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CASE REPORT

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From thrombosis to tamponade: unveiling severe pericardial effusion in a misdiagnosis case

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Abstract

Background Anticoagulants increase the risk of cardiac tamponade in patients with pericardial effusion (PE). Therefore, inappropriate administration of them in the presence of PE can lead to a catastrophic outcome. This study presents a patient with a provisional misdiagnosis of venous thromboembolism (VTE).

Case Presentation An 83-year-old Iranian female was transferred to the emergency department of a tertiary cardiology hospital complaining of neck swelling concomitant with chest pain and dyspnea. The patient had been diagnosed with jugular vein thrombosis in another local center, and since the chief complaint was neck swelling, she underwent Doppler sonography, and the diagnosis was confirmed. Subsequently, the treatment with unfractionated heparin was started. After 5 h, considering the worsening of symptoms with the suspicious diagnosis of COVID-19 based on her symptoms and laboratory data, a chest computed tomography scan was requested, which showed a massive PE. Subsequently, transthoracic echocardiography confirmed the diagnosis. The patient was immediately transferred to the operating room and underwent pericardiotomy. The post-surgery period was uneventful, and she was discharged 5 days later.

Conclusion Patients with viral infections, specifically COVID-19, are at risk of undiagnosed severe pericardial effusions. Venous stasis in the jugular veins due to PE can mimic jugular vein thromboembolism, causing a wrong diagnosis. Since treating thrombosis can exacerbate tamponade to hemodynamic instability and collapse, sufficient investigation before starting anticoagulants is necessary.

Clinical key message Distinguishing VTE from PE is not always straightforward. Therefore, it is important to ensure physicians have reached an appropriate level of certainty about their diagnosis by performing precise diagnostics before using anticoagulants. Mismanagement with anti-thrombotics can result in catastrophic consequences. Therefore, taking an accurate history, performing a precise physical examination, and using rapid and available diagnostic modalities can avoid delays in definitive management.

Keywords Pericardial effusion, Tamponade, Thromboembolism, Misdiagnosis, Case report

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Introduction

Pericardial effusion (PE) is an accumulation of more than 50 milliliters between the visceral and parietal layers of the pericardium caused by pathologic etiologies [1]. Generally, there are two types of pathologies: inflammatory (infections, inflammations, malignancies, and cardiac ischemia) and non-inflammatory (perforating or blunt cardiac traumas, vascular structural diseases), which can cause massive PE (tamponade) [1, 2]. Administrating anticoagulants in cases of PE can cause an increase in the bleeding volume and accumulated fluid and worsening of the clinical condition, even as severe as tamponade and death [3–5]. Internal jugular vein thrombosis, which is the intraluminal formation of thrombus, is usually triggered by hereditary or acquired prothrombotic risk factors such as intravenous drug use, factor V Leiden mutation, malignancies, hormone replacement therapy, immobilization, trauma, and pregnancy [6]. The mainstay of treatment for venous thrombosis is anticoagulants with the consideration of the bleeding risk [6, 7]. Therefore, the risks and benefits of anticoagulant administration in patients with concomitant venous thrombosis and PE should be evaluated cautiously.

It has been proven that COVID-19 can trigger inflammatory pathways and result in multi-organ damage, including lung, heart, and the surrounding serosal surfaces [8]. One of the unusual but life-threatening presentations of COVID-19 is tamponade, which is defined as the acute accumulation of liquid inside the pericardium, causing elevation of the intrapericardial pressure, impairment of filling during the diastole and declining output, which results in shock [9–11]. Although rarely reported, COVID-19 can present with isolated signs and symptoms of PE; however, it might be accompanied by other symptoms of this infection [8, 12]. The hallmark triad of tamponade diagnosis (hypotension with a narrowed pulse pressure, Jugular venous distention (JVD), and muffled heart sounds) have mutual presentations with JVT, mainly bulged jugular vein in the neck [13–15]. Therefore, there is a high risk of being misdiagnosed and mismanaged this infection, which can result in adverse outcomes and deterioration of patients' clinical condition [15, 16].

Herein, we report an educational case of massive PE secondary to COVID-19, which was misdiagnosed with venous stasis, and the wrong treatment made the condition more complicated.

Case presentation

History and physical examination

An 82-year-old Iranian woman was transferred by emergency medical service to the emergency department of a tertiary cardiology hospital, complaining of neck swelling accompanied by chest pain and shortness of breath compatible with functional class New York Heart Association IV. The symptoms had started less than 24 h ago and were exacerbated by the time. Due to the right-sided neck swelling, a Doppler ultrasonography (US) was performed in another center before her transfer, which revealed the right JVT. Since the chief complaint of the patient was neck swelling, she underwent Doppler sonography in the hospital, and the diagnosis was confirmed. Therefore, the treatment with unfractionated heparin was started. She did not mention any symptoms of infection or pneumonia, such as fever, cough, or dyspnea. The patient declared no remarkable past medical history.

On her physical examinations, she was an anxious, discomfortable, and frail woman using abdominal muscles for respiration. The vital sign measurement demonstrated a blood pressure of 100/60 mmHg, a heart rate of 70 beats/min, and a temperature of 37.1℃. Her oxygen saturation on ambient air was 85%. During her head and neck examination, the swelling and erythema were obvious on the right side of the neck. Cardiopulmonary examination showed no other remarkable signs or symptoms. The Wells' Score of the patient was calculated, which was −1 (+1 for "collateral (non-varicose) superficial veins present" and −2 points for "Alternative diagnosis to deep vein thrombosis as likely or more likely").

Diagnostic and therapeutic procedures

Considering the neck swelling and the previous ultrasonographic diagnosis of jugular vein thrombosis, treatment with unfractionated heparin was continued for 5 h. The patient's electrocardiogram revealed low-voltage QRS complexes in limb leads and T wave inversions in precordial leads V1 to V3 (Fig. 1). Laboratory data were also requested, which revealed remarkably elevated levels of C-reactive protein (90 µg/ml), normal levels of high-sensitivity cardiac troponin I, and a D-Dimer of 1.1 µg/d (Table 1). A nasopharyngeal swab was positive for severe acute respiratory syndrome coronavirus-2 by reverse transcription polymerase chain reaction.

Since the event occurred during the COVID-19 pandemic and the patient's laboratory data with the suspicion of concomitant COVID-19 infection, a chest CT scan was performed, which showed massive PE without substantial evidence of interstitial involvement of lungs by COVID-19 (Figs. 2 and 3). The bedside TTE also confirmed the presence of Tamponade and right ventricle collapse with abnormal tricuspid annular plane systolic excursion (TAPSE) and Reduced left ventricular ejection fraction (LVEF) due to the compressive effect of fluid accumulation (Fig. 4).

The patient's hemodynamic condition gradually deteriorated, leading to instability. The systolic BP declined to 80mmHg, Oxygen saturation was 75% despite respiration via a mask, and the respiratory rate was increased

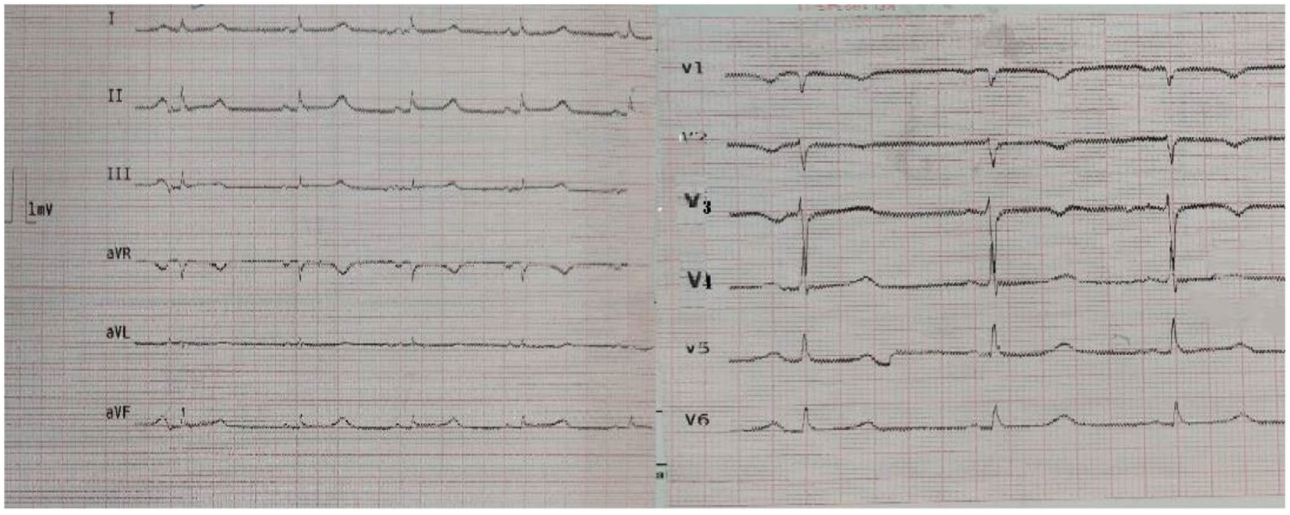

Fig. 1 Electrocardiography demonstrating low voltage QRS complex in limb leads and T wave inversions in precordial leads V1 to V3

Table 1 Comparison of key laboratory values upon admission and at discharge

Test	Result		Reference range
RBC (10^6/µl)	5.3	5.9	4.2–5.5
Hemoglobin (gr/dL)	12.1	12.4	12–16
WBC (per µl)	6.600	10,200	4.000–11.000
MCV (fL)	75	77	80–99
Hematocrit (%)	40	43	37–47
Platelet (per µl)*10^3	207	201	150–400
MCH (pg/cell)	23	25	27–31
MCHC (g/dL)	30	34	32–36
PT	16.2	15	11–13 s
PTT	38	28	25–38 s
INR	1.3	1.2	1-1.5
CK-MB	Undetectable	Undetectable	< 25u/l
Troponin I (Second sample in 6 h)	Negative	N/A	Negative
ESR (Millimeters per hour)	110	56	0–20 (Millimeters per hour)
CRP	94	25	< 4

Abbreviations: RBC: Red blood cell, WBC: White blood cell, MCV: Mean corpuscular volume, MCH: Mean corpuscular hemoglobin, MCHC: Mean corpuscular hemoglobin concentration, ESR: Erythrocyte sedimentation rate, CRP: C-reactive protein

to 35 per minute. Considering the suspicious diagnosis of the tamponade, an emergency cardiac surgeon consult was requested. Due to the availability of an expert cardiac surgery team, after confirming the presence of tamponade, the patient was transferred to the operation room immediately. She underwent pericardiotomy, a subxiphoid pericardial window was fixed, and about 1300 cc of bloody turbid fluid was drained and sent for more cellular and immunohistochemistry evaluation. The evaluation of the fluid showed a completely bloody fluid

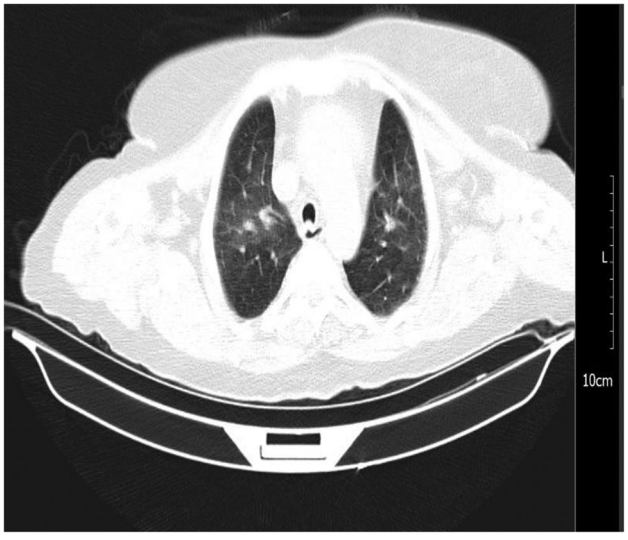

Fig. 2 A chest computed tomography scan showed no parenchymal involvement in favor of COVID-19

with numerous red blood cells and some reactive mesothelial cells with completely bloody fluid with numerous red blood cells and some reactive mesothelial cells RBCs. The pericardial fluid culture was negative for bacteria. A biopsy of the pericardium was taken, which was reported as a patchy lymphocyte infiltration with no evidence of malignancy. The patient was then transferred to the cardiology intensive care unit post-surgery.

Conclusions and follow-up

Jugular and subclavian Doppler US were repeated on the first postoperative day, and no evidence of thrombosis was found. Post-procedural TTE revealed a preserved left ventricular ejection fraction with no significant valvular disease concomitant and mild PE lateral to the left ventricular (8 mm). Besides, no signs of respiratory

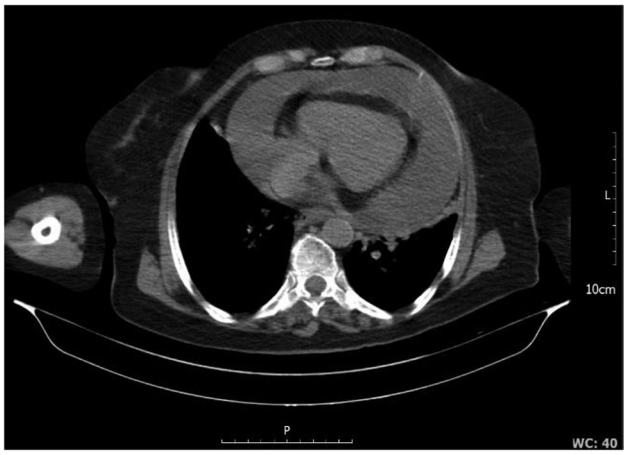

Fig. 3 A chest computed tomography scan showed massive circumferential pericardial effusion

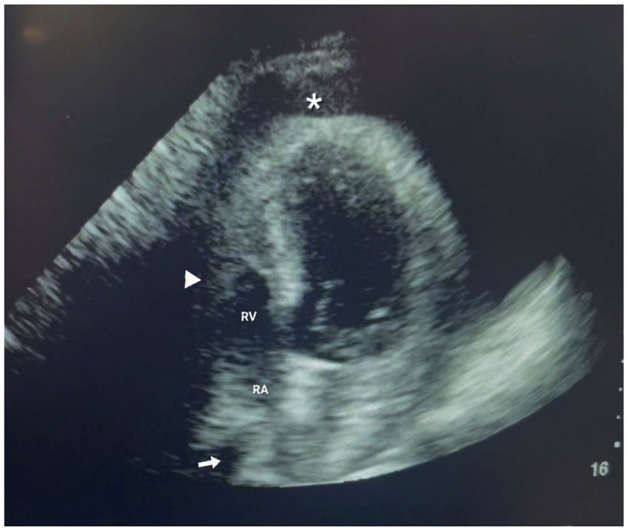

Fig. 4 Transthoracic echocardiogram (4-chamber view) revealed a massive circumferential pericardial effusion (*) accompanied by RA invagination (arrow) and RV collapse (arrowhead). RA: right atrium, RV: right ventricle

collapse were detected. The patient's post-surgery recovery time was uneventful. She was transferred to the cardiology ward two days after the surgery and discharged with appropriate medical conditions 4 days later. One-, six- and 24-month follow-up visits showed no evidence of new complaints or recurrence of symptoms, and follow-up TTE revealed no remarkable abnormal findings. Figure 5 shows the timeline of the events.

Discussion

In this study, we presented a case of severe pericardial effusion, probably induced by COVID-19 infection, which was mistakenly diagnosed with jugular vein thrombosis. After performing a chest CT scan to rule out COVID-19, the tamponade was detected, and considering the critical clinical condition, the patient was immediately transferred to the cardiac operation room, and the removal of fluid was conducted successfully. This case underscores the importance of avoiding premature decision-making in critical conditions and not starting an invasive treatment (anticoagulants, in the presented case) before sufficient evidence for the presumed diagnosis. The continuation of this therapeutic medication could be accompanied by hemodynamic collapse and the patient's death.

COVID-19 may activate cascades of inflammatory pathways, like other viral infections, that could damage multiple organs, such as the heart, lungs, and surrounding serosal surfaces. When COVID-19 triggers this inflammatory response, it is plausible that it could cause pericarditis and pericardial effusion, much like other viral infections [8]. There is a wide range of presentations of COVID-19, from mild flu-like symptoms to severe fatal cardiopulmonary symptoms [17, 18]. It is reported that almost 12% of COVID-19 patients have experienced cardiovascular complications, which have a significantly higher risk of mortality [19]. Although rare, severe pericardial effusion can be the first presentation of COVID-19 [20]. The inflammation caused by the spread of viral infection can involve the heart and pericardium structures. This inflammation mostly presents with myocardial injury, arrhythmia, and myocarditis, while in a lower number of patients, it is initiated with the production of pericardial fluid, resulting in exudative pericardial effusion [21]. The severity of the presentation depends on the volume of fluid accumulated in the pericardial sac [22]. The pattern of symptom progression in the presented patient is aligned with these previous findings. While the initial symptoms were tolerable and less severe, fluid accumulation had been accelerated with the infusion of heparin, resulting in hemodynamic instability.

Evidence from two prospective and observational trials has shown that almost one-fifth of severe COVID-19 infections have been associated with various degrees of pericardial effusion [23, 24]. Moreover, more severe COVID-19 is reported to be associated with a higher risk and more voluminous PE. In a study that had almost 23% death in patients with COVID-19 and had undergone transesophageal echocardiography, it was shown that the mortality rate among those with PE was significantly higher than those who did not have PE (33% vs. 15.8%). In this study, the prevalence of PE across all COVID-19 patients was 14%. Moreover, those with COVID-19 had higher mortality rates and worse right ventricular function [25]. Some cardiac markers, including increased B-type natriuretic peptide and echocardiographic parameters, have shown prognostic value in these patients. For example, reduced LVEF and abnormal TAPSE have demonstrated the potential correlation with higher rates of

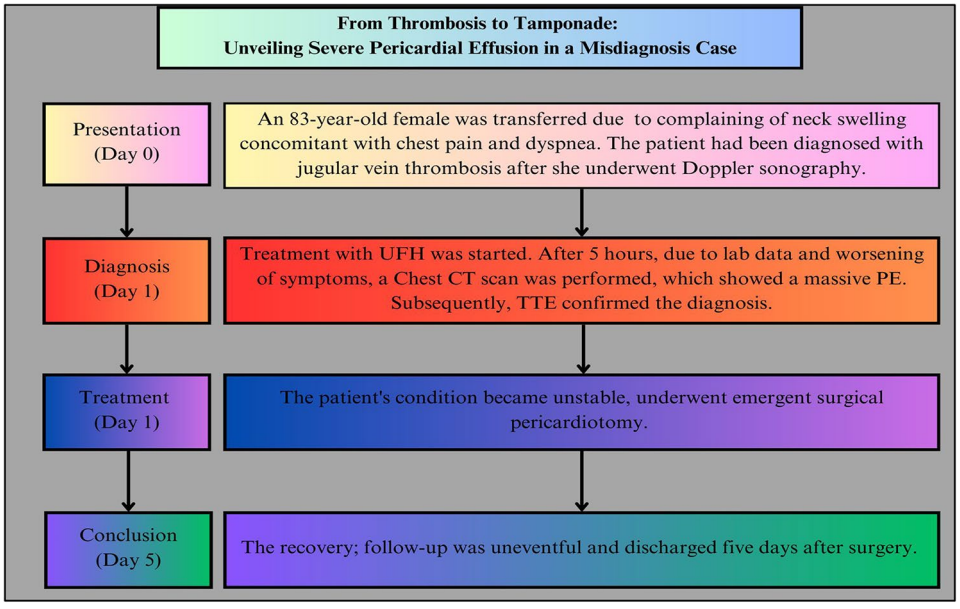

Fig. 5 Events timeline

mortality and worse clinical outcomes in these patients. In a large prospective study of hospitalized COVID-19 patients, PE is frequently observed, though it is rarely linked to acute myocarditis or pericarditis. However, it is associated with impaired myocardial function and increased mortality. Therefore, limited early echocardiography, including measurements of LVEF, TAPSE, and the presence of PE, is adequate and effective for risk assessment [26]. One of the interesting points observed in the presented case was severe PE in a patient without significant involvement of the pulmonary system and general symptoms of COVID-19. Therefore, tamponade symptoms should be considered one of the rare but possible presentations of COVID-19 despite the absence of other typical signs and symptoms of viral infection.

JVT, which is an obstruction or narrowing of the jugular vein caused by thromboembolic events, is mainly seen in patients with risk factors such as Virchow's triad: (1) hypercoagulability, (2) stasis or turbulence in blood flow, and (3) endothelial dysfunction or catheter placement in these vessels [27]. However, it might be seen unexpectedly in patients without significant risk factors. Although most patients are asymptomatic, symptomatic patients mainly come with bulging of the left lateral neck, erythema, tenderness, and warmth [28]. Based on the European Society for Vascular Surgery (ESVS) 2021 Clinical Practice Guidelines in the acute phase JVT, low molecular weight Heparin and unfractionated heparin are most commonly used [29, 30]. The medication used for this condition can exacerbate pericardial fluid accumulation. Therefore, it is crucial to have sufficient confidence in the diagnosis to initiate and continue the infusion.

The similarity between the typical symptoms of these two conditions, mainly neck swelling, can be misguiding. The main diagnostic evaluations are D-dimer measurement, which is very sensitive but nonspecific, compression Doppler US of the lateral neck (JVjugular vein), neck CT scan, and venography, which is the gold standard of the diagnosis. Since venography is a relatively invasive procedure, the diagnosis is mainly made by considering the combination of clinical findings, laboratory data, and Doppler US, which has 96% sensitivity and 93% specificity [28, 29]. The pitfall of the Doppler US is being operator-dependent, which can result in misdiagnosis and misinterpretation if other differential diagnoses are ignored [31]. In a study that evaluated cases of false positive thrombosis in Doppler sonography, chronic diseases, weak ultrasound signaling, and artifacts in ultrasound signaling secondary to obesity were accounted as leading causes of false positive reports of thrombosis [16]. It has also been reported that compression of veins in the distal part of the evaluated area can result in false results of venous thrombosis, which should be interpreted attentively [32]. In our patient, the same condition caused the false positive result of the Doppler ultrasonography. The obstruction of the outlet of the jugular vein caused stasis, and the resolution of the stasis resolved the misleading appearance of venous thrombosis. We have provided similar cases of misdiagnosed COVID-19 infections that are available in the literature review (Table 2).

Clinical learning point

COVID-19 can have multiple unrelated presentations, and this can cause misdiagnosis and mismanagement. Especially when there is suspicion to diagnose the

Table 2 Iatrogenic severe pericardial effusion literature review

Article and authors	Age and gender	Presentation	Treatment/progression or remission of the disease
Subacute hemorrhagic pericardial tamponade after COVID-19 infection mimicking carcinomatous pericarditis: a case report, Yamamoto H. et al., 2024 [33]	64-year-old male	CC: Dyspnea due to COVID-19 PMH: DM, CKD, PH/EX: High bp and temp, left great and second toes revealed ulcers with pus, swelling, and surrounding erythema. LAB data: suggestive of infection, but nasopharyngeal COVID-19 was negative. Became positive on the third day. Provisional Dx: Acute osteomyelitis of the toes. ECG: sinus rhythm and concave ST-segment elevation in precordial leads, suggestive of early repolarization TTE: LVH and LA compression	Initial TX: Ampicillin-sulbactam On the 13th day, the condition became worse, and dyspnea, orthopnea, elevated JVP, and lower limb edema. CECT (day 17): revealed moderate pericardial and bilateral PE RHC: PT + equalization of diastolic pressures across all chambers and marked hemodynamic pulsus paradoxus Tx: urgent pericardiocentesis with placement of pericardial drainage, showing 750 ml of hemorrhagic exudate fluid Pericardial fluid (PF) analysis showed hypercytokinemia consistent with an inflammatory process
Cardiac tamponade due to apixaban therapy in a patient with unknown pericardial hemangioma Sbrana F et al., 2018 [34]	78-year-old Female	CC: SOB + Palpitation + tachycardia and skin pallor. due to PE after DVT◊addmitted worsening of resting SOB. PMH: recent pulmonary embolism secondary to deep VTE + arterial HTN CXR: increased cardiac shadow size ECG: Reduced amplitude + 110 bpm TEE: massive PE, LAB: Declined Hb 2 gr/dl	VTE TX: 25 days with enoxaparin 8000, then LMWH, started apixaban TX of massive PE: pericardiocentesis ◊700 ml bloody drainage◊ Subsequent improvement in the patient's clinical conditions. Apixaban discontinued◊ low dose of enoxaparin (4000 UI s.c. BD) started, + colchicine (1 tab OD) + methylprednisolone◊ After 1 month, the chest X-ray study showed a normal heart silhouette in the presence of a minimal PE
Cardiac tamponade after thrombectomy Sivakumar S.et. al. 2024 [35]	88-year-old-Fe	CC: SOB + weakness, PH/EX: HR: 108, O2 sat: 80◊98 with 4lit/min O2 canola Bedside US: Dshape RV like LV LAB: Elevated trop and Pro-BNP ECG: S1Q3T3 CECT: Saddle-shaped PE extended to both PA DUS: Lower extremity VTE to common femoral and then popliteal. Heparin infusion started, delayed ATT due to colon cancer + Mechanical thrombectomy	Two Hours after MT◊severe back pain and declined BP and HB declined to 8.8 g/dl. TEE: massive PE◊ pericardiocenthesis + pericardial window + rapid transfusion emergent bedside thoracotomy ACLS was done due to no heart beat◊ caused death.
Primary cardiac angiosarcoma masquerading as intracardiac thrombus, Villano N. rt. Al. 2020 [36]	49-year-old male	CC: Bloody stool and SOB PMH: Recent atrial tachycardia and intracardiac thrombus DMII and HTN + mobile, multilobulated mass in the RA PDH: Warfarin + antidiabetics and anti-HTN Enteroscopy: Target lesion with a friable ulcer in the gastric body CMRI: primary cardiac angiosarcoma (PCAS)	DX: PE + Tamponade TX: Pericardiocentesis with 260 mL of bloody output Next day TEE: A large mass in RA 4.0 × 2.3 cm, encasing SVC with a fixed, non-mobile component and a mobile oscillating component < 2.8

Table 2 (continued)

Article and authors	Age and gender	Presentation	Treatment/progression or remission of the disease
Cardiac tamponade as a presentation of COVID-19 after cardiac surgery Gopal et al. 2021 [37]	40-year-old male	CC: Presented with CAD symptoms PMH: CAD + underwent OPCAB 5-day post-op: TTE: moderate PE + early signs of tamponade and global biventricular dysfunction ECG: concave upwards modest ST elevation all leads and reciprocal ST depression and PR elevation in aVR	TX: NPO for anesthesia but gradually more severe SOB◊ ICU; during preparation for OR, he was arrested →Rsuscitation was fruitless, and the chest was opened. The fluid was drained, and the internal massage was successful The heart was inflamed and warm◊ COVID OCR was sent◊ became + ◊ LAB: very high inflammatory (D-dimer, CRP, ESR) markers◊ multiorgan dysfunction◊ death after 36 h
Huge Pericardial Effusion in Neglected Adolescent with Parachute Mitral Valve — A Case Study Abdou AM [35]	13-year-old male	CC: Dyspneic, Tachycardic, HR:140, RR:25, O2Sat: 75% BP: 90/40 T: Low-grade fever, bad capillary refill, pulsus paradoxus, hypotension, engorged neck veins vein. CP PH/EX: Rib deformity and crackles decrease air entry. Galloping heart sound Apex: 7th space outside midclavicular line, pansystolic murmur V/VI, accentuated 2nd heart sound. The liver is tender with sharp borders, Mild abdominal pain with no tenderness, no scrotal swelling, nor lower limb edema; ECG: sinus tachycardia:	DX: neglected CHD + long-standing cardiomegaly TTE: Large PE + collapse of RA (near the peak of the R wave), Long RA collapse duration + LA compression DX: Huge PE with fibrin threads and suspected intracardiac thrombus Coronary dilatation + Lasix, Aldactone, digoxin, PPI, oxygen, + tapping of pericardial fluid LAB: The revealed multisystem inflammatory response was confirmed by modifying the management plain. Glucocorticoids, low-dose aspirin, anti-factor 10, and IVIG were added to the previous management. The patient stabilized with medication w/o Tapping In 3-week FU: NYHA class II, after three weeks of intensive care management, the patient was referred to surgery for surgical cardiac intervention.

ACLS: Advanced Cardiac Life Support, ATT: Antithrombotic Therapy, CAD: Coronary Artery Disease, CC: Chief Complaint, CECT: Contrast-Enhanced Computed Tomography, CHD: Congenital Heart Disease, CRP: C-Reactive Protein, CXR: Chest X-Ray, DUS: Doppler Ultrasound, DVT: Deep Vein Thrombosis, Dx: Diagnosis, ESR: Erythrocyte Sedimentation Rate, Hb: Hemoglobin, HR: Heart Rate, HTN: Hypertension, IVIG: Intravenous Immunoglobulin, LA: Left Atrium, LAB: Laboratory, LMWH: Low-Molecular-Weight Heparin, LV: Left Ventricle, LVH: Left Ventricular Hypertrophy, MT: Mechanical Thrombectomy, NYHA: New York Heart Association, OCR: COVID-19 Reverse Transcriptase Polymerase Chain Reaction, OD: Once Daily, O2 Sat: Oxygen Saturation, OPCAB: Off-Pump Coronary Artery Bypass, PA: Pulmonary Artery, PE: Pericardial Effusion / Pulmonary Embolism (context-dependent), PF: Pericardial Fluid, PH/EX: Physical Examination, PMH: Past Medical History, PPI: Proton Pump Inhibitor, Pro-BNP: Pro-Brain Natriuretic Peptide, RA: Right Atrium, RHC: Right Heart Catheterization, RV: Right Ventricle, SOB: Shortness of Breath, SVC: Superior Vena Cava, TEE: Transesophageal Echocardiography, Trop: Troponin, TTE: Transthoracic Echocardiography, Tx: Treatment, UI s.c. BD: International Units Subcutaneous Twice Daily, US: Ultrasound, VTE: Venous Thromboembolism

thromboembolic events and anticoagulants are aimed to be administered. Patients with neck swelling and dyspnea should be investigated meticulously by considering an overall picture of signs, symptoms, and laboratory and radiologic evidence. Although JVT and pulmonary thromboembolism are a probable combination of etiologies that can result in the typical symptoms, other life-threatening possible causes, such as pericardial effusion, should not be excluded or ignored prematurely and without sufficient diagnostic evidence. The wrong treatment for the first scenario (with anticoagulants) can lead to catastrophic exacerbation of PE, hemodynamic collapse, and death.

Conclusion (clinical learning point)

Patients with viral infections, specifically COVID-19, are at risk of undiagnosed severe PE. Due to PE, venous stasis in the jugular veins can mimic venous thrombosis, causing a wrong diagnosis. We should make sure that the diagnosis is accurate enough to prevent catastrophic events such as tamponade and cardiovascular system collapse.

Abbreviations

EMS	Emergency medical service
ED	Emergency department
SOB	Shortness of breath
NYHA	New york heart association
DUS	Doppler ultrasonography
PFH	Past family history
PMH	Past medical history
BP	Blood pressure
HR	Heart rate
CRP	C-reactive protein
hsTI	High-sensitivity cardiac troponin I
SARS-CoV-2	Severe acute respiratory syndrome coronavirus-2
TTE	Transthoracic echocardiogram
CT	Computed tomography
PE	Pericardial effusion
LDH	Lactate dehydrogenase
IHC	Immunohistochemistry
CCU	Cardiac care unit
FU	Follow-Up
JVP	Jugular vein pressure
DVT	Deep Vein Thrombosis
JVT	Jugular vein thrombosis
PTE	Pulmonary thromboembolism

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Author contributions
A.B., M.S.N., R.N., and T.A. contributed to the Conceptualization, Resource data curation and analysis, project administration, and writing of the initial draft. A.B., P.E., P.R., and T.A. contributed to the supervision, validation, visualization, investigation, methodology, software, and revision of the final draft of the manuscript. All authors read and approved the final manuscript.

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Data availability
Data is available on reasonable request due to privacy/ethical restrictions.

Declarations

Ethics approval
The study was performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments. Considering not including any revealed information about the patient and the breach of confidentiality, the committee waived the requirement for an ethics code.

Consent to participate
Written informed consent was obtained from the patient.

Consent for publication
Written informed consent was obtained from the patient for publication of this study and accompanying images.

Competing interests
The authors declare no competing interests.

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