CONTRAST MEDIA INDUCED ACUTE PULMONARY EDEMA DURING COMPUTED TOMOGRAPHIC EXAMINATION: A CASE REPORT

BİLGİSAYARLI TOMOGRafi ÇEKİMİNDE KONTRAST MADDENİN İNDÜKLEDİĞİ PULMONER ÖDEM: OLGU SUNUMU

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ÖZET

Son yıllarda kontrast maddeler, kliniği radyolojik görüntüleme işlemlerini için; hem girişimsel olan hem de olmayan tanı ve tedavi yöntemleri arasında sıkça kullanılmaktadır. Ancak kullanılan, dermatolojik, gastrointestinal, kardiyovasküler sistem, solunum sistemi ile ilgili istenmeyen yan etkilerle sebep edilebilir. Basit allergik reaksiyonlar olümde kadar değişebilen sonuçlar doğurabilir ve bu reaksiyonlar genellikle önceden tahmin edilemez. Her ne kadar bu tip reaksiyonlar iyonik olmayan iyotlu kontrast maddelerle kayasa iyonik iyotlu kontrast maddelerle daha sık görülecektir, ölençül olantları her iki grup kontrast maddelerle benzer şekilde hakkında bilgi sağla bilgisayarlı tomografi çiçek esnasında, iyonik olmayan iyotlu kontrast maddesinin sonucu akut pulmoner ödem gelişen bir olaydır. 

INTRODUCtION

Contrast agents have become commonly used in both interventional and non-interventional diagnostic and therapeutic procedures in recent years, and their practical applications are continuously increasing. However, ionic or...
not, all contrast agents may result in a large spectrum of side effects involving mild ones such as a simple allergic reactions or more severe reactions such as nephropathy, anaphylactic reaction, hypotension, respiratory or cardiac arrest, and events that may result in death (1,2). Fatal anaphylactic reactions occur shortly after exposure to a contrast agent and account for 0.04% of all cases (3).

Pulmonary edema developing after exposure to a radiological contrast agent generally results in death in the absence of a rapid and coordinated intervention (4, 5). We hereby present a rare case in the literature of a patient who developed acute pulmonary edema upon the administration of non-ionic iodine containing contrast agent and was discharged with full recovery upon early and appropriate treatment.

CASE REPORT

An 85 year-old female patient, who was examined and referred to us by an external healthcare center due to a single episode of hemoptysis 15 days prior, was assessed at our outpatient department. Her medical history included hypertension and cardiac disease and she was using furosemide, benidipine, diltiazem, olmesartan + hydrochlorothiazide. She rarely smoked, and did not smoke for the last 25 years. She did not illustrate any allergic history. Physical examination findings were as follows; arterial blood pressure was 120/70 mmHg, pulse rate was 78/min, inferior inspiratory rales were noted in the respiratory examination, room-air saturation measured with a pulse-oximeter was 96%, and the other system examinations were normal.

The laboratory analysis showed that the hemogram and biochemical parameters were normal. Posteroanterior lung graphy showed normal cardiothoracic ratio, clear aortic knob, diffuse descending aorta, both costophrenic sinuses open, left hemidiaphragma positioned upwards, and mild reticular density increase at left inferior zone of the paracardiac region (Figure 1); thorax computed tomography was requested.

The patient developed shortness of breath, wheezing, and cyanosis of the hands and lips after thorax tomography with intravenous iohexol and transferred to the emergency unit. During the emergency unit admission, the patient was pale and dyspneic, and cyanosis was observed in the finger tips and lips. Her arterial blood pressure was 180/90 mmHg and pulse rate was 110 per minute. The respiratory system examination showed bilateral inspiratory rales extensive at the superior, intermediary, and inferior zones. Cardiac sounds were tachycardic, no additional sound was heard. There was jugular venous distension. Saturation was 74% on pulse-oximeter and radial arterial blood gases measurements showed apparent hypoxic respiratory failure with ph: 7.46, pCO2: 37.7, pO2: 38, and saturation: 74%. Results of the other biochemical and hematologic analyses were within normal limits.

Electrocardiography did not show pathology other than sinus tachycardia. The left ventricle was hypertrophic, but wall movements were normal, ejection fraction was 60-65%, right structures were normal, and pulmonary artery pressure was 45 mmHg. Posteroanterior lung graphy showed diffuse bilateral reticulonodular patch infiltration zones (Figure 2).
Following the oxygen therapy, bronchodilators and diuretic treatment were administered at the emergency unit, and the patient was transferred to the service unit with the diagnoses of acute pulmonary edema + respiratory failure.

The patient was administered nasal oxygen and intravenous furosemide, she was closely monitored, and saturation was followed-up daily. Posteroanterior lung graphies (Figure 3 and 4) were obtained and upon stabilization of her clinical presentation, radiological regression and improvements observed in respiration. The patient was discharged upon recovery on the third day.

**DISCUSSION**

Intravenous or oral contrast agents are frequently used for radiological imaging procedures and their practical applications are continuously increasing.

Ionic or not, all contrast agents may result in certain reactions within the body. Although the ionic agents are associated with a higher rate of reactions, the rate of severe reactions is similar between both types of agents (7, 8).

Most of these reactions are allergic reactions; anaphylactic reactions resulting in death are rare and observed at similar rates with ionic or non-ionic agents (7).

Hypersensitivity or an anaphylactic reaction that develops after administration of intravenous contrast agent can also be encountered in cases with no previous history of allergic reactions. They are more common with IV administrations and mostly associated with the contrast agents with high osmolarity (9, 10).

The development of pulmonary edema after the administration of contrast agent is rare. Pulmonary edema can be classified into two groups as cardiogenic and non-cardiogenic edema.

Cardiogenic pulmonary edema is caused by the increased intravascular volume by hyperosmolar agents, which in turn results in cardiac failure (11, 12).
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The increased microvascular permeability caused by the leaked fluid is held responsible for non-cardiogenic edema (3). This type of reaction is generally associated with the toxic effects of the contrast agent on the pulmonary capillary membrane and the volume load; however, the pathogenesis of the vascular permeability increase is not clear. Two hypotheses have been suggested in the literature; allergic or immunologic reaction and the direct chemotoxic or osmotic effects on the vascular endothelium were held responsible, and it has been argued that the administered agent causes endothelial damage through the activation of the mediator and complement leading or through direct effects (3, 6).

In the present case, the echocardiography showed a hypertrophic left ventricle, but revealed normal wall movements, and the ejection fraction was reported as 60-65%.

The recommended treatment approach includes opening the airways, oxygen therapy, furosemide, cardiac output enhancers when required, volume enhancing agents, positive pressure ventilation, and mechanical ventilation. Corticosteroid treatment for anaphylaxis may also be helpful (3).

Following nasal oxygen and diuretic (furosemide) therapy, the patient presented here-in, showed clinical stabilization, radiological regression, and recovery in respiratory failure on the third day and she was discharged with recovery.

In conclusion, although rare, acute pulmonary edema developing after the administration of a radiological contrast agent is a life-threatening condition and should be suspected in patients with rapid onset of shortness of breath after radiological examination. Effective treatment should be immediately initiated in these patients.

REFERENCES


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