CASE REPORT

A case of heart failure mimicking COVID-19 pneumonia: The role of clinical and chest computed tomography findings in the differential diagnosis

COVID-19 pnömonisini taklit eden bir kalp yetersizliği olgusu: Ayırıcı tanıda klinik ve akciğer bilgisayarlı tomografi bulgularının rolü

Zekeriya Doğan, M.D.,[1] Ciğdem İleri, M.D.,[2] Çağan Yıldırım, M.D.,[1] Halil Ataş, M.D.,[1] Altuğ Çinçin, M.D.,[1] Beste Özben, M.D.,[1] Bülent Mutlu, M.D.[1]

[1]Department of Cardiology, Marmara University Faculty of Medicine, İstanbul, Turkey
[2]Department of Cardiology, Ümraniye Training and Research Hospital, İstanbul, Turkey

Summary– Acute heart failure (HF) is one of the most common cardiac emergencies. Pulmonary edema caused by HF may mimic an exudative disease on chest computed tomography scans. Coronavirus disease 2019 (COVID-19) emerged in China in December 2019 and quickly spread around the world. During this pandemic period, the need to exclude the possibility of COVID-19 pneumonia in patients with acute dyspnea may cause a delay in the diagnosis and treatment of patients with acutely decompensated HF who have similar symptoms. This case report describes a diabetic patient admitted with dyspnea one week after she suffered an acute myocardial infarction. The objective of this report is to draw attention to the differential diagnosis of HF and COVID-19 pneumonia.

Özet– Akut kalp yetersizliği en sık görülen kardiyak acil durumlardan biridir. Kalp yetersizliğinden kaynaklanan akciğer ödemi, akciğer bilgisayarlı tomografi taramalarında eksüdatif bir hastalığı taklit edebilir. Aralık 2019'da Çin'de Coronavirus Hastalığı-2019 (COVID-19) salgını başlamış ve kısa sürede tüm dünyaya yayılmıştır. Bu pandemi döneminde, akut dispne ile başvuran hastalarda COVID-19 pnömonisini dışlama eğilimi artmış olup bu durum benzer semptomlar ile gelen akut dekompanse kalp yetersizliği olan hastaların tanı ve tedavisinde gecikmeye yol açabilmektedir. Bu yazıda, akut miyokart enfarktüsü geçirmesinden bir hafta sonra dispne ile acil servise başvuran diyabetik bir hastayı sunuyoruz. Amacımız kalp yetersizliği ve COVID-19 pnömonisinde ayırıcı tanıya dikkat çekmektir.

In December 2019, the outbreak of a novel coronavirus was identified in China and coronavirus disease 2019 (COVID-19) subsequently spread around the world.[1] COVID-19 pneumonia is now considered in the differential diagnosis of patients with dyspnea, particularly if they also have a dry cough and a fever. This may cause a delay in the diagnosis and treatment of patients with acutely decompensated heart failure (HF), who may display similar symptoms.

This case of a diabetic patient who presented with dyspnea is offered in order to draw attention to elements of the differential diagnosis of HF and COVID-19 pneumonia.

CASE REPORT

A 36-year-old type 1 diabetic woman was admitted to the clinic with a dry cough and shortness of breath. She had undergone primary percutaneous intervention for an acute inferior myocardial infarction (MI) 7 days prior and had been discharged with prescriptions for dual antiplatelet therapy, an angiotensin-converting enzyme inhibitor, a beta-blocker, and a statin.

On admission, the patient had a subfebrile body temperature (38°C) and exhibited tachypnea (28 bpm). Other findings were a blood pressure of 95/60

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Correspondence: Dr. Beste Özben. Marmara Üniversitesi Tıp Fakültesi, Kardiyoloji Anabilim Dalı, Maltepe, İstanbul, Turkey.
Tel: +90 216 - 421 22 22 e-mail: besteozben@yahoo.com

mmHg, pulse rate of 90/min, jugular venous pressure of 6 cm H_2O, and an oxygen saturation rate of 89%. Bibasilar crackles, a third heart sound with a soft, early systolic murmur, and bilateral pretibial edema were observed. An electrocardiogram demonstrated a sinus rhythm with fragmented QRS at the DIII and aVF derivations. Echocardiography revealed regional wall motion abnormalities (inferior septum, inferior, inferolateral, and posterior walls were akinetic) with a left ventricular ejection fraction (LVEF) of 40%, moderate mitral regurgitation, a normal left atrium size, and mild tricuspid regurgitation (systolic pulmonary artery pressure: 40 mmHg). Coronary angiography performed 7 days earlier showed successful revascularization of the circumflex artery. The patient was hospitalized with a preliminary diagnosis of decompensated HF and given intravenous diuretics in addition to her recently prescribed medical treatment.

Blood tests revealed an elevated level of high-sensitivity C-reactive protein (hs-CRP:17.4 mg/L), high-sensitivity cardiac troponin T (hs-cTnT:1.55 ng/mL), N-terminal pro-brain natriuretic peptide (NT-proBNP: 2937 ng/L), and a normal white blood cell count (neutrophil: 6600/μL, lymphocyte: 1700/μL). Other biochemistry test results were normal.

A chest X-ray revealed bilateral pleural effusion and opacities with a normal cardiothoracic ratio (Fig. 1a). Since the COVID-19 outbreak was ongoing and the patient had a subfebrile temperature, dry cough, dyspnea, elevated hs-CRP, and opacities mimicking infiltration visible on the chest X-ray, the infectious diseases department was consulted. A nasopharyngeal swap sample was taken for COVID-19 testing. A chest computed tomography (CT) scan revealed bilateral, widespread, ground-glass opacities and consolidation that could be compatible with viral pneumonia (Fig. 2a, b; Video 1[*]). There was also evidence of cardiomegaly, bilateral effusion, an increase in reticular density, and interseptal thickening, which could be attributed to decompensated HF. As a result of uncertainty in the differential diagnosis, COVID-19 treatment was planned if polymerase chain reaction (PCR) test results were positive. Empirical oral oseltamivir treatment (75 mg twice daily for 5 days) was administered since viral pneumonia could not be excluded.

Abbreviations:

COVID-19	*Coronavirus disease 2019*
CT	*Computed tomography*
HF	*Heart failure*
hs-CRP	*High-sensitivity C-reactive protein*
hs-cTnT	*High-sensitivity cardiac troponin T*
LVEF	*Left ventricular ejection fraction*
MI	*Myocardial infarction*
NT-proBNP	*N-terminal pro-brain natriuretic peptide*
PCR	*Polymerase chain reaction*

During follow-up, a negative fluid balance was maintained with intravenous furosemide. The patient's body temperature did not exceed 38°C. NT-proBNP, hs-CRP, and hs-cTnT levels decreased gradually to 952 ng/L, 6.3 mg/L, and 0.81 ng/mL, respectively. The effusion and opacities seen on the chest radiography regressed (Fig. 1b). COVID-19 PCR testing results were twice found to be negative. On the fifth day of hospitalization, no effusion or opacities were visible on a chest X-ray (Fig. 1c). The patient was discharged with optimal medical treatment recommendations for HF with a reduced LVEF.

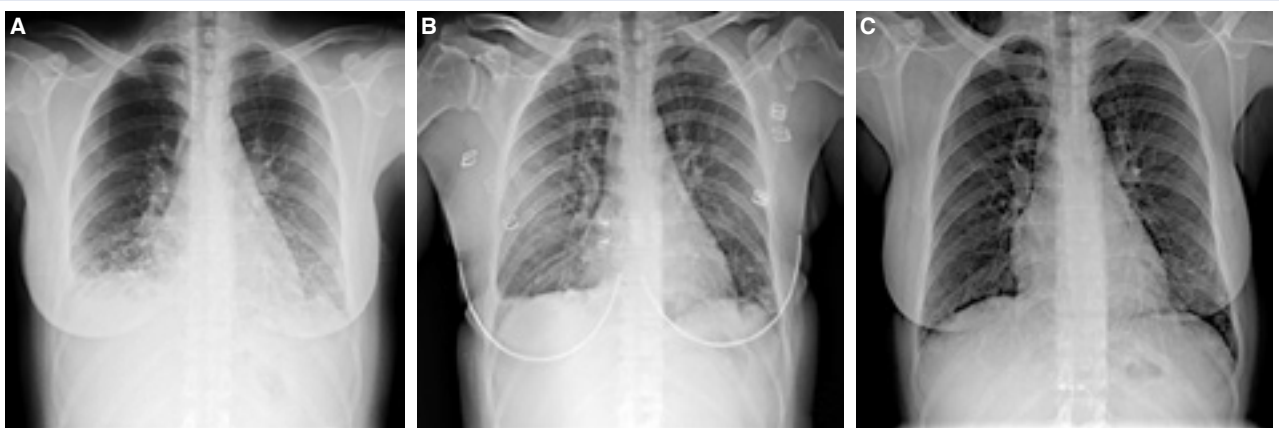

Figure 1. Chest radiography images showing **(A)** prominent, bilateral pleural effusion at the right costaphrenic sinus with central opacities on day 1 and **(B)** regression of lesions and effusion with intravenous diuretic treatment on day 3 **(C)** and day 5.

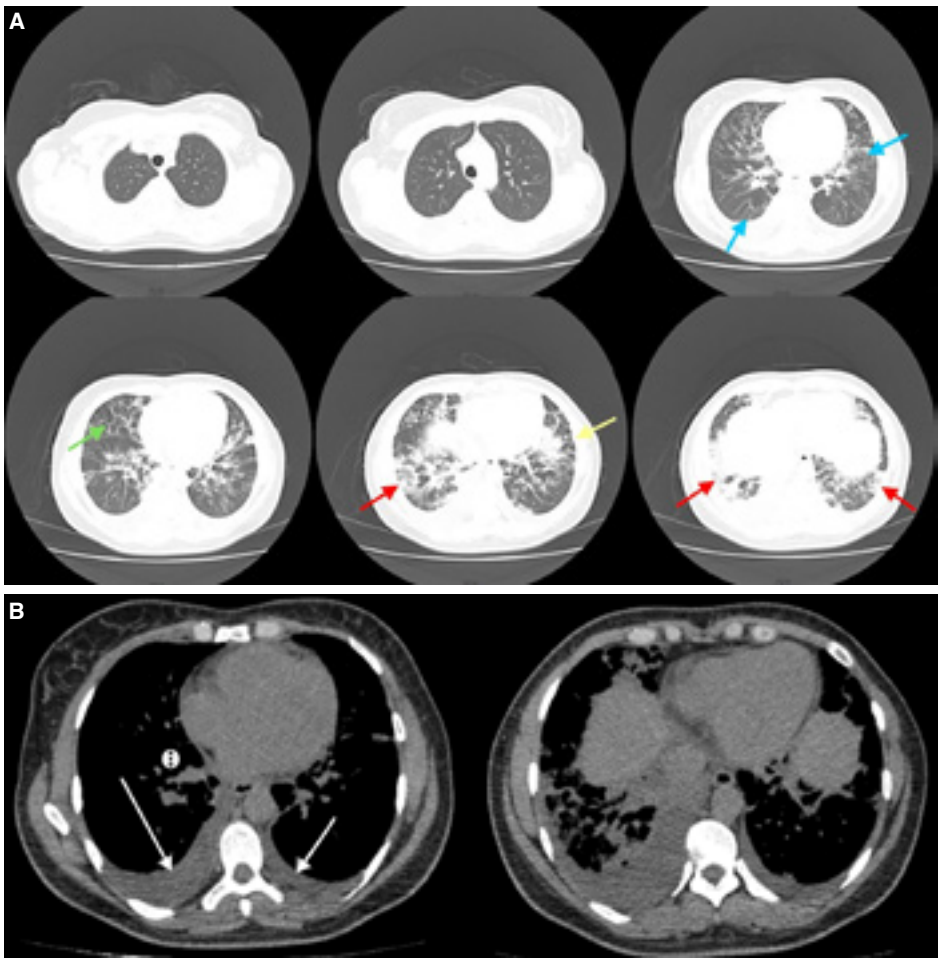

Figure 2. High-resolution computerized tomography images of **(A)** lateral, widespread, ground-glass opacities (blue arrows), septal thickening (green arrow), consolidation in lower lobes (red arrows), subpleural fibrotic bands with retraction (yellow arrow) and **(B)** pleural effusion and cardiomegaly.

DISCUSSION

Acute HF is one of the most common cardiac emergencies. Pulmonary edema caused by HF may mimic an exudative disease on a chest CT, making differentiation difficult. During this pandemic period, there is a need to exclude COVID-19 pneumonia in patients with acute dyspnea. Clinical symptoms, laboratory findings, and imaging tests are all important in the differentiation of HF from COVID-19 pneumonia (Table 1).

The primary symptoms of COVID-19 are fever (83%), cough (81%), and shortness of breath (31%), followed by a night cough, fatigue, and myalgia. These symptoms may also be present in HF, although fever is not an expected finding in HF unless there is an accompanying infection. Patients with COVID-19 pneumonia may have chest pain due to underlying pneumonia or ischemia induced by either hypoxemia or tachycardia. Oxygen desaturation and tachypnea may be observed in both diseases. However, jugular venous distension, hepatojugular reflux, third heart sound, edema, ascites, and weight gain are generally seen in HF.[2,3] Our patient had a dry cough, shortness of breath, low pulse oxygenation, and a subfebrile body temperature, all of which may be seen in both HF and COVID-19 infection. The presence of a third heart sound was interpreted as a sign of HF in our patient. She had suffered from an acute inferior MI 1 week earlier and had a depressed LVEF, moderate mitral regurgitation, and a normal left atrial size, which also suggested that she might develop acute mitral regurgitation and HF.

Table 1. Differentiation of heart failure from COVID-19 pneumonia

		Acute heart failure	COVID-19 pneumonia
Symptoms	Fever	- /+	++
	Cough	+	++
	Dyspnea	++	++
Physical examination	Tachypnea	++	++
	Tachycardia	++	++
	Rales	++	++
	S3–S4 heart sounds	+	–
	Jugular venous distention	+	–
	Hepatojugular reflux	+	–
Blood tests	C-reactive protein ↑	+	++
	D-dimer ↑	+	++
	Lymphopenia	–	+
	Troponin ↑	+	– / +
	BNP/proBNP ↑	++	– / +
Chest computed tomography	Ground glass opacity	++	++
	Consolidation	++	++
	“Crazy paving” pattern	++	++
	Septal thickening	++	+
	Bilateral involvement	++	++
	Lesion location	Central-stepwise	Peripheral
	Rounded morphology	+	++
	Cardiomegaly	++	–
	Pleural effusion	++	– / +
	Pulmonary venous congestion	++	–
	Fissural thickening	++	–
	Improvement with diuretics	++	–

COVID-19: Corona virus 2019; BNP: Brain natriuretic peptide.

The patient had elevated hs-CRP, hs-cTnT and NT-proBNP levels, all of which might be seen in HF or COVID-19 infection. However, significant elevations of hs-cTnT and NT-proBNP are more likely to occur in cases of HF. Lymphopenia and a low neutrophil/lymphocyte ratio, which seems to be specific to COVID-19 infection,[3] were not present in this patient.

Since the laboratory parameters may not provide certainty, a chest CT becomes even more important in the differential diagnosis and follow-up, although there are some overlaps between HF and COVID-19 infection in CT imaging findings.[4,5] Ground-glass opacity, consolidation, crazy-paving pattern, and septal thickening may be seen in both diseases. However, the ratio of central and stepwise distribution, peribronchovascular thickening, fissural thickening, and cardiomegaly with bilateral subpleural effusion, as in our case, is higher in HF than COVID-19.[6] Subpleural, well-circumscribed, multiple ground-glass lesions with more rounded morphology and peripheral distribution are more specific to COVID-19.

A chest X-ray may also be useful in the differentiation of HF from COVID-19 pneumonia, especially if a chest CT scan is not available. A rapid regression of lesions after diuretic treatment is consistent with HF; the regression of lesions in cases of COVID-19 takes more time and is not responsive to diuretic treatment. In our case, while waiting for the PCR results, the regression of the effusion and opacities on the chest

X-ray with diuretic treatment confirmed that they were associated with HF.

Conclusion

HF may mimic COVID-19 pneumonia, as they share some symptoms. Laboratory and imaging tests may provide help in the differential diagnosis, as well as an evaluation of the response to diuretics.

*Supplementary video file associated with this article can be found in the online version of the journal.

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