

All that Spikes Temperature is Not COVID: The Importance of Systematic Clinical Assessment of Patients Presenting with Pyrexia during the SARS-Cov-2 Pandemic

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Abstract

Since the diagnosis of first case of corona virus disease (COVID-19) in China six months ago, the contagion rapidly managed to grip the world in its stranglehold. The infection spread around the globe at a staggering pace and overwhelmed the healthcare infrastructures across the continents. Health authorities were quick to shift the focus on early identification and isolation of COVID-19 patients. The emphasis on COVID-19, while entirely justifiable on one hand, created challenges in approaching patients with common respiratory symptoms due to non-COVID disease on the other hand. We describe a case of young man who presented with chest pain and fever during the peak of pandemic and was mistaken for COVID-19 initially. Following further investigations, he was eventually diagnosed with pericarditis and a large pericardial effusion. This case serves as a reminder for the clinicians to systematically assess every individual patient presenting with pyrexia in order to ensure that appropriate care is provided.

Keywords: COVID-19; Pericarditis; Systematic Assessment; Echocardiography

Introduction

Currently, the total number of confirmed COVID-19 cases stands at 10 million with over half a million deaths across the world [1]. COVID-19's salient symptoms such as fever, cough and shortness of breath have been widely publicised by the public health bodies in order to raise awareness amongst the general population and health care professionals [2]. Whilst it is paramount that patients with SARS-Cov-2 infection are promptly diagnosed; it is equally important that other mimicking conditions are also effectively identified and treated [3]. We must not allow our vigilance about SARS-Cov-2 transmission detract us from following the medicine's foundational principles [4].

Case Presentation

This 31-year-old, previously fit and well man presented to the emergency department with fever and chest pain. He described the chest pain as constant, sharp, non-exertional and pleuritic in nature, which was worse on lying down. This was associated with rigors and chills, high grade fever, cough and shortness of breath. He was smoker of 15 cigarettes a day and was on no regular medications. On examination, he was pyrexial with a temperature of 39.9°C, with a heart rate of 102 per minute, respiratory rate was elevated at 30 per minute, blood

pressure and oxygen saturations were within the normal range. Physical examination revealed no respiratory distress, heart sounds were normal, and chest was clear to auscultation. Initial laboratory investigations showed normal white cell count with no lymphopenia, renal and liver function tests were within the normal range, d-dimer was raised at 4.12 $\mu\text{g/l}$ (0.0 - 0.5 $\mu\text{g/ml}$), c-reactive protein (CRP) was elevated 120 mg/ml (normal range 0.5 - 5.0 mg/ml) and serial cardiac troponin T levels were within the normal range. Electrocardiogram (ECG) showed sinus rhythm, ST segment elevation in V3 and V4 (arrows) and widespread T wave inversion in the inferior and lateral leads (Figure 1). Chest x-ray revealed cardiomegaly (arrows) and left basal shadowing (Figure 2). Initial clinical impression of COVID-19 related lower respiratory tract infection was made and as per local antimicrobial guidelines, he was treated with analgesia, intravenous antibiotics and intravenous fluids. Due to a significant elevation of d-dimer level, low molecular weight heparin was also administered to treat suspected pulmonary embolism. Subsequently, computerised tomography pulmonary angiogram (CTPA) revealed bilateral ground glass changes and a left lower consolidation (Figure 3). Additionally, it also showed a large pericardial effusion (Figure 4). There were no pulmonary emboli. Transthoracic echocardiogram confirmed a large global pericardial effusion with on maximum depth of 2.2 cm at the lateral wall (Figure 5 and 6). Cardiac contractility was normal and there were no regional wall motion abnormalities. Polymerase chain reaction (PCR) for SARS-Cov-2 viral RNA was negative on 3 occasions (0 hours, 12 hours and 72 hours post admission). Following this, his diagnosis was changed to 'Pericarditis with large pericardial effusion' and he was started on non-steroid anti-inflammatory drugs (NSAIDS) and Colchicine.

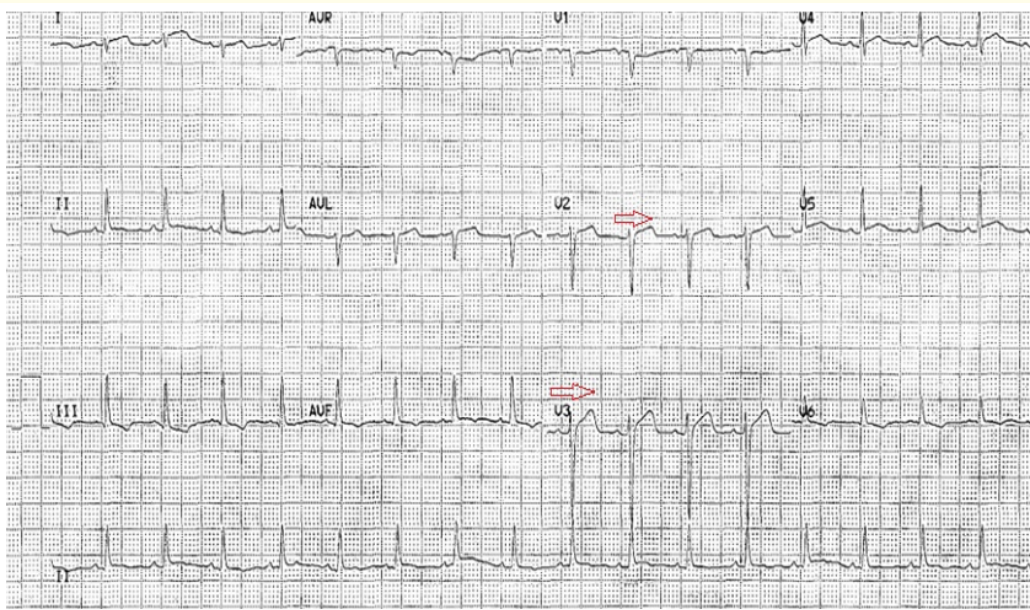


Figure 1: 12-lead ECG shows sinus rhythm, ST elevation in pre-cordial leads (Arrows) and widespread T wave inversion.

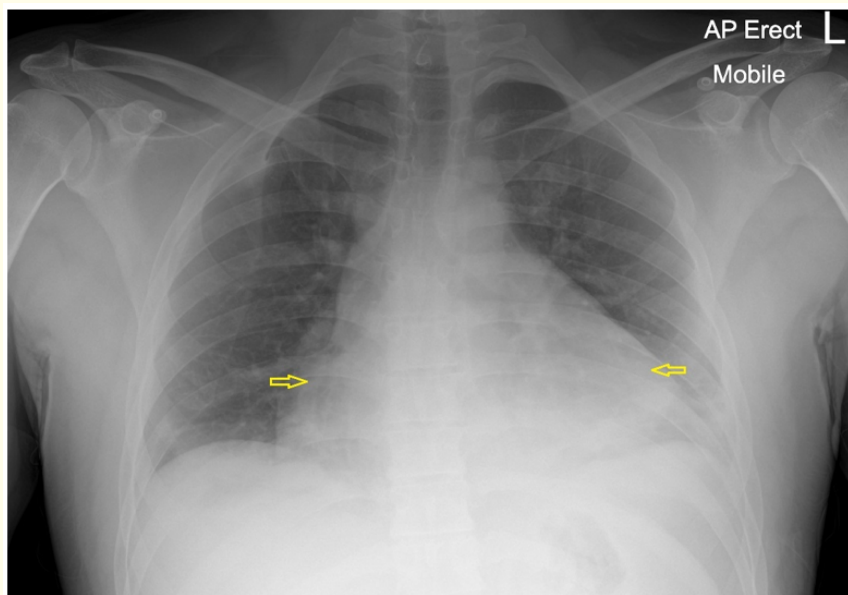


Figure 2: AP erect chest x-ray reveals enlarged cardiac shadow (arrows) and increased shadowing at the left base.

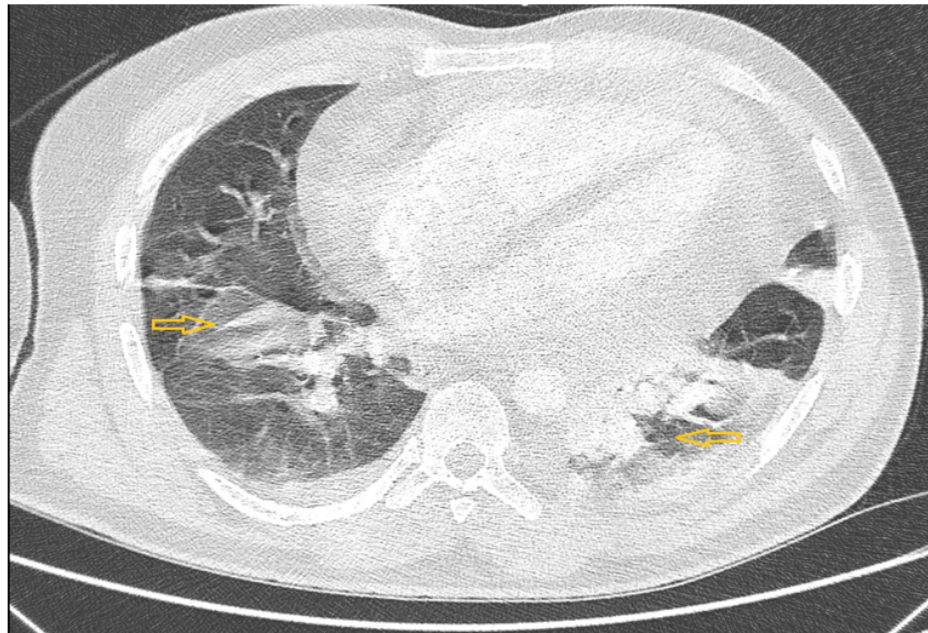


Figure 3: CT scan of thorax (lung windows) shows consolidation and ground glass changes at both bases.

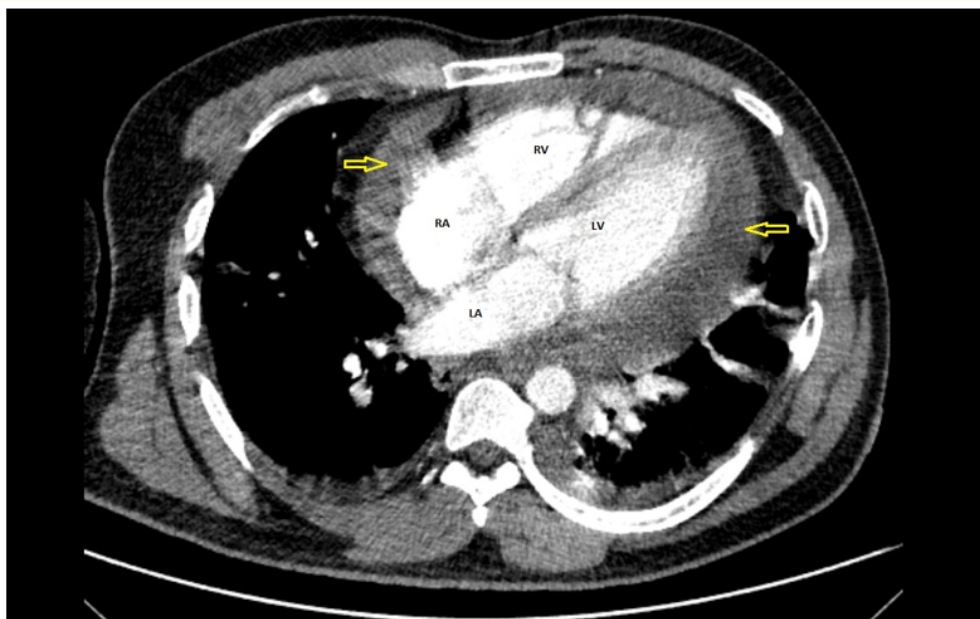


Figure 4: CT scan of thorax (with intravenous contrast) shows heart with labelled cardiac chambers and a large global pericardial effusion (arrows).

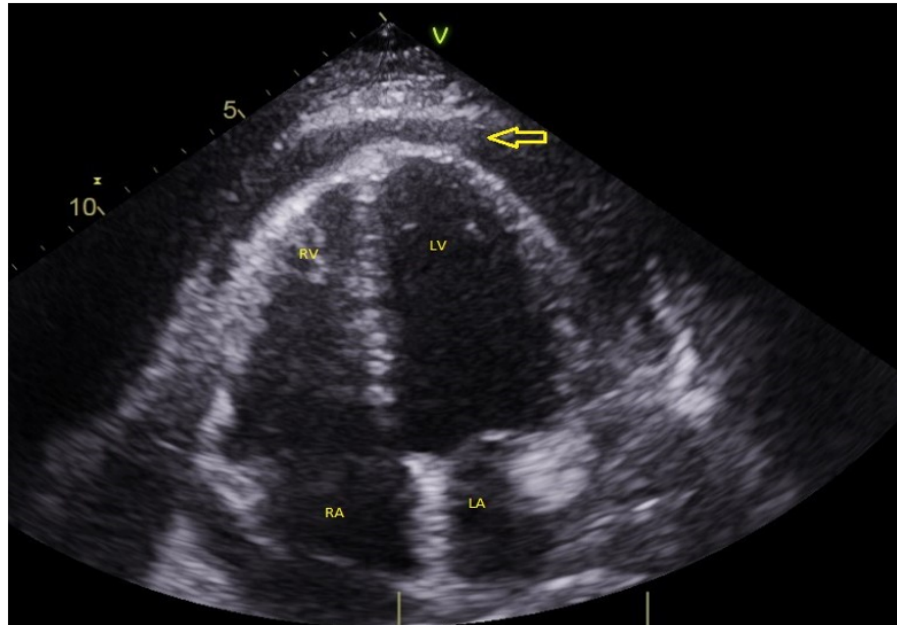


Figure 5: Transthoracic echocardiogram (apical 4 chamber view) demonstrates large pericardial effusion (arrow).

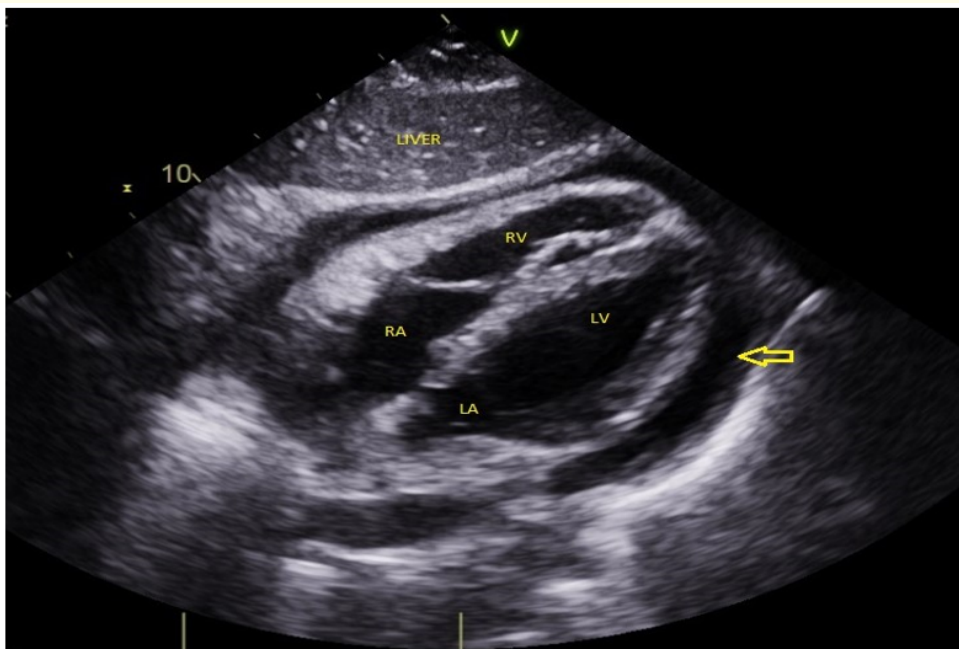


Figure 6: Transthoracic echocardiogram (subcostal view) confirms pericardial effusion (arrow).

Over the course of hospital stay, his condition improved; chest pain, pyrexia and inflammatory markers, all settled. Repeat echocardiogram on day 5 of admission showed a significant reduction in the size of pericardial fluid, leaving only a small rim of pericardial effusion. He was discharged home following his clinical improvement on oral Colchicine. A repeat echocardiogram performed 4 weeks after discharge showed a significant resolution of pericardial effusion. SARS-Cov-2 antibody test performed 4 weeks after the hospital admission was found to be negative, virtually ruling out any recent SARS-Cov-2 infection.

Discussion

The COVID-19 global pandemic caused by the SARS-CoV-2 virus has affected over 10 million people with an estimated death toll exceeding 5,00,000 at time of writing this article [1]. The characteristic symptoms include fever (98%), cough (76%), dyspnoea (55%) and altered sense of smell and/or taste (44%), alongside other constitutional features such as fatigue, myalgia and sore throat [5]. The incidence of chest pain is low at approximately 2% and is thought to be due to pleurisy [6]. However, these symptoms are not unique to SARS-Cov-2 infection; a number of other common viral and bacterial infections may present also with similar clinical features.

Pericarditis is long known to have a close association with viral infections, as demonstrated with influenza, coxsackie and parvovirus B-19 viruses [7]. Clinical features of acute pericarditis include a classic chest pain that improves with leaning forward, pericardial friction rub, typical electrocardiogram (ECG) changes and pericardial effusion [8]. No clear cause is found in over 80% of cases which are presumed to viral in origin. Laboratory investigations such as complete blood count, C-reactive protein measurement and cardiac troponin levels, though non-specific, but may help to aid the diagnosis. Electrocardiogram (ECG) classically shows a concave ST elevation in multiple leads with a relative PR depression. Bedside echocardiography can demonstrate pericardial effusion which can be occasionally large enough to cause cardiac tamponade [9].

Non-steroidal anti-inflammatory drugs (NSAIDS) and colchicine are the first line treatments options and yield a good initial response on most patients. Whilst a short duration of treatment with NSAIDS is generally sufficient to resolve the symptoms; it is advised to continue colchicine for up to 12 weeks to prevent recurrence [10]. There is no real benefit of gradual tapering of the dose over abrupt discontinuation of treatment. Glucocorticoids can be used for refractory cases however there is a higher recurrence risk with their use [11]. Cardiac tamponade is a medical emergency suspected in the presence of raised jugular venous pressure, muffled heart sounds and hypotension. This warrants an urgent echocardiography followed by drainage of the pericardial fluid to reduce the pressure within the pericardial sac and allow adequate ventricular filling. Recurrent pericarditis can be disabling but will often respond to NSAID-colchicine treatment. Pericardiectomy can be performed for resistant cases; however it is not always effective in relieving the symptoms [11].

Conclusion

This case provides an insight into clinicians' thought process and clinical reasoning during the pandemic. In our patient, the clinical suspicion of a large pericardial effusion rose from cardiomegaly on the chest radiograph. This case also demonstrates the importance of early echocardiography in patients with pyrexia and chest pain to facilitate the diagnosis. It also highlights an important clinical dilemma; COVID-19 symptoms such as cough and pyrexia are also found in other infections hence clinician should always keep an index of suspicion for other differential diagnoses. Case recognition and early isolation is the cornerstone of our fight against the pandemic however this should not happen at the cost of disproportionately affecting the care of non-COVID patients.

Conflict of Interest

None.

Author Contributions

SA and WJ wrote the initial manuscript. TN and NS edited the manuscript and images.

Patient Consent

An informed consent was obtained from the patient.

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