

## Coincidental Finding of Large Thoracic Aortic Aneurysm in A Patient with COVID 19 Pneumonia: A Case Report

Madeleidis Lopez Leon, Dariex Rodriguez, Sahar S Abdelmoneim\*, Sandy Espinosa Hernandez,  
Hamlet Monteagudo, Manuel De La Cruz Seoane, Saba I Gomez, Odalys Frontela

Division of Internal Medicine, Larkin Palm Spring Community Hospital, Hialeah, Florida, USA

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\***Corresponding author:** Sahar S Abdelmoneim. Division of Internal Medicine, Larkin Palm Spring Community Hospital, Hialeah, Florida, USA

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### ABSTRACT

The pathological involvement of the aorta with atherosclerotic damage, infectious process or immune dysregulation can co-occur and may have some mimicry symptoms in common. This is becoming more apparent in the current era where there is mixed population with immigrations, increased life expectancy and co-occurrence of infectious etiologies in the setting of COVID 19, aortopathy, and atherosclerotic pathology all in one single patient. We herein, present a case of an 83-years Hispanic female in whom chest X ray revealed mediastinal widening. The patient presented with COVID 19 pneumonia. CT Chest revealed the incidental finding of a massive Thoracic Aorta Aneurysm (TAA). Patient was treated for the COVID 19 pneumonia. She remained asymptomatic from the TAA and opted for conservative medical management as the choice of care. Six-month follow-up showed evidence of repeated hospitalization and complications

**Keywords:** COVID 19; Thoracic Aorta Aneurysm

### INTRODUCTION

Research data remains limited to answer the question of the cooccurrence of COVID 19 and aortic disease aneurysm and its complications.<sup>[1]</sup> Interesting pathophysiologic mechanisms underlying aortitis in COVID 19 patients has been reported. Possible pathophysiology mechanisms include acute endothelitis (Occurring from virion and anti-inflammatory response infiltration of the endothelium) and pan arteritis with leucocytoclastic vasculitis resulting in hypersensitive vasculitis (with immune complexes depositions). Such COVID 19 pathophysiology favors a preexisting atherosclerotic plaque pathology which is common in the elderly with cardiovascular risk factors. Theoretically, COVID 19 can also worsens the existing vascular pathology leading to complications and challenging differential of acute aortic conditions.<sup>[2]</sup> It is unclear if the co - occurrence of COVID 19 and aortic aneurysm pathology is becoming more apparent in the current era given there is mixed population with immigrations, increased life expectancy and co- occurrence of infectious etiologies in the

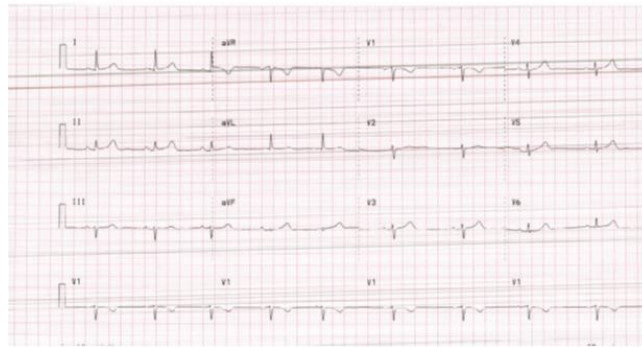
setting of COVID 19, aortopathy, and atherosclerotic pathology all in one single patient.<sup>[3]</sup> Combining clinical data from thorough history taking and physical examination in a multisystem approach represents the first step in the diagnostic workup and followed by diagnostic imaging studies that leads to a presumptive diagnosis in most patients. Computed tomography chest (CT) provides invaluable information regarding the aortic pathology, characteristics, location, size, and relationship to surrounding structures.<sup>[1]</sup> In this case report, we present a case of an incidental finding of a large Thoracic Aorta Aneurysm (TAA) in an elderly female who presented with COVID 19 pneumonia. Patient was treated for the COVID 19 pneumonia. She remained asymptomatic from the TAA. The case was challenging because determining the optimal elective surgical TAA repair timing is challenging especially in the setting of COVID 19 and other individual patient risk factors (age, body habitus, comorbidities, and rate of TAA expansion). In this case report, given the increased surgical risk and patient refusal of any surgical intervention, conservative medical management was undertaken. Conservative medical management aim to decrease the wall stress on the aorta by maximizing risk factor control to limit aortic wall expansion. Furthermore, patient education regarding the need for ongoing aneurysm size surveillance with imaging and follow up is of paramount importance. The patient was informed that data concerning the case would be submitted for publication, and she provided an informed consent.

## **CASE REPORT**

An 83-year-old Hispanic female lifetime nonsmoker with past medical history of hypertension, hypothyroidism, asthma. Patient has a known allergy to Iodine and penicillin. She presented for further evaluation of worsening shortness of breath and feeling ill and subjectively feverish and one episode of vomiting for one day after being in contact with the daughter who had COVID 19 infection. She denied cough, chest pain, orthopnea, paroxysmal nocturnal dyspnea, any gastrointestinal or urinary symptoms. The patient had no significant personal of carcinoma and there were no complaints of hoarseness of voice or dysphagia. Family history was non-contributory specifically for additional clinical risk factors including Marfanoid habitus or positive family history of possible underlying genetic conditions known to be associated with Thoracic Aortic Aneurysm And Dissection (TAAD) as Ehler Danlos. Her home medications included albuterol sulfate inhaler, montelukast 10 mg tablet once daily, Levothyroxine 75 mcg tablet once daily, and losartan 25 mg tablet once daily. During evaluation, the patient's blood pressure was 147/86 mmHg, pulse was regular at 65 beats/min, Temp 98.1 F, oxygen saturation of 93% on Room Air but 97% on 2 Liter Nasal canula, and respiratory rate of 18/min, Body surface area (1.49m<sup>2</sup>). On examination, the Chest examination was unremarkable apart from mild basilar inspiratory

crackles. Neck examination did not reveal any lymphadenopathy or thyroid gland enlargement. Abdominal, cardiovascular, and neurological examinations were unremarkable, and no evidence of pulsating abdominal pulsations noted. Extremities examination was negative for edema, and peripheral pulsation was palpable. Labs was as follows: White Blood Cells of 7.7 (reference range 3.4-11.0 10<sup>3</sup>/uL), serum creatinine of 0.86 (reference 0.8-1.4 mg/dL), Blood Urea Nitrogen 29 (reference 8-26 mg/dL), and BUN/create 34 (reference 10-14 mg/dL), D-Dimer 15.05 (reference range 0-0.5 mg/dL), PCT 0.096 (reference range 0.0-0.07 ng/ml), CRP > 1.5 (reference range 0- 0.5 mg/dL), Ferritin 200 (reference range 11.1-264 ng/ml), LDH 266 (reference range 120-246 U/L), ESR 17 (reference range 21mm/Hr.), Vit D levels <12.8 (reference range 30-100 mg/dL),

Troponin < 0.012 (reference 0-0.04 ng/ml), TSH 1.48 (reference range 0.46- 4.68 uIU/ml) and a COVID positive rapid test. Twelve lead Electrocardiogram (ECG) showed a normal sinus rhythm (Figure 1). A chest radiograph (Figure 2) Showed large left paratracheal opacity with curvilinear calcification suggestive of thoracic aortic aneurysm an enlarged upper mediastinal silhouette with no focal pulmonary consolidations, pleural effusions, or pneumothorax. Patient was admitted to the hospital and COVID protocol was initiated including Azithromycin 500mg Intravenous, Q24H Vitamin D3 10 mcg (400 UNIT) orally daily, Zinc sulfate 220 mg orally daily, ascorbic acid 1000 mg orally daily, Solumedrol 60mg IVP twice daily, Benzonatate 100 mg orally daily Q8H Per need, and oxygen supplementation as needed per saturation. In hospital medications also included the patient home medication listed above and in addition metoprolol tartrate 25 mg orally Q12H and Atorvastatin 80 mg orally daily were added. Given elevated D dimer, the patient underwent an ultrasound vein bilateral on lower extremities and there was no evidence of acute deep venous thrombosis in the visualized bilateral lower extremity veins. Furthermore, lung NM VQ scan showed homogeneous perfusion activity throughout the bilateral lungs with no evidence of segmental perfusion defects or definite areas of abnormal perfusion were identified. Further clinical workup for the enlarged mediastinal silhouette was undertaken. Comprehensive 2D, Doppler, and color-flow echocardiogram (Figure 3) Revealed a normal size Left Ventricle (LV), normal LV segmental wall motion with preserved systolic function (LVEF 55%) but moderate diastolic dysfunction is present (pseudo normal filling). Normal Right Ventricular (RV) size and systolic function and no valvular pathology, specialty bicuspid aortic valvopathy. Abdominal ultrasound was also performed to evaluate and screen for abdominal aneurysm, and it showed mild diffuse atherosclerosis of the aorta with no evidence of aneurysms or areas of hemodynamically significant stenosis or occlusion. With the limitation of presenting with mild prerenal acute kidney injury and being allergic to Iodine, CT scan of the chest was performed using individualized dose optimization techniques including automated exposure control, adjustment of the mA and/or kV and without the use of intravenous contrast agent. CT chest (Figure 4 A-D) demonstrated fusiform dilation of aortic arch [end diastolic aortic diameter of 6.9 cm, with aortic size index (aortic diameter, cm divided by body surface area, m<sup>2</sup> of 4.63 cm/m<sup>2</sup>, with the reference range for surgical intervention being aortic size index  $\geq 2.75$  cm/m<sup>2</sup>] (4). Additionally, there were focally displaced intimal calcification suggestive of possible penetrating atherosclerotic ulcer. Extensive circumferential atherosclerosis with severe intimal calcification and atherosclerotic plaques in the visualized aorta. In addition, bilateral non-specific geographical ground-glass opacity was noted. However, no pulmonary consolidation, pulmonary mass, or significant pulmonary nodule was detected. Further testing to exclude syphilis infection, a screening Rapid Plasma Reagin (RPR), a nontreponemal test, was not reactive. The patient was informed with the test result. Cardiothoracic team evaluated the case and concluded that she would need a surgical repair, However the patient opted to continue with conservative management. The hospital course was benign and COVID infection resolved. She was clinically and hemodynamically stable. She was discharged from the hospital with instruction to follow up with his primary care physician and cardiologist on a regular basis. Six-month follow-up showed evidence of repeated hospitalization and complications



**Figure 1:** A 12-lead electrocardiogram showing normal sinus rhythm and normal QRS, S-T segment, and T waves.



**Figure 2:** Postero-anterior chest X-ray showing widened mediastinum, large aortic knob, and mild displacement of the trachea from the midline to the left. Additionally, large left paratracheal opacity with curvilinear calcification suggestive of thoracic aortic aneurysm. There was no focal pulmonary consolidations or pleural effusions.



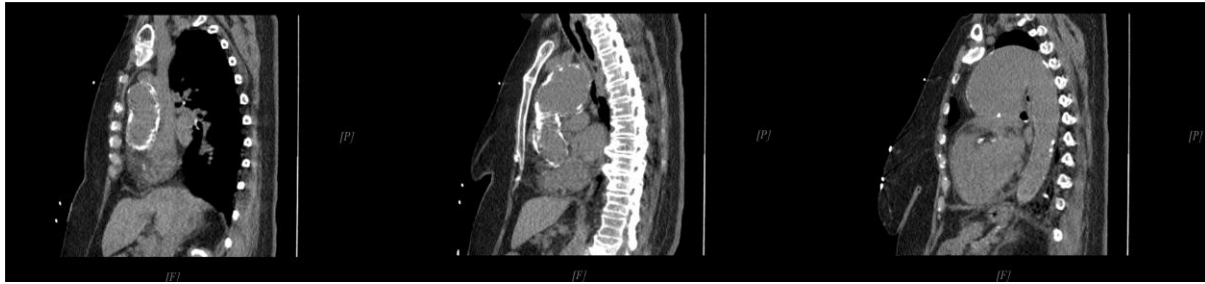
3A)

3B)

**Figure 3:** Transthoracic Echocardiogram showing (A) Short Axis View of the aortic valve, (B) M mode of the aortic valve.



4A)



4B)

**Figure 4:** Thoracic CT in an 83-year-old female (body surface area of  $1.49 \text{ m}^2$ ) illustrating the mass in different sections (A) Coronal CT section showing fusiform dilation of aortic arch (end diastolic aortic diameter 6.9 mm) with focally displaced intimal calcification suggestive of possible penetrating atherosclerotic ulcer. Extensive circumferential atherosclerosis with severe intimal calcification and atherosclerotic plaques in the visualized aorta (B) Sagittal Section demonstrates similar findings. The calculated aortic size index (aortic diameter, cm divided by body surface area,  $\text{m}^2$  was  $4.63 \text{ cm}/\text{m}^2$ , with the reference range for surgical intervention being aortic size index  $\geq 2.75 \text{ cm}/\text{m}^2$ ).

## DISCUSSION

In our case, we present an incidental finding of diagnosing Thoracic Aortic Aneurysm (TAA) in an elderly 83 female who is presenting with COVID 19 pneumonia. Our patient exhibited atypical symptoms with no symptom or signs specifically related to TAA, hence is the reason for delayed diagnosis. The co-occurrence of TAA with COVID 19 illness is not well researched.<sup>[5]</sup> However, the Pandemic was shown to affect many patients in seeking medical routine follow up with primary care physicians. As we demonstrated in our case, chest radiographs were the first commonly performed imaging modality that led to the incidental discovery of the large Thoracic Aortic Aneurysm (TAA). Our patient denied any of personal diagnosis of any genetically mediated conditions associated with aortopathy (bicuspid aortic valve, Marfan's or Ehler Danlos syndrome) and was not aware of having TAA. Hence it was assumed more likely that her TAA is of degenerative related to cardiovascular risk profile.<sup>[2]</sup>

It was shown that the risk of rupture or dissection in cases of TAA increases with larger aortic diameter, especially expansion of ascending TAA beyond 6 cm, or the descending TAA beyond 7 cm. Hence, the consensus for degenerative TAA is elective repair cutoff for TAA include (In low- risk perioperative complication: if ascending TAA end-diastolic aortic diameter  $>5.5 \text{ cm}$ , and descending TAA end-diastolic aortic diameter  $>5.5 \text{ cm}$ ; and in higher risk perioperative complication if end diastolic aortic of any TAA diameter  $> 6 \text{ cm}$ ).<sup>[6]</sup> Our patient had a fusiform aortic arch TAA, with an end diastolic aortic diameter of 6.9 with calculated aortic size index of  $4.63 \text{ cm}/\text{m}^2$  (reference range for surgical intervention being aortic size index  $\geq 2.75 \text{ cm}/\text{m}^2$ ). The patient was asymptomatic and was also deemed to be high risk based on the Society of Thoracic Surgeons Predicted Risk of Mortality (PROM)<sup>[7]</sup> score  $>8$ , frailty score  $>2$ , and having more than one major organ system impairment (in her case is acute kidney injury). Furthermore, her informed decision was refusing any surgical intervention. This led to the consensus between cardiologist and cardiothoracic surgeon team to opt for conservative medical management and frequent surveillance for aortic size as the best choice for her. The

standard surgical option for aortic arch TAA is conventional surgical total arch replacement (TAR) using cerebral perfusion with hypothermia for brain protection.<sup>[8]</sup> However, TAR carries a major surgical risk of morbidity and mortality. With the emergence of the less invasive option of Thoracic endovascular aortic repair (TEVAR) as an alternative treatment for TAA, and in aortic arch TAA using the supra-aortic bypass (Hybrid arch TEVAR), successful outcomes have been reported especially in patients > 75 years of age.<sup>[8]</sup>

The co-occurrence of COVID 19 and large TAA in our patient can be coincidental. We were limited in this case by not having any available imaging data or a prior diagnosis of TAA in this patient. Hence, we cannot exclude the possibility of COVID 19 induced inflammatory effect that led to aortic aneurysm expansion. Several published case reports have noted incidental findings of aortic aneurysms perse or presentation with complicated aortic aneurysm in the COVID 19 era (3), (9). We believe this case is worth sharing and publishing to raise awareness about the possible association of COVID 19 and Aortic pathology or increased complications from rapidly progressing expansion or other complications of dissection and rupture. Further studies are recommended.

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