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Haemorrhagic *Mycobacterium avium* complex pericarditis presenting with cardiac tamponade in an immunocompetent woman

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SUMMARY

A young woman in her mid-40s was referred by her primary care physician for fever, worsening shortness of breath, pleuritic chest pain and tachycardia. CT angiogram of the chest revealed a large pericardial effusion. Echocardiogram confirmed tamponade physiology despite her being haemodynamically stable. She had an emergency pericardiocentesis which revealed evidence of a haemorrhagic pericardial effusion. However, the patient was still symptomatic after treatment and had to undergo video-assisted thoracoscopic surgery with a pericardial window and chest tube. Postoperatively, her fevers resolved. Pan-culture was initially negative, and all antibiotics were discontinued. Acid-fast bacilli cultures later grew *Mycobacterium avium* complex. She continued to have chest discomfort postoperatively, but follow-up CT of the chest 3 months postoperatively showed continued resolution of her pericardial effusion. The patient's symptoms improved, and she has had no recurrence of effusion without the need for anti-tuberculosis drugs.

BACKGROUND

Cardiac tamponade is a syndrome caused by an accumulation of fluid in the pericardial sac (pericardial effusion) that is characterised by resultant diastolic filling impairment of the ventricles, leading to a reduction in cardiac output.[1 2] This syndrome is life-threatening and, if not diagnosed and treated promptly, could lead to cardiac arrest.[1 2] This case highlights the important fact that the clinical presentation of cardiac tamponade is variable, and a good understanding of tamponade physiology is key to identifying and managing patients who present with atypical findings.

There are several causes of pericardial effusion which can lead to tamponade. Infectious causes are common. However, *Mycobacterium avium* complex (MAC)-induced pericardial effusion is extremely rare with very few cases documented to date.[3] Furthermore, in the cases which have been documented, MAC-induced pericardial effusion is usually found in immunocompromised male patients.[3]

We present a rare case of MAC-induced pericardial effusion in an immunocompetent woman with no predisposing factors. Her case was even more interesting as she presented in a haemodynamically stable state despite having active tamponade from her large pericardial effusion. We explain the physiology behind her atypical presentation.

CASE PRESENTATION

A young woman in her mid-40s with a medical history of hypertension presented with 10 days of a non-productive cough, worsening shortness of breath, chest tightness and pleuritic chest pain relieved by sitting up. She reported fevers of up to 103°F. She denied any nausea, vomiting or diarrhoea but did admit to decreased appetite.

On examination, she was ill appearing but in no acute cardiopulmonary distress. Her pulse was regular but tachycardic. She had normal heart sounds with no rubs, murmurs or gallops. She had no jugular venous distension. The pulmonary examination was unremarkable. She had no lymphadenopathy. The remainder of her examination was unremarkable. She was also normotensive, tachycardic up to 138 beats/min, afebrile and not hypoxic when evaluated in the emergency department.

INVESTIGATIONS

Initial ECG showed evidence of sinus tachycardia with a heart rate of 124 beats/min, low voltage QRS complexes and poor R wave progression. Chest X-ray showed evidence of an enlarged cardiac silhouette (see figure 1). Her complete blood count was significant for haemoglobin was 10.1 g/dL with a platelet count of 515/nL and a normal white cell count of 4.96/nL. Complete metabolic panel showed only mild transaminitis. C reactive protein, erythrocyte sedimentation rate and D-dimer were significantly elevated (see table 1).

Given her elevated D-dimer, dyspnoea and tachycardia, pulmonary embolism (PE) was a concern. CT angiogram of the chest was negative for PE but revealed a 5.4 cm pericardial effusion (see figure 2A). STAT echocardiogram showed a large pericardial effusion with findings positive for tamponade physiology (plethoric inferior vena cava, collapse of the right atrium and right ventricle).

DIFFERENTIAL DIAGNOSIS

This patient's large haemorrhagic pericardial effusion could have been caused by malignancy, tuberculosis (TB) or possible viral pericarditis, with MAC representing contamination. However, we argue against this as none of these differentials would explain this patient's clinical course. The patient's flow cytometry was negative, cytology was negative, breast biopsy was also negative and the lack of a recurrent nature was not in keeping with a malignant effusion with contamination. TB was ruled out as all her sputum samples were negative

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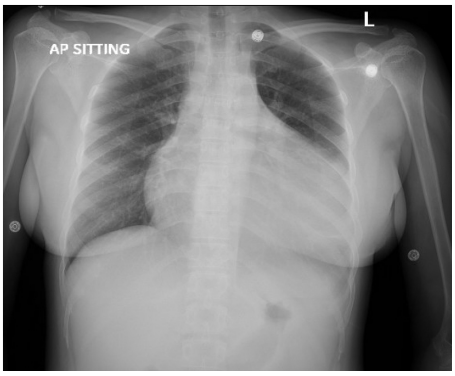

Figure 1 Initial chest X-ray. Note the enlarged cardiac silhouette.

for *M. tuberculosis* (MTB), in addition to the fact that PCR and gene sequencing for MTB were negative on all samples tested (see table 1). Given the haemorrhagic nature, viral pericarditis was less likely. In addition, testing for common viruses, including coxsackie virus on the pericardial and respiratory samples, was negative. Hence, the large haemorrhagic pericardial effusion, recurrent fevers, inflammation and necrotising granulomas seen on histopathology, growth of MAC in multiple pericardial samples and a positive QuantiFERON-Gold test in the setting of negative PCR for MTB made a localised MAC-induced pericardial effusion the most likely diagnosis in this case.

TREATMENT

She underwent emergency pericardiocentesis, during which 1 L of bloody pericardial fluid was drained (see table 1 for analysis). There was a concern for possible malignancy or tuberculous pericardial effusion. The patient was isolated, and serial sputum cultures were sent for acid-fast bacilli (AFB) staining. Immunoglobulin levels were measured and found to be normal. Rheumatological workup (table 1) was unremarkable; HIV, and hepatitis C and B were non-reactive.

Repeated echocardiogram and CT of the chest post-pericardiocentesis revealed residual moderate pericardial effusion (figure 2B), multiple small pulmonary nodules and bilateral breast lesions. There was a concern for malignancy. Symptoms persisted despite treatment with colchicine. Though she continued to spike fevers, blood cultures were negative, and serial sputum cultures were negative as well. On day 4 of admission, she underwent a right video-assisted thoracoscopic surgery (VATS) procedure with pericardial window and chest tube insertion.

During the procedure, the pericardium was noted to be tense and thickened posteriorly but not to the degree that warranted excision. A 400 mL of serous fluid was drained. Samples of the pericardium were also sent for microbiology and pathology. Inspection within the pericardial cavity revealed loculated fibrous material, which required blunt dissection to create a continuous cavity.

A decision to perform pericardiectomy, in this case, was not considered. This procedure is associated with a higher mortality and complication rate than a pericardial window and is usually indicated in cases of constrictive pericarditis, which this patient did not have.[4] Hence, the risk of performing such a procedure greatly outweighed any benefits and was not performed. Furthermore, loculations found on thoracotomy could be removed by simple adhesiolysis through blunt dissection. No tight fibrous bands were found that would have required the removal of the pericardium.

Table 1 Results of laboratory investigations

Test	Significant results
CBC	Mild anaemia, thrombocytosis and normal white cell count throughout hospital course
CMP	Mild transaminitis resolved
ESR	Elevated at approximately 3× the upper limit of normal
CRP	Elevated at approximately 6× the upper limit of normal
Troponins	Negative ×6
Microbiology	
Blood cultures	Negative ×2
Urine cultures	Negative
AFB sputum cultures	MAC in 4 samples out of 5
AFB pericardial fluid culture	MAC in 2 samples out of 3
AFB pericardial tissue cultures	Negative
Anaerobic and aerobic pericardial fluid cultures	Negative ×2
Anaerobic and aerobic pericardial tissue cultures	Negative
PCR and gene sequencing for *Mycobacterium tuberculosis* on sputum and pericardial fluid	Negative
PCR and gene sequencing for MAC on sputum, pericardial fluid or pericardial tissue	Not performed as MAC was already growing in several samples of sputum and pericardial fluid
Histology	
Histopathological report on pericardial tissue and intrapericardial debris	► Multiple fragments of benign pericardial tissues with inflammatory changes, and necrotising granulomas of the lymph nodes of the attached adipose tissue ► No malignancy identified ► No AFB and no fungal elements identified on Ziehl-Neelsen or silver stains, respectively
Malignancy workup	
Pericardial fluid cytology	Negative ×2
Pericardial fluid biopsy pathology	No evidence of malignancy
Flow cytometry	Negative ×2
Breast nodule biopsy	Benign fibroadenoma with sclerosing adenosis
Rheumatology/immunology/serology work-up	
ANA	Negative
RA latex	Negative
IgM levels	Normal
IgG levels	Normal
IgA levels	Normal
HIV	Negative ×2
Hepatitis C	Non-reactive
Hepatitis B	Immune

AFB, acid-fast bacilli; ANA, antinuclear antibody; CBC, complete blood count; CMP, complete metabolic panel; CRP, C reactive protein; ESR, erythrocyte sedimentation rate; MAC, *Mycobacterium avium* complex; RA, rheumatoid arthritis.

Postoperatively, she did well, and chest X-rays showed no evidence of pneumothorax with minimal drainage from the chest tube. She was started on indomethacin 50 mg orally every 8 hours. Her pericardial drain and pleural chest tubes were removed.

Postoperatively, her fevers and her chest pain resolved. AFB stains were all negative initially, cytology was negative, and viral studies for coxsackie and adenovirus from pericardial fluid were negative. Flow cytometry on pericardial

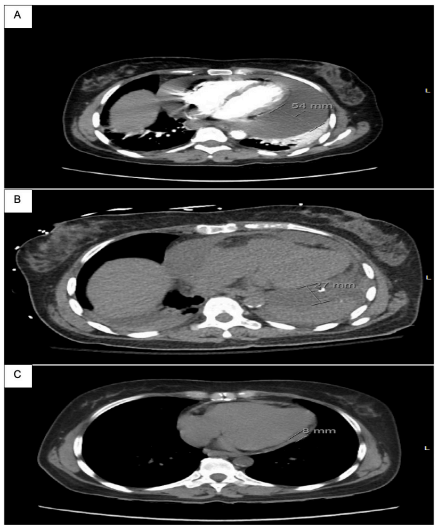

Figure 2 The changes in the size of the patient's pericardial effusion throughout her treatment course. (A) The patient's initial CT angiogram of the chest. Note the very large pericardial effusion on presentation measuring 54 mm in the widest portion. (B) Size of pericardial effusion 2 days after initial pericardiocentesis. Note there is still a moderate-sized residual pericardial effusion after pericardiocentesis measuring 27 mm. (C) The CT of the chest 3 months after discharge. Note that there is only a minimal amount of fluid measuring 8 mm.

fluid was also negative. Cultures from the pericardial fluid showed no growth initially.

Her initial QuantiFERON-Gold test was positive. However, all sputum cultures were negative for MTB, and PCR/gene sequencing for MTB was negative. Her symptoms resolved with drainage of her pericardial effusion and the use of non-steroidal anti-inflammatory drugs (NSAIDs) before discharge. Infectious disease specialists involved in the case from the beginning agreed that the patient had a negative PCR for MTB; thus, they effectively ruled out active and latent TB. However, given her initial positive QuantiFERON-Gold test, there was a plan to have it repeated and to closely monitor her in the outpatient setting.

She was discharged to complete an extended course of NSAIDs with plans for follow-up of her breast and pulmonary lesions as well as her pending cultures.

OUTCOME AND FOLLOW-UP

Her pericardial fluid cultures later grew MAC confirming a diagnosis of a localised MAC-induced haemorrhagic pericarditis complicated by cardiac tamponade. Infectious disease specialists evaluated her in the outpatient setting for latent TB. Her repeat QuantiFERON-Gold test was negative, and the patient's symptoms had resolved. As such, latent TB was determined to be unlikely and the initial QuantiFERON-Gold test was likely a false positive from her MAC infection at the time.

Infectious disease specialists agreed that since the patient was immunocompetent and was already showing significant clinical resolution with pericardial fluid drainage and NSAIDs alone, it was not worthwhile to subject this patient to the toxic side effects associated with an extended course of anti-mycobacterial medications.

Three months after her discharge, she was feeling better after having some residual chest discomfort. She had a biopsy of her right breast lesion, which was found to be benign. A repeat CT of the chest revealed a stable small 6 mm right upper lobe nodule, resolution of her previous smaller multiple pulmonary nodules and minimal fluid around the pericardium (figure 2C). The patient continues to do well and has had complete resolution of her symptoms and no recurrence of her effusion since being drained.

She has had several repeat CT scans of the chest which have shown resolution of her multiple pulmonary nodules, which were seen on scans during her illness. Her 6 mm right upper lobe nodule is stable in size. Given the fact that she is low risk for lung cancer, close monitoring with serial imaging was the appropriate plan. It is possible that it represents a post-inflammatory change.

The patient was also evaluated by oncology in the outpatient setting, who determined that malignancy was unlikely in this case and did not advise for further oncological workup.

DISCUSSION

Patients with cardiac tamponade usually present with dyspnoea at rest and/or on exertion, orthopnoea, chest discomfort or pleuritic chest pain.[1 2] Depending on the severity, the patient can also present with lightheadedness and syncope. On examination, the patient may present with the classic Beck's triad: hypotension, distended neck veins and muffled heart sounds.[1] Pulsus paradoxus is another common finding.[1] However, physicians should be very careful to avoid dismissing the diagnosis in the absence of these 'classic' clinical findings, as this is not a purely clinical diagnosis.

This case is a primary example of how a patient may present with cardiac tamponade in the absence of typical signs. She had no distended neck veins, her heart sounds were audible and she was normotensive. Her only positive findings were pleuritic chest pain, dyspnoea, orthopnoea, fever and sinus tachycardia on examination.

As pressure from the expanding fluid volume builds within the pericardium, the elastic fibrils of the sac stretch to accommodate the change.[2] When this happens gradually, the volume–pressure relationship is adjusted as extra space for expansion is created by the pericardial stretching over time. As such, a higher volume-to-pressure ratio is needed to impair ventricular filling pressures, thereby causing minimal effects on haemodynamics.[2] This accommodation will happen until the elastic fibrils reach the threshold of their maximum stretching capacity.[2]

However, if these increases in volume within the pericardial sac happen over a short period of time, even with a much smaller volume of fluid in the sac, there will not be time for fibrils to stretch to accommodate, and so it will take less volume to create enough pressure to overpower the diastolic ventricular filling pressures and impair cardiac output.[2] Therefore, the patient's clinical manifestations do not only depend on the amount of fluid present within the pericardial sac but, more so, the rate of fluid accumulation.[1 2] This explains why our patient had over 1 L of fluid in the pericardium and was normotensive and haemodynamically stable on presentation. Had this much fluid built up acutely over a short time, her heart would not have had enough time to adequately compensate, and she would have likely presented with Beck's triad or in cardiac arrest.

The unique cause of her pericardial effusion

The patient was found to have MAC-induced haemorrhagic pericardial effusion complicated by cardiac tamponade. This is a rare finding, as MAC-induced pericarditis is usually seen in

HIV-infected patients or those with other significant immunocompromised states. Historically, MAC infections in immunocompetent patients manifest as pulmonary infections in older white men with underlying lung diseases such as chronic obstructive pulmonary disease, lung cancer, prior TB or bronchiectasis, and a history of alcohol abuse.[3 5]

Our patient did not have any significant evidence of extensive pulmonary disease, and her sputum cultures were consistently negative for MTB. Furthermore, during the VATS procedure, there were no reported granulomas of the epicardium. However, she had multiple fibrous loculations within the pericardial space. This would indicate that her infection was primarily localised to the pericardium.

MAC is often found in tap water, soil, milk, domesticated and wild animals.[5 6] Like other non-tuberculous mycobacteria, it is non-communicable from person to person. However, it is clinically significant, as it can cause severe disseminated disease, particularly in immunocompromised patients, and has a remarkable propensity to develop resistance to most anti-mycobacterial agents.[6 7] Table 2 shows a summarised list of other cases of MAC pericarditis in immunocompetent patients. There are very few cases, and like our case, the mechanism by which MAC enters and infects the pericardium remains an intriguing enigma.

Treatment of MAC-induced pericarditis in an immunocompetent patient

Another interesting part of this case is that this patient's symptoms resolved without any anti-mycobacterial therapy. She underwent pericardiocentesis, then VATS drainage of her residual effusion, and was treated with a 6-week course of NSAIDs. Her fevers resolved, her cardiopulmonary symptoms resolved and there is no evidence of recurrence of her

Table 2 A list of four other documented cases of MAC-induced/related pericarditis in immune-competent patients and comparing them with this case

Case #	Title	Year	Summary of findings	Comparison with this case
1	A case of Mycobacterium avium complex infection in an immunocompetent man presenting with pericarditis and an HRCT pattern of lymphangitis[3]	2008	► A man in his early 50s presented with low-grade fever, malaise, dry cough and chest pain. ► Transthoracic echocardiography confirmed the presence of a mild pericardial effusion. ► HRCT showed evidence of pulmonary nodules in a lymphangitic pattern. ► The patient was immunocompetent. ► He was treated with clarithromycin and rifabutin for 10 months.	► This patient is a middle-aged man with evidence of primarily pulmonary MAC with a mild pericarditis. ► Our patient was a young woman with a large pericardial effusion with primarily pericardial MAC. Unlike this patient, she had no evidence of disseminated disease and so was treated with NSAID and drainage. She did not require antimicrobials.
2	A case study of Mycobacterium avium complex infection presenting with acute pericarditis[6]	2014	► A man in his early 70s presented with dyspnoea and pleuritic chest pain. ► A retired plumber, non-smoker and no history of lung disease ► On presentation was febrile, tachycardic and normotensive. ► Found to have moderate pericardial effusion up to 15 mm on CT and on echocardiogram ► Echo showed evidence of tamponade physiology, and he had 400 mL of pericardial fluid drained, which grew MAC. ► He was immunocompetent. ► He declined anti-mycobacterial and was discharged with NSAIDs. ► Poor compliance with NSAIDs led to recurrence. ► The patient resumed NSAIDs and was treated with an extended course and fully resolved without anti-mycobacterial.	► This patient is an elderly man. ► Non-smoker and no history of pulmonary disease like our patient. ► Has a moderate effusion with cardiac tamponade compared with our case where effusion was large with tamponade. ► Like our case, the condition was resolved with surgical intervention and NSAIDs without anti-mycobacterial.
3	Mycobacterium Avium Complex-Related Pericardial Effusion in an Immunocompetent Patient[11]	2017	► A woman in her late 50s with a 25 pack-year active smoking history presented with a cough productive of white sputum associated with 12-pound weight loss and decreased exercise tolerance. ► CT showed evidence of bronchiectasis, numerous pulmonary nodules, a large pericardial effusion with pericardial enhancement and a 2 cm hypodense hepatic lesion. ► The echocardiogram did not reveal any signs consistent with tamponade physiology. ► CT-guided biopsy of her pulmonary nodules revealed non-necrotising granulomas. ► Serial sputum samples showed MAC. ► Autoimmune workup and HIV testing were negative. ► The patient declined pericardiocentesis, and was treated with rifampin, azithromycin and ethambutol. ► After 3 months, her symptoms resolved, and pericardial effusion decreased in size on repeat serial echocardiogram.	► This patient is also female; however, she has an extensive smoking history and has primarily pulmonary MAC. ► She also has a large effusion like our patient. ► Unlike our patient, she did not have cardiac tamponade, declined pericardiocentesis and was treated with anti-mycobacterial.
4	A hemodialysis patient with *Mycobacterium avium* complex pericarditis in which remarkable presepsin elevation was not accompanied by procalcitonin elevation[7]	2020	► A man in his late 60s with a history of ESRD on HD and diabetes presented with cough and dyspnoea. ► He was found to have cardiac tamponade. ► 900 mL of pericardial fluid was drained, which grew MAC. ► He was HIV negative, had no evidence of malignancy or systemic immune dysfunction. ► He was not treated with anti-mycobacterial agents, and his condition resolved.	► This patient was a male with a history of DM and ESRD on HD. ► Our patient did not have such significant comorbidities. ► The management and outcomes were similar in that the patient was treated with surgical drainage and resolved without anti-mycobacterial agents. ► Pericardial effusion was also large with cardiac tamponade.

DM, diabetes mellitus; ESRD, end-stage renal disease; HD, haemodialysis; HRCT, high-resolution CT; MAC, *Mycobacterium avium* complex; NSAID, non-steroidal anti-inflammatory drug.

haemorrhagic effusion after 3 months. This case is one of very few documented where MAC pericarditis resolved with surgical drainage and an extended course of NSAIDs alone (see table 2 for a list of similar cases).

Like these cases, there was no evidence of disseminated infection. We postulate that since our patient, in addition to the above-stated patients, was immunocompetent, drainage of the effusion was enough to significantly decrease the bacterial load within the pericardial space, allowing the immune system to clear the infection more easily (much like with the drainage of an abscess). In the case described by Shiota, MAC antibody titres were measured post-pericardial drainage, which was noted to decrease gradually despite no anti-mycobacterial agents.[7] Such a finding supports our argument that in an immunocompetent patient with an absence of disseminated infection, surgical drainage and the use of NSAIDs are sufficient to prevent the recurrence of MAC-induced pericarditis. However, larger studies will be required to validate this argument.

One could argue that since this patient had such a significant illness from the infection, treatment with anti-mycobacterial therapy was warranted. However, as the studies in table 2 prove, this is not needed in every case to resolve the infection. While anti-mycobacterial therapy is an option, it is not the best option in this case. This patient showed clinical resolution of her symptoms even before her cultures were positive for MAC. At that point, her only treatment was surgical drainage and NSAIDs. Anti-mycobacterial treatment of MAC requires a very extended course as MAC is known to be notoriously resistant to anti-mycobacterial agents.[8] Given the higher dosing and longer treatments required, the side effects are significant.[8] Since the patient was clinically improving and immunocompetent, the risks of anti-mycobacterial therapy greatly outweighed the benefits. As such, the decision was made to treat conservatively with NSAIDs after the pericardial window.

Limitations

Some may argue that since MAC was only found in some samples and did not grow on histology samples, positive cultures could have resulted from contamination, given that this organism is ubiquitous in the environment. However, true infection cannot be reasonably excluded in this case for several reasons:

1. There were three pericardial samples taken on different dates in different sterile environments. The first was in the cardiac catheterisation laboratory from the pericardial tap done by the interventional cardiologist. The second was 2 days later in the operating room where thoracotomy and the pericardial window were done by the cardiothoracic surgeon. The third was taken as a sterile sample from the chest tube drain 2 days postoperatively. Two out of the three samples grew MAC.
2. This was further supported by four of five sputum samples growing MAC.
3. The patient had a positive QuantiFERON-Gold test initially. However, PCR and gene testing for MTB on pericardial fluid and sputum samples were negative. According to Harirzadeh *et al*, PCR is the gold standard for detecting MTB as it is highly sensitive and specific. It can reliably detect MTB in both active and latent TB.[9] Hence, this initial QuantiFERON-Gold likely represented a false positive. After resolution of her pericarditis with surgical drainage and NSAIDs, a repeated QuantiFERON-Gold test months later was negative, confirming that the patient did not have latent TB. Hence, the false-positive initial QuantiFERON-Gold test was likely from a true underlying MAC infection, as infections with non-tuberculous bacilli such as MAC can cause false-positive QuantiFERON-Gold tests.[10]
4. Lastly, though pericardial tissue cultures did not yield MAC, histopathology did show evidence of inflammatory changes and granuloma formation in attached lymph nodes. This further supported the presence of true infection.

Since anaerobic and aerobic cultures and viral studies were all negative and MTB PCR was negative, a true MAC-induced pericarditis remained the most likely explanation. Hence, we maintain that this patient had a localised infection of the pericardial space from MAC which led to her haemorrhagic pericardial effusion.

To our knowledge, this is the first documented case of MAC-induced pericarditis with associated cardiac tamponade in a young woman with no smoking, alcohol abuse or pulmonary disease

Patient's perspective

I was diagnosed with pericarditis 8 months ago. I still cannot believe what happened to me and how critical a situation I was in. I was in a state of denial in the beginning, I thought I was a relatively healthy person who is a non-smoker, non-drinker, who did not have any health issues except for hypertension. When my primary care doctor suggested I go to the emergency room as soon as possible to do EKG I did not realize it was life-threatening. I had fever for almost two weeks and in the later stage shortness of breath with some chest pain. I walked into the emergency room and walked back home after ten days, and that has changed my life forever. I cannot thank enough all the doctors, nurses, and other technicians involved in my treatment.

The emergency pericardiocentesis was done with local anesthesia, so I was watching the whole process and talked to the doctors. That was very scary. Later, VATS [video-assisted thoracoscopic surgery] surgery was performed with a pericardial window and chest tube on the right side. The ten days in the hospital were very overwhelming as I was the center of attention. The pain was immense but controlled with medications. The doctors explained the whole process and the treatments provided very well. The most difficult stage was coping with all these emotionally and mentally.

A week after I was discharged I was rushed to the ER [emergency room] due to immense pain in the lower ribs area. I was told the pain was due to the air sacs forming in the lungs area which took a couple of weeks to subside after the continuous breathing exercise. But this led me to be emotionally unstable and depressed. It is great that the pericardial effusion has not relapsed, but I am still concerned. I consider myself extremely lucky that the timing was right for me and I am in a country where the best treatment is available.

Looking back, I think if I had not gone to the doctor that day who knows if I would be alive today or if I was back in my home country, I am sure I would not have received the timely diagnosis and treatment which means I would not have survived. However, I am grateful. I appreciate that I am still breathing and continuing the journey of life. Each medical case is different and if my case could assist doctors, and researchers in understanding and explore in any way I think that's a good thing. A huge THANK YOU to all the doctors, nurses, and technicians in the hospital where I was treated and across the globe.

Learning points

- Cardiac tamponade is not a purely clinical diagnosis as patients can present without the typical hypotension, muffled heart sounds and distended neck veins.
- As was demonstrated in this case, the rate of pericardial fluid buildup is a more important contributing factor to the patient's clinical presentation than the size of the effusion itself.
- *Mycobacterium avium* complex (MAC) is a non-tuberculous *Mycobacterium* that has the potential to cause severe disseminated infections and localised infection of the pericardium.
- MAC is non-communicable from person to person and does not require airborne isolation.
- Like other non-tuberculous mycobacteria, MAC has a high resistance to anti-mycobacterials, and treatment of localised infections to the pericardium does not always warrant anti-mycobacterial therapy.
- In MAC-associated pericarditis with effusion, patients who are immunocompetent and without any signs of disseminated infection can be successfully treated with pericardial fluid drainage and non-steroidal anti-inflammatory drugs alone without the need for anti-mycobacterial agents.

history who was successfully treated with surgical drainage and NSAIDs only.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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