World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-34, May 2024
ISSN: 2749-3644

PULMONARY EDEMA IN PATIENTS WITH INTERSTITIAL PNEUMONIA AND CORONARY HEART DISEASE: RADIOLOGY DIAGNOSTIC

Nizamova Madina Mirgabtizyanovna[1, 2]
Yuldasheva Manzura Akromovna[2]
Borodavko Tatyana Gennadyevna[2]
Sergey Sergeyevich Kozivko[2]
[1]Center for the Development of Professional Qualifications of Medical Workers, Tashkent, Uzbekistan
[2]Republican Scientific Center for Emergency Medical Care
Tashkent, Uzbekistan

Article history:

Received: March 11th 2024

Accepted: April 10th 2024

Abstract:

Pulmonary edema, a critical complication in emergency medical scenarios, often leads to terminal states and fatalities. This study investigates specific X-ray and computed tomography (CT) signs for the differential diagnosis of hemodynamic and membranogenic pulmonary edema in emergency patients with diverse pathologies.

Methods: Clinical and radiological data from 30 patients with acute pulmonary edema were analyzed. Two groups were formed based on the type of pulmonary edema: interstitial pneumonia-related membranogenic edema (Group 1) and coronary heart disease-related cardiogenic edema (Group 2). X-ray and CT scans were performed and compared with the clinical progression.

Results: Group 1, predominantly with membranogenic edema, exhibited interstitial pulmonary edema on X-ray (80%), progressing to alveolar edema. CT scans revealed asymmetrical "ground glass" appearance, often with zones of consolidation. Group 2, with cardiogenic edema, presented enhanced interstitial patterns, evolving into alveolar edema with central distribution on CT. Effusions and heart chamber dilation were noted in Group 2.

Conclusion: Comprehensive and timely diagnosis plays a crucial role in unraveling the pathophysiological intricacies of pulmonary edema. CT emerges as a highly effective tool, achieving a notable 96–98% accuracy in differentiating between cardiogenic and non-cardiogenic edema. The significance of detailed imaging is underscored by the distinct radiographic patterns observed in each group. Specifically, CT illuminates unique features of alveolar pulmonary edema, enhancing its role in the differential diagnosis. Moreover, the correlation between CT findings and clinical dynamics, along with specific nosologies, provides valuable insights into the nature and severity of pulmonary edema.

Keywords: pulmonary edema, computed tomography (ct), cardiogenic, non-cardiogenic, alveolar edema

INTRODUCTION

Relevance of the Problem: Pulmonary edema is a common complication of several urgent medical conditions in the emergency medical system and often leads to the development of a terminal state and the death of the patient. The pathogenesis of pulmonary edema is well-known at present. Balanced fluid exchange through the microvessel walls in the lungs is the foundation for adequate gas exchange and protects against edema. Pulmonary edema is a complex process involving disruptions in the body's fluid-electrolyte and colloid-osmotic balances. Depending on the primary cause, four types of pulmonary edema can be distinguished: hemodynamic (cardiogenic), membrane-related (non-cardiogenic), central, and mixed [1,6].

Hemodynamic (cardiogenic) pulmonary edema, as a rule, is a consequence of left ventricular failure in hypertension, heart defects, myocardial infarction, due to impaired hemodynamics of the pulmonary circulation (pulmonary congestion and increased pulmonary capillary pressure) [3]. The main cause of the development of cardiogenic pulmonary edema is acute left ventricular failure associated with a decrease in myocardial contractility [6].

World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-34, May 2024
ISSN: 2749-3644

Membranogenic (non-cardiogenic) edema implies deep disturbances in the permeability of pulmonary capillaries with the release of fluid with a high protein content, including fibrin, into the lumen of the alveoli. The classic manifestation of this type of edema is acute respiratory distress syndrome (ARDS). Acute respiratory distress syndrome (ARDS) is a clinical syndrome characterized by non-cardiogenic edema of the lung tissue with severe impairment of gas exchange and respiratory mechanics, ventilation/perfusion ratio in the lungs [5]. The development of ARDS can be caused by both "direct" damaging factors - aspiration of aggressive substances, trauma, inhalation of irritating gases, and "indirect" factors, which include inflammatory mediators that have a damaging effect on the endothelium of the microcirculatory bed [4 ,5]. Reactions occurring in lung tissue during ARDS are always universal and nonspecific [6,7]. Today it is becoming clear that ARDS is a non-homogeneous, polyetiological disease, has complex, not fully understood mechanisms of pathogenesis and requires differentiated and individual treatment in each specific clinical situation [2].

In many manuals, articles, reviews, the X-ray picture is called one of the leading criteria for diagnosing severe pneumonia, viral-bacterial pneumonia, viral pneumonia, ARDS, but the wording in the section "Changes in the lungs" is very sparse - "bilateral infiltrates", "diffuse damage", "extensive areas of consolidation," which requires consideration of more detailed X-ray and CT semiotics of ARDS [4].

The differential diagnosis between cardiogenic and non-cardiogenic pulmonary edema is difficult - both conditions have largely similar clinical and radiological manifestations [6,8].

The purpose of the study was to identify specific X-ray and computed tomography (CT) signs that allow for differential diagnosis of hemodynamic and membranogenic pulmonary edema upon admission of emergency patients to a multidisciplinary hospital with various pathologies.

PATIENTS AND METHODS

An extensive analysis of clinical and radiological data was carried out in 30 patients (18 men and 12 women) from the therapeutic and cardiology departments with acute pulmonary edema syndrome. The average age of patients was 47.3±18.4 years. Of these, 15 patients were included in the first study group, 15 patients in the second group.

The patients were divided into two conditional groups depending on the type of pulmonary edema. The first group included 15 patients (10 men and 5 women) who were treated for interstitial pneumonia, patients with severe forms of pancreatic necrosis and massive blood loss with the development of membranogenic pulmonary edema. The second group included 15 patients (8 men and 7 women) with various manifestations of coronary heart disease (CHD): acute and recurrent myocardial infarction with the development of the classic picture of cardiogenic pulmonary edema.

X-ray examination was carried out on a digital X-ray apparatus Apelem, "Platinum 2" (France), "MobiRen-MTU" (Uzbekistan). Multislice computed tomography (MSCT) of the chest organs was performed using an Aquilion Prime 160 MSCT device (Canon Medical System) and an MSCT 640 "Aquilion one" Genesis version (Canon).

Radiological studies were compared with the clinical picture at various stages of the disease.

RESULTS

The first group of the study included 15 people, of whom 9 were men and 6 women. The average age of the patients was 39.4±16.6 years. Death was recorded in 11 patients (73.3%).

According to the data obtained, in the clinical picture of patients of the first group, already upon admission to the hospital, all symptoms were dominated by signs of respiratory failure, which was manifested by increased heart rate, shortness of breath, cyanosis, and a compensatory increase in blood pressure.

In parallel with the development of the clinical picture, the patients underwent X-ray examination and computed tomography. In 3 patients (20%) in the first or third day from the onset of respiratory failure, the X-ray picture was practically not manifested in anything; in 12 patients (80%) in the phase of interstitial pulmonary edema, a diffuse increase in the pulmonary pattern was noted, predominantly of a fine-mesh type, root pattern was characterized by little structure, the sinuses were free, the borders of the heart were not expanded (Fig. 1a). Taking into account the radiological studies performed, in 3 patients (20%) with ARDS, no pathological changes were noted on the radiographs, but the clinical picture was consistent with this pathology. This was the indication for CT scanning. In all patients in this group, the "ground glass" was asymmetrical, diffuse, spreading throughout all lobes of both lungs. (Table 1), more pronounced in the peripheral zone and posterior segments. When perform-

World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-34, May 2024
ISSN: 2749-3644

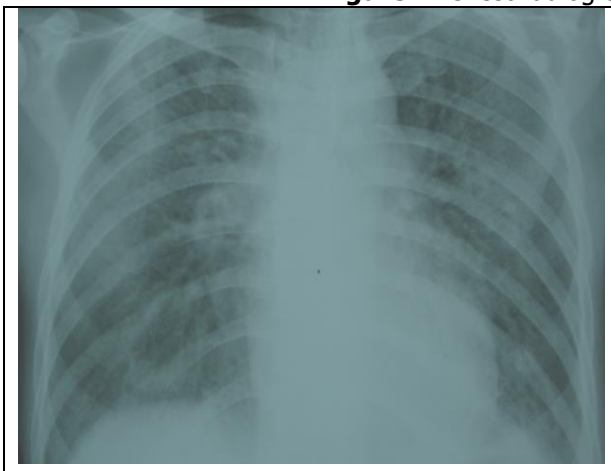
ing densitometric analysis of lung tissue, in areas of "ground glass", an increase in density was noted on the right to -499±29 units N., on the left to -482±19 units N. The effusion in the pleural cavity was not detected, the heart chambers in almost all patients were not dilated (Fig. 2a).

Subsequently, starting from the fourth day, dyspnea continued to progressively increase in all patients, hypoxemia developed (PaO2 <55 mm Hg, SO2 <80-82%), which was poorly corrected by mechanical ventilation with high positive pressure at the end of the day. -doha (PEEP) and high O2 content in the gas mixture. Auscultation data indicated a negative development of the situation and the involvement of an even larger area of damage to the alveoli of the lung tissue in the pathological process. Failure of compensatory processes led to multiple organ failure. During this period of time, diffuse intense focal-focal confluent shadows were detected on the radiograph, more in the peripheral parts, but with symmetrical damage to both lungs. The interstitial phase of pulmonary edema gave way to the alveolar phase. Focal focal shadows merged over the course of observation, forming large infiltrates, mainly in the basal segments, against the background of which "air bronchography" was traced. At the same time, the root picture of the lungs was not clearly differentiated; the sinuses were traced in the majority of patients (Fig. 1b, c). The CT picture in most cases was characterized by focal uniform (100%), asymmetrical (66.7%) compaction of the lung tissue of the "ground glass" type and zones of focal alveolar consolidation, of an asymmetrical nature, against which "air bronchografia" was observed fiya (Fig. 2b). When carrying out densitometric analysis, the density of lung tissue in areas of compaction according to the type of alveolar consolidation in the basal segments was: on the right -65.5±0.5 HU, on the left -66±1 HU. In 33.3% of patients, there was an effusion in the pleural cavity, mainly on the left, up to 23 mm thick, with a density up to +8 HU (figure 2b,c).

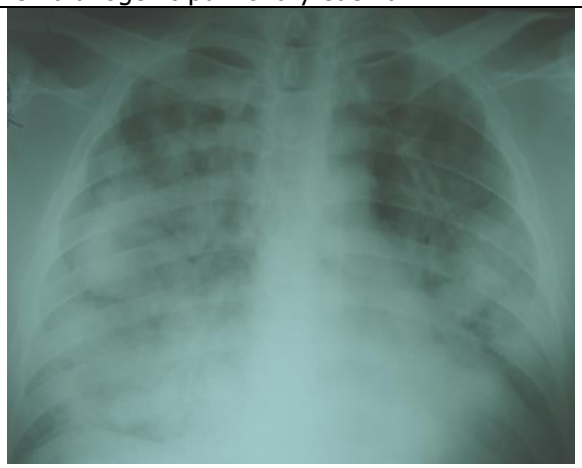
On the 7-10th day, the progression of the pathological process forced respiratory support for patients and, as a rule, to transfer patients to artificial ventilation when oxygen saturation drops to critical levels. The condition of the patients was characterized as extremely severe, there was diffuse cyanosis of the skin, acrocyanosis, depression of consciousness, which indicated a disruption of normal gas exchange in the lungs with the development of hypoxia, hypoxemia and indirectly caused damage to target organs (impaired hemodynamics, decreased blood pressure, cardiac arrhythmias, signs of multiple organ failure). Hypoxemia (PaO2 < 50 mm Hg, SO2 < 75%) could not be corrected even with a high level of (full name) PEEP, and pulse oximetry was often impossible due to poor blood flow in the periphery.

On radiographs of the chest organs during this period of time, a fine-medium cellular pulmonary pattern was visible in all lung fields and zones, but more in the basal segments (Fig. 1d). The CT picture was characterized by uneven reticular changes, predominantly subpleural, in the posterior and basal segments of the lungs (Fig. 2d).

Figure 1. Chest radiographs of membranogenic pulmonary edema



a) diffuse, symmetrical enhancement of the pulmonary pattern according to the cellular type; interstitial phase of pulmonary edema.



b) symmetrical focal-focal shadows, mainly in the basal zones; alveolar phase of pulmonary edema.

World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-34, May 2024
ISSN: 2749-3644

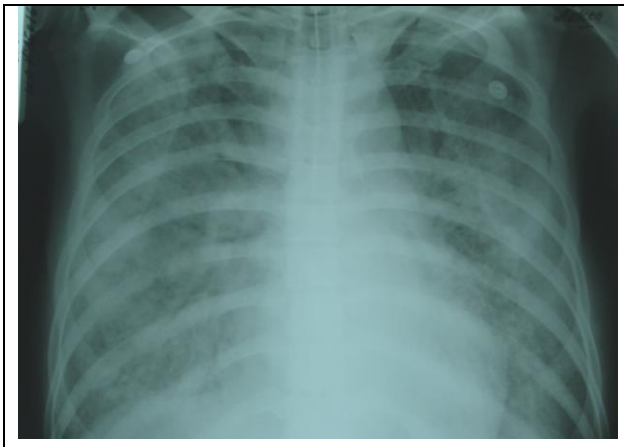
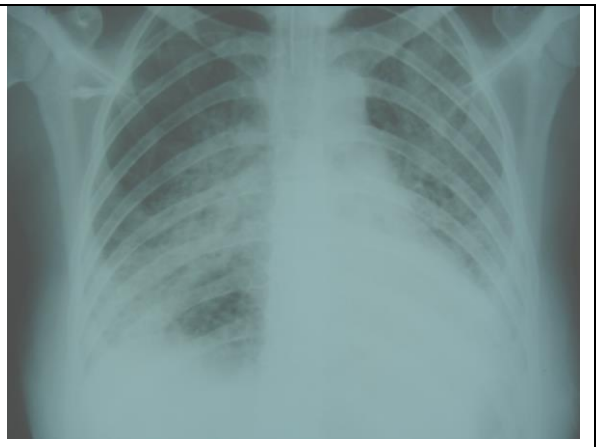
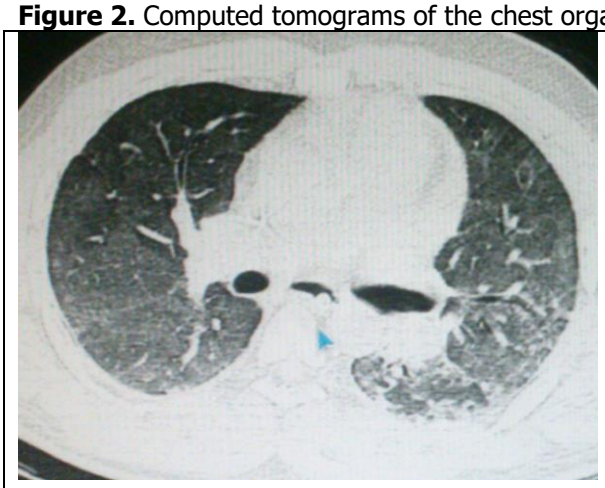
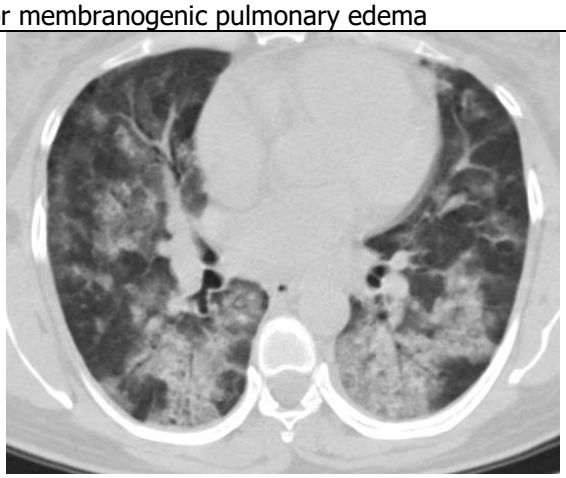
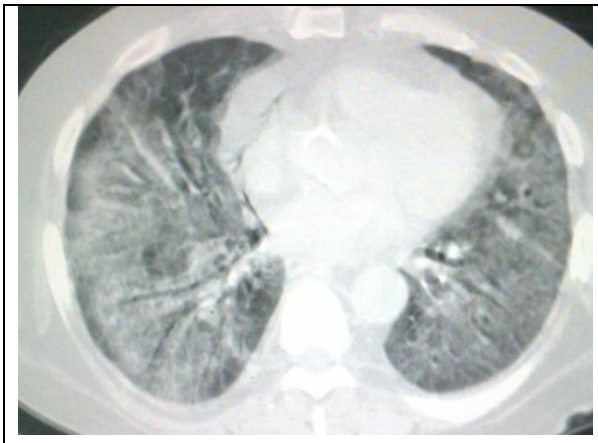
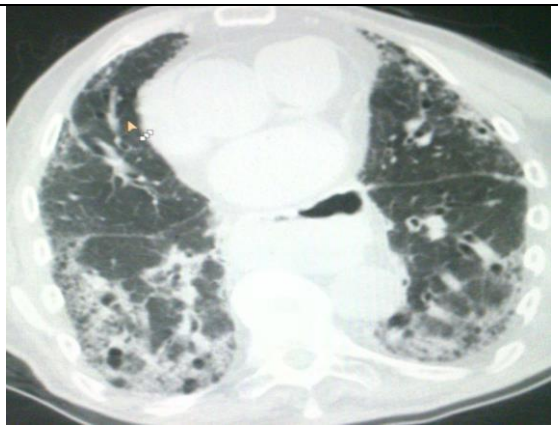
	
c) diffuse symmetrical focal-focal shadows, mainly in the lateral and basal zones; alveolar phase of pulmonary edema.	d) deformation of the pulmonary pattern

Figure 2. Computed tomograms of the chest organs for membranogenic pulmonary edema

	
a) bilateral compaction of the lung tissue like "ground glass"; interstitial phase of pulmonary edema.	b) compaction of lung tissue according to the type of alveolar consolidation; alveolar phase of pulmonary edema.

World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-34, May 2024
ISSN: 2749-3644

	
c) diffuse compaction of lung tissue according to the type of alveolar consolidation and "ground glass", in dependent areas, in the basal and posterior segments of the lungs; alveolar phase of pulmonary edema.	d) reticular changes, mainly in the lateral zones

The second group of the study included patients with cardiogenic pulmonary edema, of which 9 patients were men and 6 women. The average age of patients was 55.3±16.7 years. Death was recorded in 6 patients (40%). The clinical picture of hemodynamic pulmonary edema, with which patients were admitted to the intensive care unit, was bright: cyanosis of the lips and nails, cold sweat, agitation. The patients had tachypnea (RR 40-60 per minute), tachycardia, noisy breathing, and wheezing was heard on auscultation. At the stage of alveolar pulmonary edema, respiratory failure, shortness of breath, puffiness of the face, and swelling of the neck veins developed. Bubbling breathing and variable moist rales, foaming from the mouth were the main criteria for diagnosing hemodynamic pulmonary edema. CVP during pulmonary edema increased to 12 cm of water. Art. and more. Changes in blood gases were characterized by certain dynamics: at the initial stage, moderate hypocapnia was noted; then, as pulmonary edema progressed, PaO2 and PaCO2 decreased; at the late stage there was an increase in PaCO2 and a decrease in PaO2.

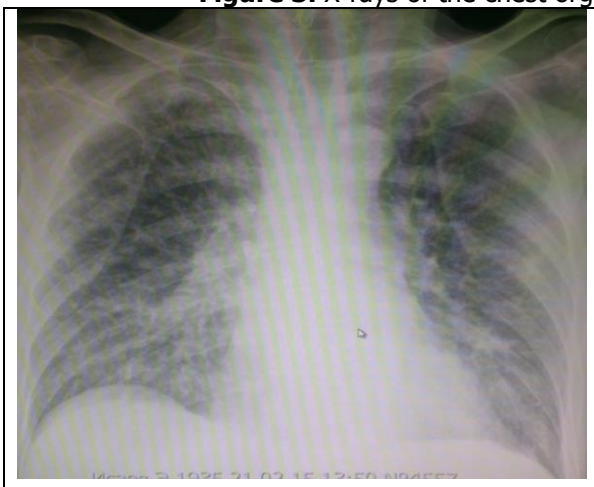
The X-ray picture in patients with myocardial infarction in the first hours of the development of the pathological process did not manifest itself in anything. In patients with chronic heart failure (CHF), against the background of heart defects, a history of myocardial infarction with focal replacement of muscle with scar tissue, venous plethora with peribronchial and perivasal infiltration was noted in 20 (3 patients), indicating more organic changes in both the heart itself and changes in hemodynamics as a result of the duration of the disease and contractile insufficiency of the left ventricle (Fig. 3a). In 80% (12 patients), the general X-ray picture was characterized by an enhanced interstitial pattern, mainly in the hilar zone. After the patients entered the phase of alveolar pulmonary edema, the radiograph showed focal shadows of a confluent nature, forming infiltrate-like formations with the symptom of "air bronchography", reminiscent of the picture of "butterfly wings" or "bat", mainly in the central, hilar zone. the picture was not differentiated; darkening of the sinuses was noted, more on the right due to the presence of effusion, which was confirmed by computed tomography. The boundaries of the heart cavities were expanded, the left parts of the heart predominated (Fig. 3 b,c,d). The X-ray picture dynamically followed the clinical course of events - relief of edema entailed a change in chest radiographs, namely the disappearance of focal shadows, the progression of the process, as a rule, led to death.

CT scan performed in patients (3 patients) with coronary heart disease in the phase of interstitial pulmonary edema was characterized by pronounced peribronchial and perivasal infiltration, symmetrical and uneven foci of "ground glass", density on the right -458±4.5 HU, on the left (Fig. 4a, b). CT scan – the picture of alveolar edema was characterized by compaction of the type of alveolar consolidation, located symmetrically in the basal, central zone, with the pres-

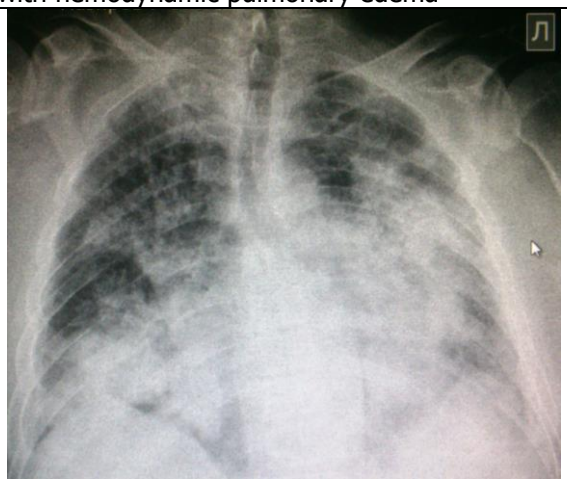
World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-34, May 2024
ISSN: 2749-3644

ence of air bronchography (Fig. 4a, b). The consolidation density of the lung tissue reached 65.5±0.5 HU on the right, and -66±1 HU on the left. There was an effusion in the pleural cavity, mostly on the right, up to 3.6 cm thick, with a density from +2 HU to +7 HU . The expansion of the left chambers of the heart predominated.

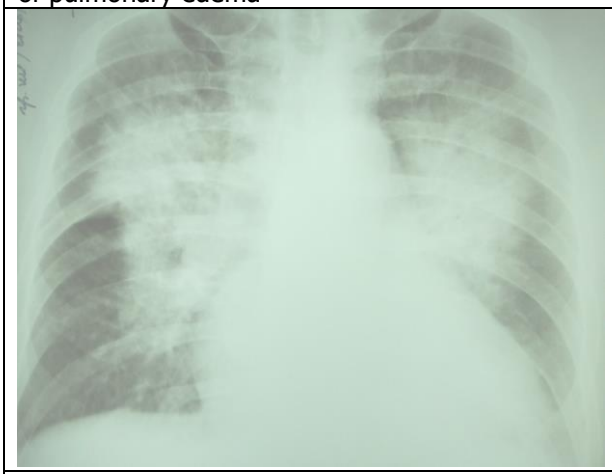
Figure 3. X-rays of the chest organs with hemodynamic pulmonary edema



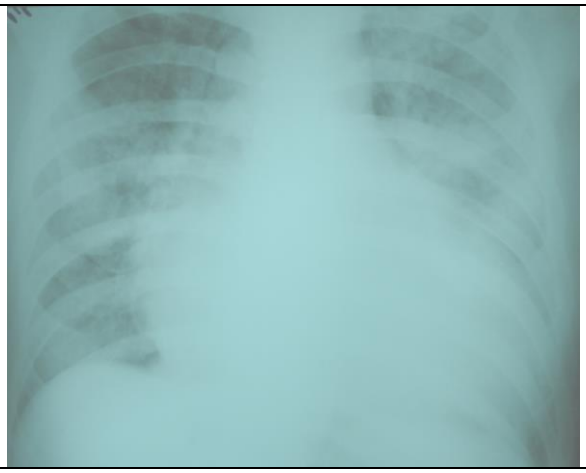
a) increased pulmonary pattern of the venous type with peribronchovasal infiltration; interstitial phase of pulmonary edema



b) symmetrical focal-focal shadows mainly in the root zone; alveolar phase of pulmonary edema.



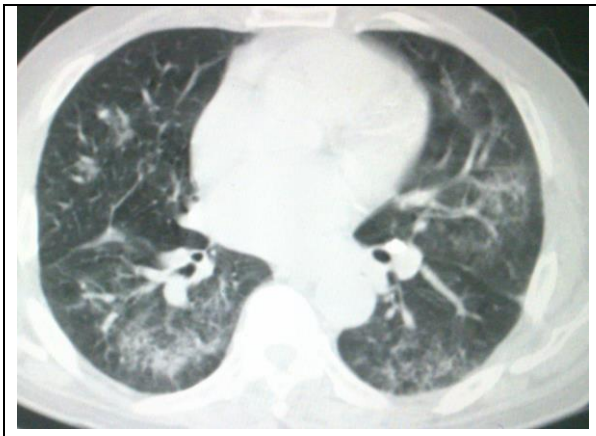
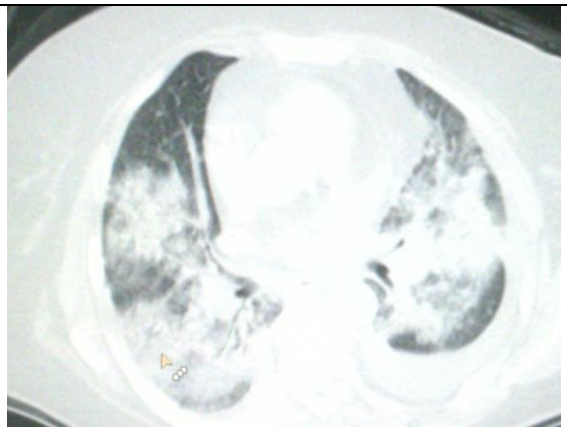
c) "bat" symptom



d) pulmonary edema in a patient with mitral heart disease; alveolar phase.

Figure 4. CT of the chest organs for hemodynamic pulmonary edema

World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-34, May 2024
ISSN: 2749-3644

	
a) increased venous pattern, with perivasal infiltration and areas of "ground glass"; interstitial phase of pulmonary edema.	b) compaction of lung tissue according to the type of alveolar consolidation in the hilar, central zone, the presence of effusion in the left pleural cavity; alveolar phase of pulmonary edema.

We present a comparative table of computed tomographic semiotics of acute pulmonary respiratory distress syndrome and cardiogenic edema in the phase of interstitial and alveolar pulmonary edema.

Table 1. Computed tomographic semiotics of acute pulmonary respiratory distress syndrome and cardiogenic edema in the phase of interstitial pulmonary edema

Signs			First group	Second group
"ground glass"	character		diffuse, uniform (100%);	diffuse, uniform (100%);
	symmetry		asymmetrical (100%)	symmetrical (100%)
	localization		all lobes (100 %)	all lobes (100%)
	Wed optical density on the	right	-499±29 HU	-458±4,5 HU
		left	-482±19 HU	-458±24 HU
Alveolar consolidation	character		none (66.7%); focal, uneven (33.3%)	none AK (100%)
	"air bronchography"		100% of total occurrence	-
	gravitational gradient		100% of total occurrence	-
	Wed optical density on the	right	-173±23 HU	-
		left	-180±110 HU	-
	localization		Inferior lobe (33,3%)	-

World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-34, May 2024
ISSN: 2749-3644

Pulmonary pattern	not changed 100%	enhanced due to venous plethora, peribronchovas-cular thickening (100%)
Intrathoracic lymph nodes	increased (66.7%)	increased (66.7%)
Pleural effusion	None	on both sides
Chambers of the heart	not expanded	expanded
Pericardial effusion	None	available (66.7%)
Character of pulmonary edema	interstitial	interstitial

Table 2.

Computed tomographic semiotics of acute pulmonary respiratory distress syndrome and cardiogenic pulmonary ede-ma in the phase of alveolar pulmonary edema

Signs			First group	Second group
" ground glass"	Character		focal uniform (100%);	-
	Symmetry		asymmetrical (66.7%)	-
	Localization		all shares (100%)	-
	Wed optical density on the	right	-499±29ед.Н.	-458±4,5 HU
		left	-482±19ед.Н.	-458±24 HU
Alveolar consolidation	Character		focal uniform (66.7%) diffuse uneven (33.3%)	diffuse uniform (100%)
	Symmetry		asymmetrical (100%)	symmetrical (100%)
	"air bronchography"		yes (100%)	yes (100%)
	Wed optical density on the	right	-65.5±0.5 HU	-45±60 HU
		left	-66±1 HU	-33±46 HU
	Localization		all lobes, more in the lower lobe (66.7%)	all lobes, more in the central one (100%)
Pleural effusion			no effusion (66.7%)	on both sides(100%)
Chambers of the heart			on both sides (33.3%)	expanded (100%)

World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-34, May 2024
ISSN: 2749-3644

Character of pulmonary edema	not expanded 100%	alveolar 100%

Thus, the main points of differential diagnosis of types of pulmonary edema presented in this article with a comparison of clinical and morphological data allow us to confidently differentiate these life-threatening conditions, which is of decisive importance in the choice of further tactics and, accordingly, treatment outcomes.

CONCLUSION

1. Comprehensive diagnosis of pulmonary edema in the early stages of development allows us to identify the main stages of the pathological process, assess its dynamics and, in most cases, clarify the pathophysiological mechanism of the occurrence of edema.
2. Computed tomography makes it possible to timely diagnose pulmonary edema and establish its nature in case of cardiogenic edema in 96%, and in case of respiratory distress syndrome – in 98% of cases.
3. The general x-ray picture of the first group of patients was characterized by interstitial pulmonary edema (80%) and in 20% of cases no changes were noted on the x-ray. In patients of the second group, interstitial edema was observed in 80% of cases, and venous congestion was noted in 20% of cases. The alveolar stage of pulmonary edema in patients of the first and both groups was diagnosed in 80% of cases.
4. In patients of both groups, on CT in the interstitial phase, the "ground glass" appearance of pulmonary edema was symmetrical, uniform, diffuse, spreading throughout all lobes of both lungs. Moreover, in patients of the first group the process was 100% asymmetrical, and in patients of the second group it was symmetrical in 100% of cases. Against the background of ground glass in patients of the first group, zones of consolidation were determined in 33% of cases. No consolidation zones were observed in patients of the second group. The pulmonary pattern was changed in 100% of cases in patients of the second group, while in patients of the first group the pulmonary pattern was not changed. Effusion in the pleural cavity and pericardial cavity was not detected in patients of the first group, but in patients of the second group there was effusion in 66.7% of cases. Dilation of the heart chambers was recorded in all patients of the second group; in patients of the first group, the heart chambers were not dilated.
5. During the phase of alveolar pulmonary edema, patients with ARDS and cardiogenic pulmonary edema were characterized by alveolar compaction; this sign was recorded in 100% of cases. According to the prevalence of the process for patients with ARDS, it is typical to spread throughout all lobes of both lungs, but more in the lower lobe (66.7%) due to the gravitational gradient. And for patients with cardiogenic edema, it is typical to spread throughout all lobes of both lungs, but more centrally (100%). The average optical density of the alveolar compaction in patients of the first group on the right was -65.5±0.5 units N., which is 19.5 units N. more than in patients of the second group, and on the left this indicator was -66±1 units N., which is 33 units N. more than in patients of the second group.
6. The dependence of the obtained CT data on the dynamics of the clinical picture and the specific nosology that determined the development of the pathological process was revealed. Patients of the first group, as a rule, were admitted with an acute, new-onset pathology (interstitial pneumonia); the dynamics of the clinical picture depended on the severity of the disease and the rate of development of pathological processes in the body. Patients of the second group had a history of coronary heart disease, with a long course, changes in hemodynamics, restructuring of the heart muscle, and congestion in the pulmonary circulation.

REFERENCES:

1. Avdeykin SN, Tyurin IN, Kozlov IA. Correction of hemodynamics in severe community-acquired pneumonia complicated by acute respiratory distress syndrome. Med Novosti. 2018;2(18):19-28.
2. Vlasenko AV, Golubev AM, Alexeev VG, Bulatov NN, Evdokimov EA. Mechanisms of pathogenesis, diagnosis, and treatment of acute respiratory distress syndrome. Part I. Med Alphabet. 2017;1(5):5-13.
3. Korovin AE, Novitsky AA, Makarov DA. Acute respiratory distress syndrome: current state of the problem. Klin Patsiol. 2018;24(2):32-41.
4. Kut'kin DV. Features of radiographic patterns in patients with viral-bacterial pneumonia and prediction of acute respiratory distress syndrome risk. Russ Med J. Med Rev. 2016;24(3):144-147.
5. Maltseva LA, Mosentsev NF, Mishchenko EA. New respiratory and hemodynamic strategies of revised Berlin definitions of acute respiratory distress syndrome. Med Neotlozhn Sost. 2016;(75):4:92-95.

World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-34, May 2024
ISSN: 2749-3644

6. Shtabnitskiy VA. Cardiogenic and non-cardiogenic pulmonary edema. Spravochnik Poliklinicheskogo Vracha. 2017;6:38-44.
7. Shatalov VI, Shchegolev AV, Gritsai AN, Afonchikov VS. Neurogenic pulmonary edema. Vestn Anesteziol Reanimatol. 2018;15(1):55-62.
8. Cereda M, Xin Y, Meeder N. Visualizing the propagation of acute lung injury. Anesthesiology. 2016;124:121-131.
9. Cereda M, Xin Y, Hamedani H, Bellani G. Tidal changes on CT and progression of ARDS. BMG. 2017 Nov;72(11):981-989.