Noncardiogenic Pulmonary Edema Associated with Intravenous Radiocontrast Administration*

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We describe a patient who developed noncardiogenic pulmonary edema (NCPE) following the administration of intravenous radiocontrast material (RCM) and review the possible pathogenic mechanisms leading to its occurrence.

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\[ \text{COP} = \text{colloid oncotic pressure}; \text{NCPE} = \text{noncardiogenic pulmonary edema}; \text{RCM} = \text{radiocontrast material}; \text{UAO} = \text{upper airway obstruction} \]

A wide variety of serious adverse reactions to the administration of radiocontrast material (RCM) have been reported and include anaphylactoid reactions,\textsuperscript{1,3} renal toxicity,\textsuperscript{4} and thyroid storm.\textsuperscript{5} We describe a case of noncardiogenic pulmonary edema (NCPE) occurring after the intravenous injection of RCM.

CASE REPORT

A 50-year-old woman underwent computed tomography (CT) of the head for evaluation of memory loss. She received 100 ml of diatrizoate meglumine (Angiovisit 282), an intravenous RCM. The patient had never previously been exposed to RCM and within 1 min of its administration, she complained of severe dyspnea and a sensation of fullness in her neck, as if someone were choking her. The radiologist evaluating the patient did not report evidence of facial swelling, stridor, or skin rash.

Following the immediate transfer to the intensive care unit (ICU), the patient began to improve as manifested by resolution of her dyspnea and the choking feeling in her neck. Physical examination was pertinent for an initial blood pressure of 80/60 mm Hg with a pulse rate of 110 and a respiratory rate of 24 breaths per minute. Soft inspiratory rales were heard throughout the lung fields and the upper airway appeared normal.

An arterial blood specimen revealed a pH of 7.37, an oxygen tension of 54 mm Hg, and a carbon dioxide tension of 31 mm Hg while receiving nasal cannula oxygen at a flow rate of 6 L/min. An electrocardiogram showed sinus tachycardia and the chest roentgenogram demonstrated bilateral pulmonary edema (Fig 1). A pulmonary artery catheter was placed revealing a cardiac index of 2.4 L/min/m\(^2\), pulmonary artery pressures of 27/11 mm Hg, and a pulmonary artery occlusive pressure of 5 mm Hg.

Hemodynamic parameters following a 1,500-ml infusion of normal saline solution were blood pressure of 105/70 mm Hg, cardiac index of 3.1 L/min/m\(^2\), pulmonary artery pressures of 32/19 mm Hg, and pulmonary artery occlusive pressure of 11 mm Hg. A follow-up chest roentgenogram performed 4 h after ICU admission (Fig 2) showed nearly complete resolution of the pulmonary edema, and the patient was discharged from the ICU 12 h later after showing no evidence of myocardial infarction.

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![Figure 1. Chest roentgenogram demonstrating pulmonary edema.](image1)

![Figure 2. Chest roentgenogram performed four hours later showing significant clearing of the pulmonary edema.](image2)
cardiac disease due to intravascular volume expansion from the osmotic effects of the RCM. NCPE appears to be a rare occurrence following the administration of RCM. In a review of the literature, only four well-described and hemodynamically verified cases of NCPE following RCM exposure were found.

The pathophysiologic state of NCPE following the administration of RCM is poorly understood. The hemodynamic profiles of patients described with this disorder and the finding of a high ratio of pulmonary edema fluid colloid oncotic pressure (COP) to serum COP described in one patient suggests a pulmonary-capillary permeability defect. Proposed mechanisms producing disruption of the pulmonary-capillary barrier in these patients have included neurogenic and inflammatory processes.

Neurogenic pulmonary edema is unlikely to be an important mechanism in RCM-associated NCPE due to the lack of clinical findings that would suggest cerebral hypoxia or dysfunction in patients described with this entity. Inflammatory mechanisms appear to play an important role, as other reactions to RCMs such as bronchospasm, urticaria, and hypotension have long been thought to be mediated by inflammation.

Possible mediators of this pulmonary-capillary permeability defect include activation of the classic or alternative complement pathways and the formation of antibodies to RCM. In addition to the classic and alternative pathways of complement activation, RCM may activate complement through a unique and nonsequential fashion. RCMs and other hyperosmolar solutions such as mannitol can also stimulate the direct release of mediators such as histamine from mast cells and basophils. This appears to be a mechanism that is independent of IgE or complement-mediated stimulation of these cells. Direct injury to the endothelial barrier due to the osmolarity of the RCMs is another possible mechanism that can contribute to the pulmonary-capillary permeability defect producing NCPE.

Our present case raises another potential cause for the NCPE occurring after exposure to RCM. Upper airway obstruction (UAO) is a well-recognized cause of NCPE. Edema of the larynx, epiglottis, or surrounding tissues can occur following RCM exposure and lead to upper airway obstruction. Our patient's description of a choking feeling in her neck suggests the possibility of UAO.

In most patients with pulmonary edema due to UAO, the pulmonary edema resolves rapidly, usually within 24 h. The previously described cases of RCM-induced pulmonary edema without evidence of UAO showed evidence of pulmonar edema lasting from one to four days. The rapid resolution of the patient's pulmonary edema and hypotension over several hours goes against a significant pulmonary-capillary injury and supports a transient hydrostatic process for the formation of her pulmonary edema as has been described to occur following UAO.

In summary, our patient represents a case of NCPE following RCM exposure. Inflammatory or osmotic injury to the pulmonary-capillary membrane appears to be the main mechanism leading to this form of pulmonary edema. In selected patients, occult UAO should also be considered since its presentation may be subtle and it represents a potentially preventable and reversible cause of NCPE.

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