Acute pulmonary oedema during intravenous urography

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Summary

Acute pulmonary oedema complicating an intravenous urography in a previously healthy young patient is reported. Although this complication is rare, it is life-threatening. The exact etiology is not clear, but the possibility of an idiosyncratic reaction to the contrast medium is speculated.

Key words – Acute pulmonary oedema, intravenous urography, idiosyncratic reaction.

Introduction

Adverse reactions following intravenous contrast media administration are uncommon. In a study of 302,083 urographic examinations, Shehadi[1] established an overall incidence of adverse reactions to be at or slightly below 5%. Based on the clinical status of the patients and the need for treatment these adverse reactions are graded into minor, intermediate, severe and death. Minor reactions such as nausea and vomiting are common and they do not require any form of treatment. Intermediate reactions like bronchospasm require some form of treatment in the radiology department, whereas severe reactions under which acute pulmonary oedema is categorised require intensive treatment and hospitalisation.

Case Report

A 20-year old female patient underwent intravenous urography for right loin pain and hematuria. Fifty millilitres of sodium iothalamate (Conray 325) was injected intravenously at the start of the urographic examination. Five minutes later she had nausea and sweating, followed soon after by difficulty in breathing and frothing of the mouth.

On examination there was peripheral cyanosis, pulse rate was 100 per minute and the blood pressure was 80 mm Hg on palpation. Auscultation of the chest revealed generalised crepitations. A clinical diagnosis of acute pulmonary edema was made. Acute treatment included intravenous fluids, chlorpheniramine 4mg (IV), aminophylline 250mg (IV), hydrocortisone, 200mg (IV), frusemide 40mg (IV) and dopamine intravenously to maintain blood pressure. A plain chest radiograph taken 40 minutes after the onset of symptoms revealed diffuse alveolar opacities consistent with acute pulmonary edema (Figure 1).

The ECG showed sinus rhythm with tachycardia at 100 per minute and there was no ischemic changes. The clinical condition of the patient did not improve following the initial treatment. She was then transferred to the intensive care unit (ICU) for further management. A portable chest radiograph taken at 6 hours after the onset of symptoms showed further confluence of alveolar opacities (Figure 2). The clinical condition and the serial chest radiographs from the second day onwards showed improvement. On the fourth day the plain chest radiograph was clear.

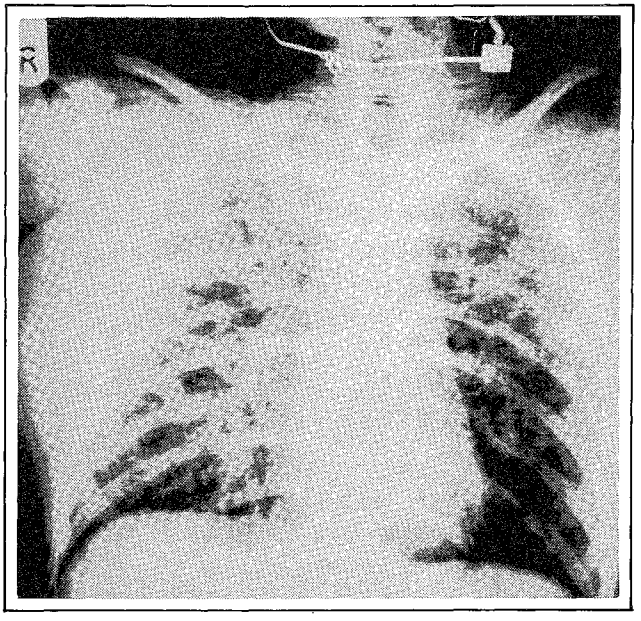

Figure 1:
Chest radiograph at 40 minutes showing diffuse alveolar opacities.

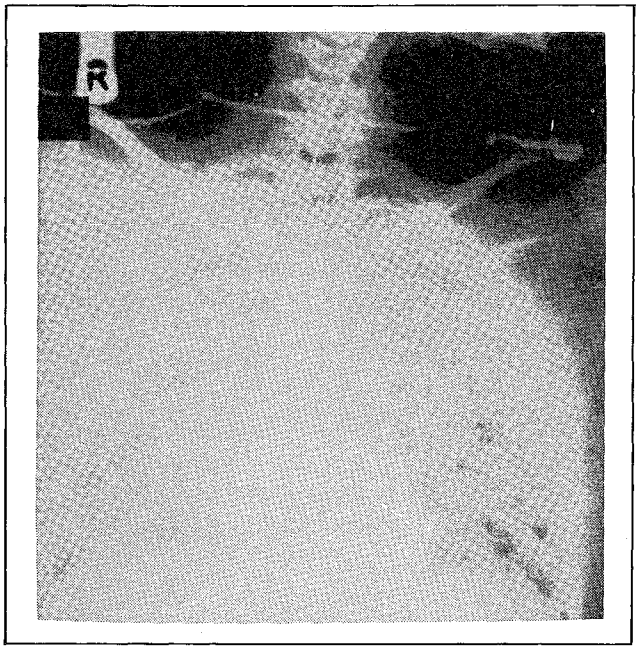

Figure 2:
Chest radiograph at 6 hours showing confluence of aveolar shadows consistent with pulmonary oedema.

Discussion

Acute pulmonary oedema is a rare but serious complication of intravenous urography. The incidence of this reaction has been reported as 1:25,000 of urographic examinations.[1] Since the condition is life threatening, institution of immediate resuscitation is mandatory to prevent a fatal outcome. Lalli[2] reviewed the postmortem results of 71 deaths following urography and found that acute pulmonary oedema, which occurred in 18 patients, was the most common underlying cause of death.

The pathogenesis of acute pulmonary oedema following intravenous constrast media administration has not been clearly defined. However, the hypotheses which were put forward appear to

focus on the effects of the contrast medium on the cardiovascular, pulmonary and the central nervous systems, which result in changes leading to pulmonary oedema. The changes occurring in these areas were thought to be initiated by the hyperosmolar, chemotoxic effects of the contrast medium and/or consequent to an idiosyncratic reaction.[1,2,3]

It has been speculated that acute cardiovascular compromise is a preceding event, which could have occurred as a result of an extra volume load in the intravascular spaces, cardiac arrhythmia or acute myocardial insufficiency from an idiosyncratic reaction. However, the extra plasma volume due to standard injected dose and the shift of fluid from the extravascular spaces into the vascular compartment as a result of the hyperosmolar effect of the contrast medium would be insignificant to cause any untoward effects so long as the cardiac function is normal. The ECG of the patient did not show any changes to indicate myocardial ischemia or infarction.

A direct effect of contrast medium on the pulmonary vascular system has also been considered. The hyperosmolarity and chemotoxicity of the contrast medium could cause damage to the pulmonary vascular endothelium resulting in 'endothelial leakage' with escape of fluid into the extravascular interstitial spaces. In addition, the lungs are known to contain a rich network of mast cells which release histamine following stimulation by contrast medium.[2]

Lalli[2] highlighted the role of the central system in the pathogenesis of acute pulmonary oedema. Contrast medium causes direct stimulation of that part of the hypothalamus which is responsible for the autonomic function resulting in activity of the sympathetic pathway to the lung where there is contraction of the precapillary and postcapillary sphincters, subsequently trapping blood and concomitant exudation. It would not be possible to state the precise mechanism for the development of acute pulmonary oedema following intravenous contrast medium administration. We were unable to explain this complication in our patient from acute cardiac failure due to volume overload or acute myocardial insufficiency as a result of ischemia or infarction. Perhaps it was a pulmonary manifestation of an idiosyncratic reaction due to intravenous contrast medium, where there was leakage of fluid from the pulmonary vessesl as postulated by Greganti et al[3]. Since the mechanisms offered are speculative it would not be possible to institute definitive preventive measures. It would be relevant to reemphasise the facts regarding safety and protection of patients.

All procedures utilising contrast media carry a definite risk of adverse reactions, and while most reactions are minor, life-threatening and fatal complications may occur. The risk-benefit factor should always be carefully evaluated and at all times a fully-equipped emergency trolley, resuscitative equipment and competent personnel be readily available.[1]

References

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