Severe Laryngeal Oedema During Injection of Sodium Metrizoate ("Triosil")

Survival after Emergency Laryngostomy

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Numerous iodide-containing compounds are used in radiographic contrast media. Severe, untoward reactions to these agents are of two types, cardiorespiratory collapse and, less commonly, anaphylactic reactions (Goodman and Gilman, 1965). This paper reports a case of severe laryngeal oedema which developed as an allergic reaction to sodium metrizoate 75 per cent (Triosil) administered during cardiac catheterization. Emergency laryngostomy was performed to prevent fatal asphyxia. Mullins *et al.* (1963) reported death due to asphyxia from laryngeal oedema after intravenous Hypaque (sodium diatrizoate), and Youngblood, Williams, and Tuggle (1956) a similar death after intravenous sodium acetrizate, but survival of a patient under these circumstances has not been previously reported.

Case Report

A man aged 59 years developed exertional angina in May 1967. Examination, including electrocardiography and radiography, confirmed important calcific aortic valve disease.

He was admitted in September 1967, for detailed assessment before aortic valve surgery. Pethidine (50 mg.) and promethazine (25 mg.) were given intramuscularly 1 hour before cardiac catheterization which was carried out under local anaesthesia (lignocaine 1%, 6 ml.). Right heart catheterization was uneventful. Normal pulmonary venous capillary and right heart pressures were recorded.

An angio-catheter (No. 7) was introduced via a right brachial arteriotomy, and its tip passed to the ascending aorta. It was difficult to see the catheter on the image intensifier, so small volumes of sodium metrizoate 75 per

cent were injected through it. A total of 10 ml. was given over a period of 7 minutes while attempts were made to advance the catheter into the left ventricle.

At this stage the patient noted difficulty in swallowing and a strange taste in his mouth. Intravenous hydrocortisone hemisuccinate (100 mg.) and subcutaneous adrenaline 0.5 ml. were given immediately. However, over the next 2 minutes he complained of difficulty in breathing, and stridor developed. Direct laryngoscopy was attempted but there was marked oedema of the soft palate and pharyngeal walls and the vocal cords could not be seen. The stridor increased rapidly and was accompanied by violent inspiratory movements. Complete airway obstruction, profound cyanosis, loss of consciousness, and an idioventricular rate of 32 a minute followed in rapid succession.

Emergency laryngostomy was therefore performed by incising the neck at the lower end of the thyroid cartilage and opening the cricothyroid membrane. A Magill's cuffed tube was passed into the trachea and the cuff inflated. The patient's colour rapidly improved, sinus rhythm returned spontaneously, and he regained consciousness within 2 minutes. At this time the neck was grossly swollen and hard with "woody oedema". There was no rash. Further intravenous hydrocortisone hemisuccinate (200 mg.) and subcutaneous adrenaline (0.5 ml.) were given. Subsequently, under general anaesthesia, an otolaryngologist performed a formal tracheostomy and repaired the crico-thyroid membrane.

An electrocardiogram showed the presence of an acute posterior myocardial infarction, and later there was a rise in serum enzymes (SGOT 125 units per ml., LDH 920 units per ml.) from the previous normal values. The myocardial infarction presumably occurred during the period of severe anoxia.

The oedema of the neck slowly subsided over the next 3 days. He was given corticosteroids for 12 days in decreasing dosage. Five days after the emergency tracheostomy tube was removed. He was able to speak normally, and the wound healed without complications. At no time did his cardiac condition give rise to further concern.

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Previously the patient had never been given iodide-containing contrast agents, though he had developed a rash while taking penicillin. There was no family history of allergic disease.

Discussion

The development of severe laryngeal oedema during the intra-arterial injection of sodium metrizoate in this patient strongly suggests that the radiographic agent was the causative factor. The reaction was almost certainly due to hypersensitivity to iodine. Many patients developing allergic reactions to radiographic contrast media have no history of previous exposure to iodide-containing compounds (Sandström, 1953), and this was the case in the patient reported here.

Intradermal, subcutaneous, sublingual, conjunctival, and intravenous test injections have been given in an attempt to detect hypersensitivity to radio-opaque media. Finby, Evans, and Steinberg (1958) considered that intravenous testing was an unreliable indicator of hypersensitivity, and that it carried a risk. No test dose had been given in this case.

Abrams (1961) considered that sensitivity testing should be abandoned in angiographic studies, except in those patients who give a specific allergic history. This advice seems reasonable in view of the finding of McClenahan, Klotz, and Wilson (1963) that mild toxic reactions to iodide-containing media occurred 15 times more frequently in patients who had any history of previous allergic drug reactions.

In this patient, a severe allergic reaction developed despite prior administration of an antihistamine, and hydrocortisone and adrenaline failed to influence the tissue oedema significantly.

Abbey (1960) has summarized the arguments in favour of temporary laryngostomy rather than

tracheostomy for the emergency relief of acute laryngeal obstruction. The major point in favour of laryngostomy is its ease of execution by an operator who is unfamiliar with the detailed surgical anatomy of the neck.

Summary

A 59-year-old man with calcific aortic valve disease developed acute laryngeal oedema and asphyxia during the intra-arterial injection of sodium metrizoate 75 per cent. Emergency laryngostomy was performed. The patient recovered.

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