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Acute Bronchospasm Associated with Polymethylmethacrylate Cement

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Polymethylmethacrylate bone cement is widely used in prosthetic joint implantation surgery and repair of bony defects. Well-recognized complications are most frequently seen during prosthetic joint implantation. We describe a patient who developed acute bronchospasm on application of polymethylmethacrylate during cranioplasty.

Case Report

A 71-yr-old, 65-kg, woman was to undergo cranioplasty to repair a 4 × 2 cm bony defect of the left temporal fossa left by clipping of internal carotid and ophthalmic artery aneurysms 9 months previously. She suffered no neurologic sequelae from the surgery. Her medical history was otherwise significant only for hypertension controlled with captopril. She reported an allergy to penicillin. She used no tobacco or alcohol. Physical examination was unremarkable.

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Just before induction of anesthesia, vancomycin and gentamicin were given without incident. The American Society of Anesthesiologists' standard monitors were used, along with airway pressure and volume monitors. Anesthesia was induced with thiopental and fentanyl, and muscle relaxation was attained with vecuronium. Proper placement of an 8.0-mm inner diameter cuffed oral endotracheal tube was confirmed by auscultation of bilateral, equal breath sounds, and positive end-tidal CO_2. Anesthesia was maintained with isoflurane (0.2%–1.5% end-tidal concentration) in 66% N_2O and 33% O_2 (adult circle circuit; total fresh gas flow 1.5–2 l/min). Mechanical ventilation by a Drager AV-2 ventilator (North American Drager, Telford, PA) was adjusted to maintain end-tidal CO_2 between 30 and 36 mmHg. Patient was in supine position, with head slightly turned to the right.

The patient's hemodynamic and respiratory parameters remained stable for the first 2 h of the procedure, during which the skull defect was exposed, with minimal blood loss. One minute after application of semi-solid polymethylmethacrylate on a steel-wire mesh spanning the bony defect, pulse oximetry reading (SaO_2) decreased from 99% to 89% (fig. 1B); heart rate increased from 62 to 85 beats/min, and blood pressure decreased from 110/65 to 80/50 mmHg (fig. 1A). Simultaneously, airway pressures abruptly increased from 32/22 cmH_2O (peak/plateau) to 40/32 cmH_2O (fig. 1C). The capnograph exhibited an upsloping plateau, and the end-tidal CO_2 decreased from 36 to 28 mmHg. Anterior chest auscultation confirmed presence of bilateral, equal breath sounds. In addition, diffuse high-pitch expiratory wheezes were heard. The blood pressure returned to baseline within 10 min after ephedrine, 5 mg, was given intravenously. Inspired oxygen concentration was increased to 100%, and positive end-expiratory pressure (PEEP) of 5 cmH_2O was added. With these maneuvers, SaO_2 improved from 89% to 92% over 5 min, but wheez-

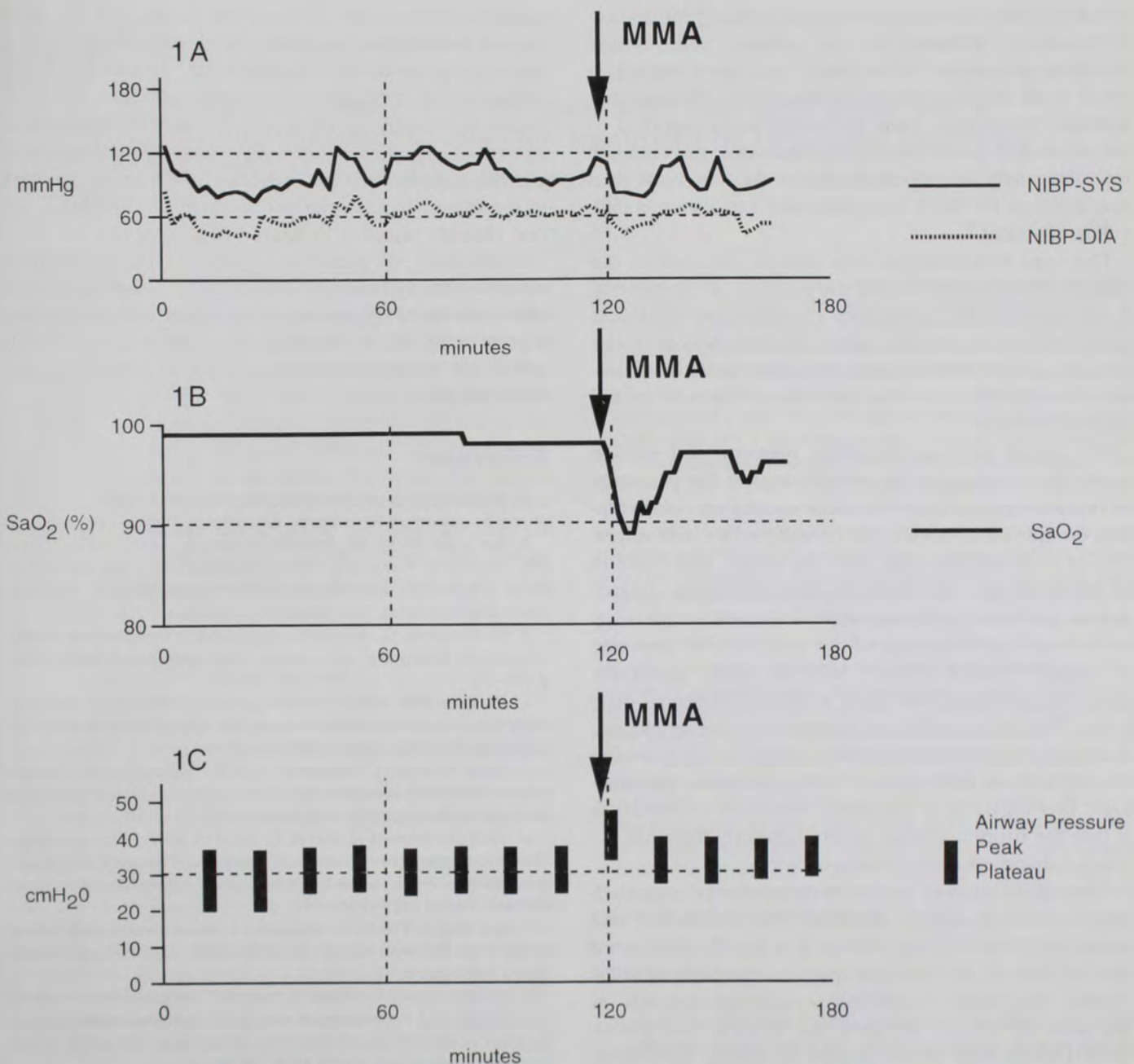

Fig 1. ***A,*** **Changes in systolic (NIBP-SYS) and diastolic (NIBP-DIA) blood pressure.** ***B,*** **Changes in pulse oximetry reading (SaO_2).** ***C,*** **Changes in peak and plateau airway pressure.** ***Large arrow*** **and MMA indicate polymethylmethacrylate bone cement application.**

ing persisted. Five puffs of nebulized albuterol (90 μg/puff) were then given through the endotracheal tube, and within 1 min the expiratory wheezes completely resolved; the airway pressure returned to baseline level, and SaO_2 increased to 97%. Anesthesia was continued with isoflurane in 100% O_2, and surgery was completed without further events. At the end of the 5-h procedure, the patient awoke easily, and the trachea was extubated. Subsequent hospital course and recovery were unremarkable.

Discussion

Polymethymethacrylate bone cement is a two-component polymer formed by mixing highly volatile liquid methylmethacrylate monomer, an accelerator, and polymethylmethacrylate powder. Multiple adverse effects have been associated with its use.[1-3] The putative mecha-

nisms of injury include a neurogenic reflex,[4] the release of vasoactive substance by the cement,[5] intravascular thrombin generation in the lungs,[6] and direct vasoactive effect from absorbed methylmethacrylate.[7] Because the adverse effects have most frequently been observed in the context of prosthetic hip implantations, the etiologic role of tissue or air embolization from the long bone marrow cavity to the heart and pulmonary circulation is difficult to discount.[8]

This case is unusual in two aspects. Because of the nature of the procedure and the position of the patient, it was unlikely that pulmonary embolization of particulate matter or air played a significant role. Secondly, the patient also exhibited acute reversible bronchospasm, an adverse effect that has not been reported in the clinical literature.

The abrupt increase in airway pressure and sudden onset of wheezing in this patient suggest the presence of bronchospasm. Other possible causes for this symptom complex that need to be considered include migration of endotracheal tube onto the carina, mobilization of tracheobronchial secretion, pneumothorax, coincidental pulmonary thromboembolism, and venous air embolism. Rapid clearing of the wheezing in response to inhaled bronchodilator, without other manipulations, helped to exclude most of these differential diagnoses. The trigger of bronchospasm may be an allergic reaction to methylmethacrylate, which cannot be excluded without subsequent challenge test or immunologic investigation. A plausible alternative explanation is that the bronchospasm resulted directly from the action of absorbed methylmethacrylate.

After application of polymethylmethacrylate, methylmethacrylate is rapidly absorbed into circulation and transported to the lungs, where it is rapidly eliminated into the alveoli, its level in expired air peaking after 2–5 min. The extent of systemic absorption depends on the area of contact between the cement and vascularized tissue and on the degree of curing. Low fresh gas flow and rebreathing of exhaled gases in a circle circuit probably prolong the exposure of the patient's lungs to methylmethacrylate. The direct pulmonary effect of methylmethacrylate in the absence of pulmonary embolization is neither well defined nor well known. There is indirect evidence suggesting that it may trigger bronchoconstriction. In workers subjected to prolonged inhalational exposure, reversible small airway obstruction could be demonstrated.[9] It also has been implicated as a trigger of occupational asthma.[10] It is therefore possible that this patient's bronchospasm was a result of exposure of her lungs to methylmethacrylate, and this together with vasodilation in the lungs resulted in significant hypoxemia and decrease in end-tidal carbon dioxide tension (increased dead space).

In summary, we presented a patient with no previous history of bronchospatic disease who developed bronchospasm after application of polymethylmethacrylate during cranioplasty. She was successfully treated with nebulized albuterol with immediate resolution of the bronchospasm.

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