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Letter to the Editor / Editöre Mektup

THE PULMONARY EDEMA AND ENCEPHALOPATHY INDUCED BY LOW OSMOLAR NON-IONIC CONTRAST MEDIA WERE SUCCESSFULLY TREATED WITH HEMODIALYSIS

DÜŞÜK OZMOLAR NON-İYONİK KONTRAST MADDELERİN NEDEN OLDUĞU AKCİĞER ÖDEMİ VE ENSEFALOPATİ HEMODİYALİZLE BAŞARIYLA TEDAVİ EDİLDİ

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To the editor,

A case of non-cardiogenic pulmonary edema (NCPE) and encephalopathy was observed following the administration of low osmolar intravenous non-ionic contrast medium (RCM) during an abdominal CT scan. This resolved with haemodialysis. A 62-year-old female patient underwent abdominal computed tomography (CT) imaging with iodinated contrast agent iohexol (Omnipaque 300) for the investigation of chronic abdominal pain the patient exhibited symptoms of emesis, dysphagia, tachypnea, and respiratory distress 15 minutes after the initial observation. In consideration of the potential for contrast agent-related hypersensitivity, a dosage of 25 milligrams of pheniramine and 20 milligrams of methylprednisolone was administered intravenously. The patient's oxygen saturation began to decline, necessitating her admission to the intensive care unit. Upon admission to the intensive care unit, the patient exhibited hypotension and tachycardia. The oxygen saturation was determined to be 80%. Upon auscultation of the lungs, the presence of diffuse rales was noted in all areas. The patient was found to be unconscious on neurologic examination. The pupils were bilaterally dilated and unresponsive to direct and indirect light. There was lack of any motor response to painful stimuli in four extremities, and plantar reflex bilaterally indifferent. Due to the patient's poor clinical condition, computed tomography (CT) and magnetic resonance imaging (MRI) of the brain could not be performed. A chest X-ray revealed diffuse pulmonary edema (Figure 1).

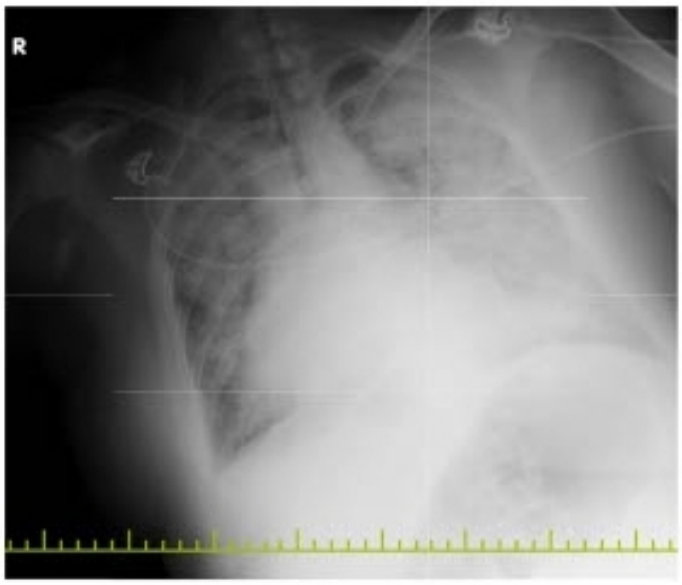

Figure 1. Posterior Anterior Chest Radiograph before Hemodialysis

Arterial blood gas analysis revealed a pH of 7.150, a pCO2 of 32 mmHg, a PO2 of 92 mmHg, a lactate level of 5.1 mmol/L, and a HCO3 level of 13 mmol/L. The patient was intubated and ventilated. The patient exhibited alveolar fluid secretion through the intubation tube for a period of two hours, during which time the saturation level could not be elevated to a value of 80 or above. Consequently, a furosemide infusion was initiated as a diuretic treatment, and methylprednisolone 40 mg was administered intravenously. A hemodialysis catheter was inserted and hemodialysis was performed for 2 hours to clear the contrast agent and reduce pulmonary edema. The patient had no previously documented history of allergic reactions. The patient was being treated with bisoprolol for arrhythmia. All biochemical laboratory tests were within normal limits prior to exposure to contrast media. Cardiac enzymes (troponin and creatine kinase) were found to be within normal limits in blood tests taken prior to intubation in the intensive care unit.

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Additionally, the electrocardiogram demonstrated sinus tachycardia. Other laboratory values were as follows; normal blood urea nitrogen (BUN): 18 mg/dL, creatinine : 1 mg/dL, uric acid: 9 mg/dL, aspartate aminotransferase (AST): 245 IU/L, alanine aminotransferase (ALT): 212 IU/L, gamma-glutamyl transferase (GGT): 332 IU/L, lactate dehydrogenase (LDH): 589 U/L, C-reactive protein (CRP): 16 mg/L, white blood cell (WBC) : 15,600/μL, hemoglobin (Hb): 16 g/dL, hematocrit (Hct): 47%, platelet (PLT): 187,000/μL, D-dimer: 8.65 mg/L. Following hemodialysis, the patient's blood gas and laboratory values demonstrated improvement. Arterial blood gas analysis revealed a pH of 7.32, PCO2:36.9 mmHg, a lactate concentration of 1.4 mmol/L, a bicarbonate concentration of 20 mmol/L, a D-dimer concentration of 7.15 mg/L, AST:145 IU/L, and ALT: 122 IU/L. Following dialysis, there was no alveolar secretion from the breathing tube, and the oxygen saturation was 100%. A chest X-ray taken after dialysis revealed a reduction in pulmonary edema (Figure 2).

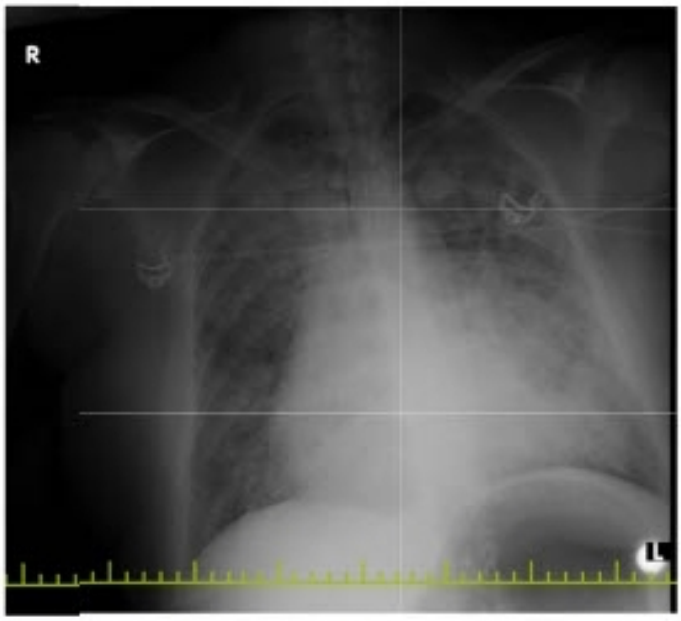

Figure 2. Posterior Anterior Chest Radiograph after Hemodialysis

Following dialysis, the patient regained consciousness and exhibited a fully normalized neurologic examination. The patient was extubated in a controlled manner 24 hours after intubation. The patient's urine output and all vital signs were within the normal range. The patient, who had returned to a normal oral intake, was mobilized in the room and exhibited a normal respiratory pattern. They were discharged from the intensive care unit 48 hours later in a healthy condition without any evidence of neurological deficit.

The occurrence of immediate, serious adverse reactions following the intravenous administration of low-osmolar RCM is exceedingly rare (1). Pulmonary edema and contrast-induced encephalopathy (CIE) have been documented as a rare adverse reaction to intravenous infusion of any type of contrast material. Pulmonary edema represents a significant proportion of fatal reactions resulting from intravenous infusion of contrast media, with a prevalence of 10-20% (2). Pulmonary edema can be classified as cardiogenic or non-cardiogenic. In the case of non-cardiogenic pulmonary edema, the underlying pathology is increased vascular permeability, which is a consequence of endothelial damage. This is caused by the release of a number of mediators and complement (3,4). It is hypothesized that low or high osmolar contrast may result in damage to erythrocyte morphology. A case of non-cardiogenic pulmonary edema following intravenous administration of low-osmolar RCM has been documented in the medical literature. The case of Laurence et al. was treated with non-invasive mechanical ventilation, corticosteroids, and diuretic therapy (5). In the case of a fatal NCPE episode, Paul and George proposed the administration of oxygen with continuous positive airway pressure or invasive ventilation with positive end-expiratory pressure as a treatment option. It was recommended that diuretics or vasodilators should be avoided(4). In another case, the patient developed shock, circulatory and respiratory failure after low osmolar RCM administration. Despite invasive mechanical ventilation and shock treatment, the patient died(6). In our case, the patient's saturation could not be increased to 85% and above with invasive ventilation, but saturation was 100% after hemodialysis. It has been reported in the literature that all types of contrast media induce CIE (7). The phenomenon of contrast-induced neurotoxicity is the result of a disruption in the blood-brain barrier (BBB) and an increase in permeability (8). The enhanced permeability permitted the exudation of contrast media and the occurrence of colloid osmotic pressure changes within the brain tissue. These changes have the potential to result in the development of cerebral edema and neurotoxicity, as well as contrast-induced encephalopathy(9). It has been observed that encephalopathy may develop in some patients following the administration of low-osmolar contrast medium during coronary angiography. In these cases, conservative treatment was sufficient to achieve recovery (10,11). The cases of encephalopathy documented in the medical literature occurred subsequent to coronary or carotid interventions. In contrast, in our patient, the condition developed following the administration of contrast material into a peripheral vessel. A conservative treatment approach was consistently employed in all cases presented in the literature. In our case, the patient underwent hemodialysis, which resulted in a rapid recovery of neurological functions.

In conclusion, a comprehensive literature review did not reveal any cases of concomitant contrast-induced pulmonary edema and encephalopathy. Furthermore, in contrast to all other cases, we performed hemodialysis and ensured that both pulmonary and neurologic functions were restored without sequelae. Although the contrast medium

has a low osmolarity, it causes an acute change in osmolarity at the cellular level within the body. It enhances permeability in both the vessel wall and the blood-brain barrier. Given that not all individuals exhibit this condition, it is imperative to investigate the underlying immunological mechanisms that contribute to its development. In the context of a rapidly evolving clinical case, the necessity for immediate intervention constrains the scope for further research. The most important issue here is to quickly identify the situation and intervene quickly. The contrast-induced clinic was identified with alacrity in our patient. It is important to note that contrast-induced pulmonary edema and encephalopathy may develop simultaneously, and that intervention with hemodialysis may be necessary to save the patient's life.

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Informed consent: The patient provided informed consent.

Conflict of interest : The authors have no conflicts of interest.

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