applied directly to the oropharyngeal mucosa, allowing rapid systemic absorption.

Clinical suspicion arises when oxygen saturation is low despite high PaO_2, a phenomenon termed "saturation gap." Definitive diagnosis requires co-oximetry to quantify methaemoglobin levels. Methylene blue, acting as an artificial electron acceptor, restores Fe^{3+} to Fe^{2+} via the NADPH pathway, with ascorbic acid serving as adjunct therapy in severe or relapsing cases[6].

Preventive strategies include minimizing use of high-concentration benzocaine sprays, using lidocaine alternatives and monitoring high-risk patients closely during and after procedures involving topical anaesthetics.

Conclusion

This case illustrates the importance of early recognition of acquired methemoglobinemia, especially following procedures like TEE that involve topical benzocaine. Prompt initiation of methylene blue therapy is critical for reversal of life-threatening hypoxia. Providers should consider alternatives to benzocaine and maintain vigilance for methemoglobinemia in post-procedural hypoxia cases.

Competing Interests

The author(s) have no competing interests to declare.

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