Annals of Clinical and Medical Case Reports

Case Report ISSN 2639-8109 | Volume 11

Anchoring Bias in the Era of Covid-19 Pandemia

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Received: 10 June 2023
Accepted: 31 July 2023
Published: 07 Aug 2023
J Short Name: ACMCR

Keywords:

Mitral valve insufficiency; Covid-19; Dyspnea; Diagnostic errors; Bias; Case report

Citation:

Crimi E, Anchoring Bias in the Era of Covid-19 Pandemia. Ann Clin Med Case Rep. 2023; V11(3): 1-6

1. Abstract

1.1. Background: Covid-19 has become the culprit for many cases of acute respiratory failure. However, some cardiogenic pathologies such as acute mitral regurgitation can present with similar clinical scenario. Mitral valve insufficiency is a known cause of respiratory failure, and if not diagnosed quickly, can lead to increased mortality.

1.2. Case Summary: We discuss a case of a 62-year-old female with rapidly decompensating acute respiratory failure. The patient presented to the hospital with shortness of breath and chest pain leading to her misdiagnosis and treatment for Covid-19. She was later found on transesophageal echocardiogram to have acute severe mitral regurgitation secondary to a flail posterior leaflet from a torn chordae tendineae. Treatment for the acute mitral regurgitation was ultimately delayed due to the initial misdiagnosis. After final discovery of the true etiology behind her rapid decompensation, the patient underwent surgical repair of the mitral valve and made a full recovery.

1.3. Conclusion: This case demonstrates the importance of identifying other causes of respiratory failure and recognizing anchoring bias to prevent diagnostic errors.

2. Core Tip

Some cardiac pathologies such as acute severe mitral regurgitation can be misdiagnosed for Covid-19 pneumonia. Failure to carefully consider alternative diagnoses during a pandemic can result in misdiagnosis. We discuss a case of rapidly decompensating acute respiratory failure in a patient, initially treated for Covid-19, who was later found to have acute severe mitral regurgitation secondary to a flail posterior leaflet from a torn chordae tendineae. Awareness to the anchoring bias that took place here is necessary to prevent future misdiagnosis and delays in life-saving treatment, especially during the Covid-19 pandemic.

3. Introduction

Severe acute respiratory syndrome coronavirus 2 (SARS-Cov-2) 2019 (Covid-19) may cause lung injury with respiratory failure requiring admission to intensive care unit (ICU) and leading to high mortality in patients over the age of 60 with comorbidities such as obesity, diabetes mellitus and cardiovascular diseases [1]. Management for Covid-19 pneumonia is mainly supportive, ranging from non-invasive oxygen supplementation to invasive mechanical ventilation [1]. Extracorporeal membrane oxygenation (ECMO) can be the last resort to provide artificial lung support [2]. Acute mitral regurgitation (MR) is a common cause of pulmonary edema leading to severe respiratory failure. The typical presentation of acute MR can have a sudden onset and include dyspnea, chest pain that radiates to the neck, weakness, dizziness, and signs of cardiogenic shock. Chronic MR on the other hand can remain asymptomatic for years due to ventricular remodeling. Because of continuous left

ventricular dilation and eventual decrease in ejection fraction, it can present with fatigue, dyspnea on exertion, or even syncope and pericardial effusion [3]. Physical examination findings suggestive of MR can be subtle, and the overall clinical presentation can be mistaken for pneumonia, sepsis, or nonvalvular heart failure [4,5]. Delays in the diagnosis and surgical intervention of severe MR can carry an in-hospital mortality of almost 80% [5]. Here we discuss a case of a patient presenting with dyspnea and chest pain, in which the diagnosis of acute MR was delayed as a consequence of the Covid-19 pandemic.

4. Case Presentation

A 62-year-old female presented to an outside hospital with shortness of breath and chest pain that started earlier that morning. The symptoms began with spasms in the neck and throat followed by chest pain radiating to the upper back. The dyspnea and chest pain were worsened by the supine position and relieved by sitting upright. She denied any fever, cough, loss of taste, loss of smell, abdominal pain, or diarrhea. She reported to be a health care worker with recent exposure to Covid-19. Past medical history included supraventricular tachycardia and hypothyroidism. Home medications included Levothyroxine, Liothyronine, and Metoprolol. A prior cardiac catheterization showed 25% stenosis of the left anterior descending artery. In the emergency department, the patient had a pulse of 114 beats per minute (bpm) and an oxygen saturation of 90% on room air (Table 1). On physical exam, patient was awake, alert, in no acute distress. Coarse rhonchi were noted on auscultation of the lungs bilaterally. Cardiovascular exam revealed a regular rate and rhythm, no rubs, capillary refill was not delayed. Electrocardiogram (EKG) showed normal sinus rhythm with no acute ischemic changes, no ST-elevated myocardial infarction, and a normal QRS complex (Figure 1).

Initial laboratory investigations were significant for elevated D-dimer and mildly elevated troponin (Table 1). The rapid Covid-19 tests performed on the first and second day of admission were negative; a polymerase chain reaction (PCR) Covid-19 test was also sent on the day of admission. Chest x-ray showed right lung alveolar infiltrates and bilateral perihilar interstitial prominence without consolidation (Figure 2A). Computed Tomography (CT) of the chest revealed severe bilateral infiltrates with small bilateral effusions (Figure 2B). CT abdomen and pelvis showed a normal appearance of the abdominal aorta, no aneurysm or dissection. Pneumonia was highly suspected and initial medical treatment included intravenous dexamethasone and azithromycin. In the setting of elevated troponins with a normal EKG, cardiology was consulted for non-ST-elevated myocardial infarction, the patient was started on heparin, and a transthoracic echocardiogram was ordered. Over the course of 4 hours, blood pressure had fallen to 70's/50's and patient was tachycardic to 110's. Oxygen saturation continued to worsen despite escalation of oxygen supplementation therapy via nasal cannula, then high flow nasal cannula, and subsequently bilevel positive airway pressure (BiPAP). Within less than 24 hours, the patient was admitted to the ICU; trachea was intubated and invasive lung protective ventilation was initiated without significant improvement in oxygen saturation (Table 1). Given the rapid deterioration and overall clinical picture suggestive of Covid-19, the patient also received remdesivir and covalescent plasma. Although echocardiogram had been ordered by this time, the immediate transfer of the patient to a facility with extracorporeal membrane oxygenation (ECMO) capability was prioritized. Thus, the patient was transferred to our hospital for initiation of veno-venous extracorporeal membrane oxygenation (VV-ECMO).

5. Final Diagnosis

Just prior to the initiation of ECMO, transesophageal echocardiogram (TEE) was finally performed and revealed flail P2 segment of the mitral valve from a torn chordae tendineae with severe mitral regurgitation and a hyperdynamic left ventricle (Figure 3).

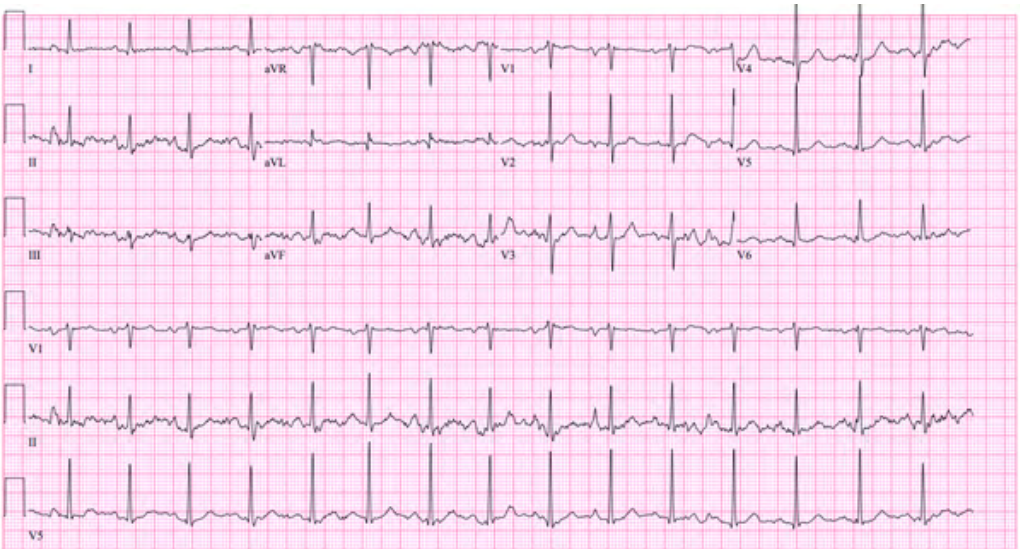

Figure 1: 12 Lead Electrocardiogram (EKG) on Admission

12 Lead EKG showing normal sinus rhythm with no ischemic changes, no ST-elevated myocardial infarction, and normal QRS.

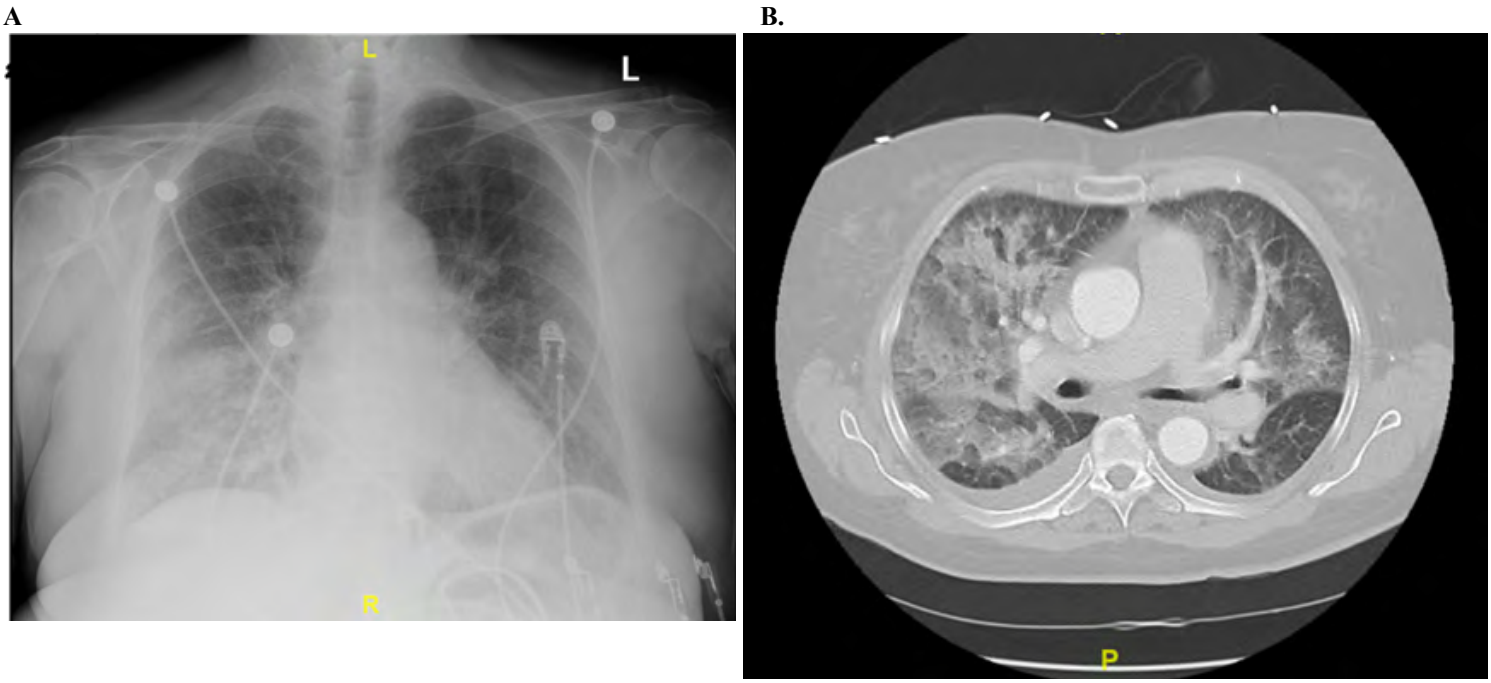

Figure 2: Chest Radiograph and Chest Computed Tomography on admission

A: Chest radiograph showing bilateral perihilar patchy infiltrates and right lower lobe consolidation.

B: Chest computed tomography (CT) showing severe bilateral infiltrates with small bilateral pleural effusions.

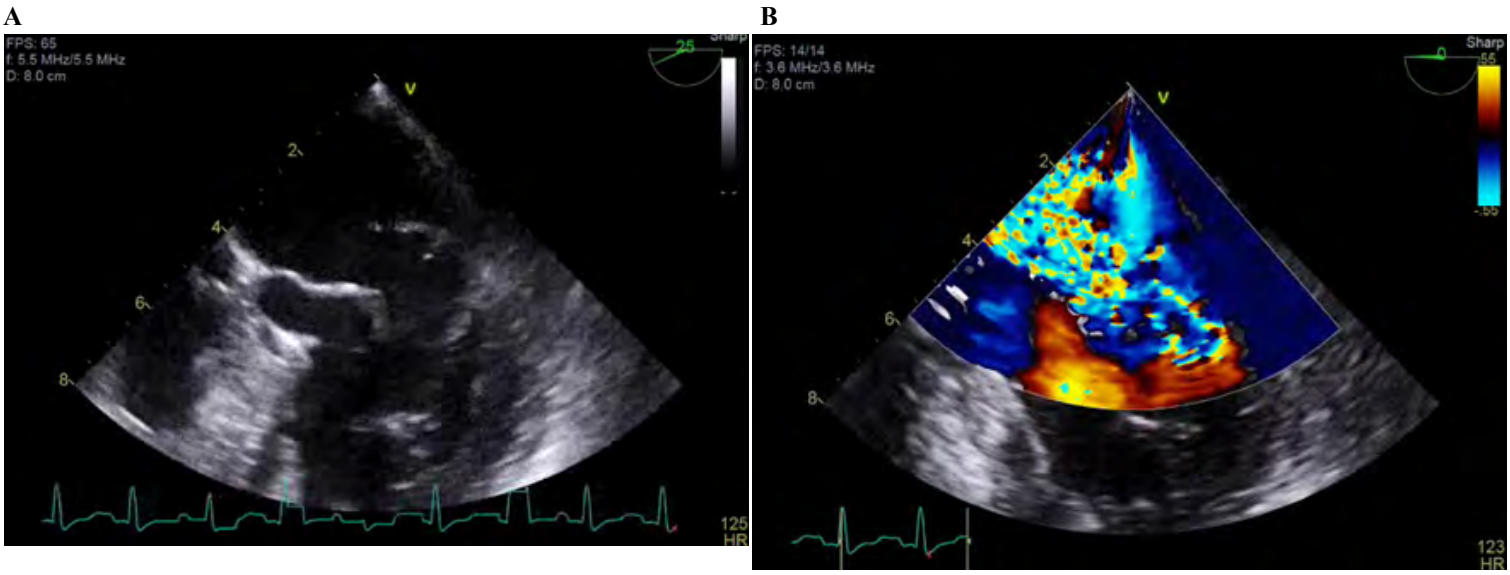

Figure 3: Mid-esophageal Four Chamber View on Transesophageal Echocardiogram

A: Mid-esophageal four chamber view showing flail posterior leaflet.

B: Color doppler midesophageal four chamber view showing anteriorly directed severe mitral valve regurgitation jet.

Table 1: Initial vital signs, laboratory studies, and oxygen supplementation

INITIAL VITAL SIGNS		
Temperature: 36.5° C	Respiratory rate: 16	
Blood pressure: 130/79	Oxygen saturation: 90%	
Pulse: 114	Oxygen flow: 3L nasal cannula	
INITIAL LABORATORY INVESTIGATIONS		
Test	Normal Range	Patient's value
Sodium	(136 - 145 mmol/L)	140
Potassium	(3.5 - 5.1 mmol/L)	4.1
Chloride	(98 - 107 mmol/L)	106

Carbon dioxide	(22 - 34 mmol/L)	26
Blood urea nitrogen	(7 - 18 mg/dL)	22H
Creatinine	(0.6 - 1.3 mg/dL)	1.1
Lactate dehydrogenase	(313 - 618 Unit/L)	476
Troponin I	(0.00- 0.034 ng/mL)	0.046 H, 0.092 H
COVID-19 nucleic acid amplification	(Negative)	Negative
D-dimer	(215.0-500.0ng/ml FEU)	2083.00 H
White blood cell count	(3.7 - 11.0 thou/mm3)	9
Hemoglobin	(11.0 - 15.4 g/dL)	13.4
Platelet Count	(150 - 440 thou/mm3)	268
Oxygen Therapy	**Oxygenation Parameters**	
Mode of Delivery	Settings	SaO2/Arterial Blood Gas
Room air (on admission)		SaO2: 90%
Nasal cannula (at 30 minutes)	3L, approximate FiO2: 33%	SaO2: 90%
High Flow Nasal Cannula (at 5 hours)	55L, FiO2: 100%	SaO2: 93%
BIPAP (at 10 hours)	IPAP 18/EPAP 14, FiO2: 100%	SaO2: 80% ; pH 7.34, PaCO2 40.6, PaO2 46, HCO3 21.9; PaO2/FiO2 ratio: 46
Mechanical Ventilation (at 18 hours)	PEEP 8, FiO2 100%	SaO2: 93%; pH 7.28, PaCO2 32.2, PaO2 76, HCO3 15.1, PaO2/FiO2 ratio: 76

*EPAP: expiratory positive airway pressure; FiO2: fraction of inspired oxygen; HCO3: Bicarbonate; IPAP: inspiratory positive airway pressure; PaCO2: partial pressure of carbon dioxide; PaO2: partial pressure of oxygen; PEEP: positive end expiratory pressure; SaO2: oxygen saturation

6. Treatment

Given the presence of profound hypoxemia, severe metabolic acidosis, and evidence of multiorgan dysfunction, the multidisciplinary ICU team decided to proceed with VV-ECMO cannulation to re-establish adequate oxygenation and to implant a left ventricular assist device to support the left ventricle. Following improvement in oxygenation and metabolic status over the next twenty-four hours, the patient underwent mitral valve replacement with bioprosthetic valve, removal of the left ventricular assist device, and VV-ECMO decannulation.

7. Outcome and Follow up

On postoperative day 1, the PCR Covid-19 test was negative. On postoperative day 4, the patient was successfully weaned from the ventilator and trachea was extubated. On postoperative day 6, a permanent pacemaker was placed for sick sinus syndrome. The patient was discharged home after a 14-day hospital stay.

8. Discussion

In this case, we report an example of anchoring bias leading to an initial misdiagnosis. Many factors oriented the diagnosis toward Covid-19: the history as a healthcare worker with Covid-19 exposure, the rapidly deteriorating respiratory status, both imaging modalities suggestive of bilateral pneumonia, and the elevated d-dimer with normal leukocytes. The suspicion for Covid-19 remained at the top of the differential despite a lack of fever, cough, the presence of normal inflammatory markers, and multiple negative Covid-19 tests.

Covid-19 infection most commonly presents with dyspnea, fever (44% of patients), and cough (68% of patients) [6]. On chest radiograph, Covid-19 pneumonia often appears as bilateral patchy opacities. Chest CT, which has 95% sensitivity in early diagnosis of Covid-19, reveals ground-glass opacities. Most common laboratory investigations include normal or decreased leucocytes and lymphocytopenia [6] and, in severe disease, elevated D-dimer, lactose dehydrogenase, serum ferritin, and C-reactive protein [7]. Mitral regurgitation (MR) can result in severe respiratory failure by causing flash pulmonary edema, which can mimic pneumonia on chest CT [8]. While acute MR commonly presents with dyspnea and hemodynamic instability, a small portion of patients may present with dyspnea alone, making misdiagnosis for other pulmonary causes more likely (Table 2). Acute pericarditis can also present similarly to acute MR with a pleuritic chest pain radiating to the neck and shoulder as well as hemodynamic instability [9]. On physical exam, acute severe MR will produce a faint systolic murmur that is not as well appreciated as the holosystolic murmur in chronic mitral regurgitation; yet, failure to appreciate this murmur does not necessarily exclude its diagnosis [4]. In addition, electrocardiography can result in nonspecific ST and T wave abnormalities. Chest radiograph often shows a normal cardiac silhouette with pulmonary edema, which sometimes can be unilateral, and infiltrative shadows along the trachea which can be confused with pneumonia [4]. Echocardiography remains the gold standard for diagnosis of acute MR. Particularly, TEE can reveal the mechanism of regurgitation and severity as well as identification of the leaflet involved and annular size to plan for operative repair or replacement [4].

Due to the high mortality associated with untreated acute severe MR, a prompt diagnosis is critical. Delays in diagnosis as a result of cognitive biases may result in increased mortality. The most common cognitive biases include anchoring bias, confirmation bias, and availability biases [10]. The anchoring bias occurs when an individual places great value on a certain piece of information, the anchor, which then influences how the individual processes subsequent data. In our case, anchoring bias to Covid-19 took place when the focus of the treatment plan was based off of the respiratory distress and imaging suggesting pneumonia while other key findings to reject a diagnosis of Covid-19 were ignored or dismissed, and further diagnostic testing such as the echocardiogram was not prioritized. If the patient had undergone an echocardiogram the first day of admission, the source of the respiratory failure would have been uncovered and the patient may have received treatment sooner. Additionally, if the patient had not had access to ECMO therapy as quickly as she did, the outcome may not have been the same. Awareness to anchoring bias is necessary in order to prevent future misdiagnosis and delays in life-saving treatment, especially during the Covid-19 pandemic.

Table 2: Comparison of Covid-19 Mimics

	Covid-19 ARDS	Acute MR	Acute Pericarditis
Common Presentations	Fever, cough, dyspnea, loss of taste or smell, diarrhea, abdominal pain	Dyspnea, fatigue, hypotension, palpitations, signs of shock	Pleuritic chest pain that can radiate to neck and shoulders, nonproductive cough, low grade intermittent fever
Key Physical Exam Findings	Decreased breath sounds, hypoxia,	systolic murmur best heard over the apex	Pericardial friction rub auscultated over left sternal border
Chest Radiograph Findings	Patchy peripheral ground glass opacities or consolidation	Pulmonary edema sometimes unilateral with normal cardiac silhouette	May show enlarged cardiac silhouette
Management	Supplementation oxygen, Steroids, Remdesivir	Valve repair or replacement	NSAIDs, Colchicine
*ARDS: acute respiratory distress syndrome; MR: mitral regurgitation; NSAIDs: nonsteroidal anti-inflammatory drugs			

9. Conclusion

In summary, we have discussed a case of a patient with acute severe mitral regurgitation, initially diagnosed and treated as Covid-19 pneumonia. The differences on chest radiograph, CT, and laboratory investigations, although subtle, are an important reminder to consider other non-pulmonary pathologies when investigating dyspnea in order to avoid potential misdiagnosis. During a time when most respiratory failure can look like Covid-19, we must be cautious in our investigation.

10. Disclosures

This research was supported (in whole or in part) by HCA Healthcare and/or an HCA Healthcare affiliated entity. The views expressed in this publication represent those of the author(s) and do not necessarily represent the official views of HCA Healthcare or any of its affiliated entities.

11. Authors' Contribution

1. Michelle Zamora: This Author contributed to the design and implementation of the case report and to the writing of the manuscript

2. Omeni Osian: This Author provided critical feedback and contributed to the writing of the manuscript

3. Ettore Crimi: This Author contributed to the design and implementation of the case report and to the writing of the manuscript

12. Conflict of Interest

None, the authors have read the CARE Checklist (2016), and this manuscript was prepared in accordance with the CARE Checklist (2016).

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