Case Report

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Extrapulmonary Complications of SARS-CoV-2 Infection: Radiology Imaging

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1. Abstract

The coronavirus pandemic is a global pandemic of coronavirus disease 2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The virus targets mainly upper respiratory tract, however, it has been proven to cause complications in systems other than the respiratory system. In this article we will discuss the extrapulmonary complication caused by COVID-19.Among 222 hospitalized individuals due to COVID-19 in Al-Koura Hospital in Lebanon, 26 suffered from such complications, 17 of them were diagnosed with extrapulmonary complications, such as pneumopericardium, thromboembolism, muscular hematoma, stenosis of arteries, muscular abscess and pericardial effusion which was the most prevalent complication. Among the 17 patients with extrapulmonary complications, 8 had no past medical history and 9 with a history of hypertension, diabetes mellitus, heart failure or combination of them. Fisher test showed that the association between extrapulmonary complication and past medical history was statically insignificant. The pathogenesis of some of these complications is well documented; for instance, thromboembolismsis caused by a prophylactic treatment of COVID-19 as muscular hematomas. However, the pathogeneses of other extrapulmonary complications are still unknown such as muscular abscess and arterial stenosis. We are herein tackling those complications and the molecular pathways involved in their development, aiming at implementing effective treatment strategies.

2. Keywords:

COVID-19; Cardiology; Thromboembolism; Hematoma; Radiology

3. Introduction

In December 2019, pneumonia of unknown origin emerged in Wuhan, China, and similar cases proceeded to occur and spread swiftly [1]. Although the disease initially confined itself to China, it rapidly propagated to over 160 different countries due to its contagious characteristics [2]. Through its contact and droplet mode of transmissions [3], the numbers of cumulative cases reached about 1,280,000,000 in July 2023 [4]. Bronchoalveolar lavage samples were collected and analyzed in order to identify the infectious agent, which ultimately led to the virus's recognition [2]. As the novel virus exhibited a strong genetic resemblance to SARS-CoV [5], it was given the name SARS-CoV-2in February 2020. With this, the technique of its diagnosis was also found and pushed forward by the WHO, nuclear acid amplification was found to be the basic test for potential COVID patients [6].The incubation phase lasts around 6 days. The disease had no distinct presentation, with symptoms fluctuating between absent to dry coughs to severe pneumonia and death [7]. Although the symptoms were mostly pulmonary in nature, extrapulmonary complications arose and caused further problems [8].This case report will look at the extrapulmonary problems of COVID patients who were hospitalized at Lebanon's Koura Hospital, as well as the underlying processes.

3.1 Pericardial Effusion

Pericardial effusion is a medical condition where there is an abnormal accumulation of fluid in the pericardium. This fluid can cause an increase in pressure within the pericardium and lead to a variety of symptoms, including chest pain, shortness of breath, rapid heartbeat, and fatigue. As they are, pericardial effusions have no effect, until they cause a tamponade. The latter happens when the increase in tension of the pericardium hampers the beating heart. In acute pericardial effusion, 150 to 200 ml can be enough to cause it, while in chronic 1 or 2 Liters can accumulate to cause it [17]. Many factors can lead to pericardial effusion, including viral or bacterial infections, certain medical conditions (such as cancer or autoimmune diseases), injury to the heart, or medications. In some cases, the cause may be idiopathic[18]. Post-viral pericardial effusion also called idiopathic pericardial effusion doesn't have a complete mechanism for its pathogenesis. Some other viruses such as influenza, cytomegalovirus, adenoviruses, and others have already been known to cause idiopathic pericardial effusion by means of acute pericarditis. COVID-19 has

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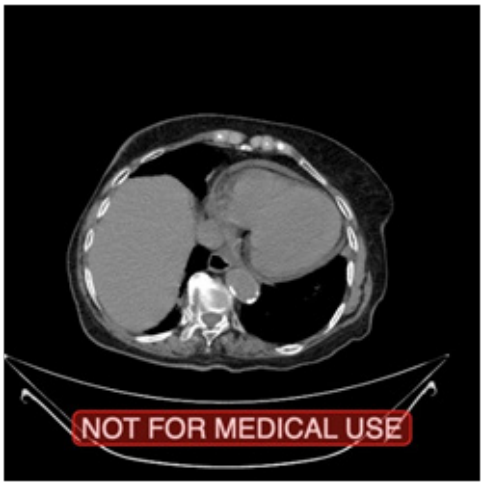
also been added to these pericardial effusion causing viruses, through possibly causing acute pericarditis or even myocarditis [19, 20]. With COVID-19 infection, pericardial effusion is found to be one of the more common complications with 22% of patients being affected [19]. Our study found that 3.66% of hospitalized COVID-19 patients developed this complication as shown in Fig.1.Treatment of pericardial effusions should be directed towards its etiology. As discussed earlier, a pericardial effusion caused by any virus including COVID-19 is idiopathic as such it should be treated with empiric pericardial effusion. NSAIDs are the drugs of choice. Colchicine or corticosteroids at low doses might be indicated in some cases [21].

Figure 1: CT scan showing pericardial effusion.



3.2. Pneumopericardium

Pneumopericardium is the presence of air in the space between the heart and the pericardium. The etiologies behind its development are multiple including trauma, fistula formation, congenital, infection with gas-forming bacteria, or congenital [9]. Traumatic type of injuries can be either blunt force, penetrating, or even caused by positive pressure ventilation called barotraumas [9]. Fistula, is a communication between the pericardial space and another air-containing structure, such as trachea, bronchus, transverse colon, esophagus, or even stomach [9]. Infection with bacteria such as Peptostreptococcus, Klebsiella, and Escherichia Coliwould lead to pneumopericardium due to the gas forming capabilities of these organisms [10]. Lastly, congenital abnormalities such as absent pericardium or absent diaphragm can allow the air found in the peritoneum to get into the pericardium[9]. Pneumopericardium like pericardial effusion can be symptomatic or asymptomatic depending on the air volume in the pericardial space. If the pneumopericardium is of small volume it can be asymptomatic and physical exams can be clear, in this case, the finding of a small volume pneumopericardium is incidental on imaging. If the volume is large, it is more probable for symptoms to develop, these symptoms will be similar to pericardial effusion symptoms like dyspnea and pericardial chest pain[11].

In COVID-19, pneumopericardium happens due to a barotrauma injury. Any act that increases intra alveolar pressure from excessive coughing, positive pressure ventilation, to the valsalva maneuver, can cause a rupture of the alveoli, then air travels backward with the bronchovascular sheath and enters the mediastinum, furthermore, air can enter the pericardium along the less collagen supported venous sheaths[12, 13]. Pneumopericardium is a rare complication of COVID-19 with 1% prevalence [13]. In our study, out of 222 hospitalized individuals 3 developed pneumopericardium (1.3%) as shown in Fig 2and Fig 3. Pneumopericardium treatment depends on its symptoms. If the air volume is small and there are no symptoms, treating its cause is usually sufficient, but monitoring is necessary [14]. However, if the air volume is prominent , chest tube is used to drain the air and alleviate the tamponade [15]. Diagnosis of pneumopericardium is based on radiological findings, X-ray showing lucent outline separating the pericardium from the heart, computed tomography scan, or even echocardiography [16].

Figure2: X-ray showing lucent outline separating the pericardium from the heart.

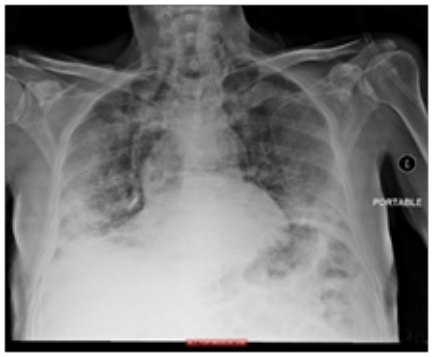
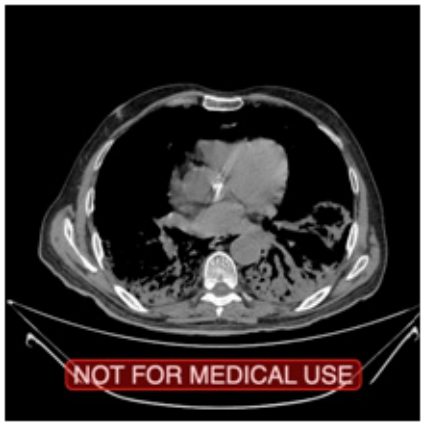


Figure3: CT scan showing pneumopericardium.

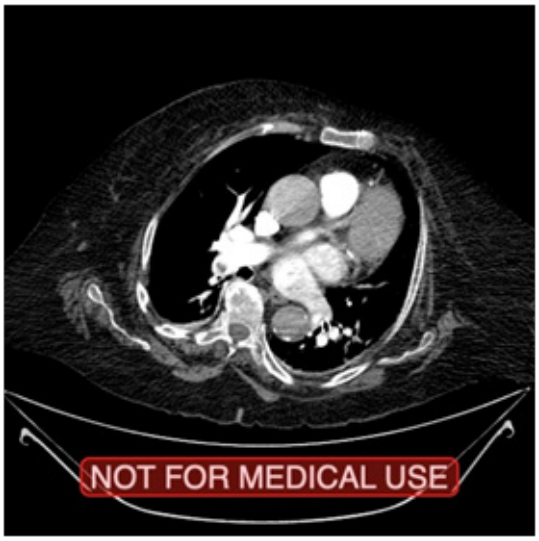


3.3. Thromboembolism

When blood clots obstruct blood vessels, this is referred to as thrombosis. Thrombosis is classified into two types: 1) venous thrombosis; when a blood clot blocks a vein 2) arterial thrombosis; when a blood clot obstructs an artery [22].The repercussions of thrombosis fluctuate depending on the area in which it occurs as it reduces blood flow to that region. If a thrombus remains untreated, it may detach and travel through the systemic circulation as an embolus, potentially leading to heart attacks, strokes, and

pulmonary embolisms,as shown in Fig 4 [22, 23].

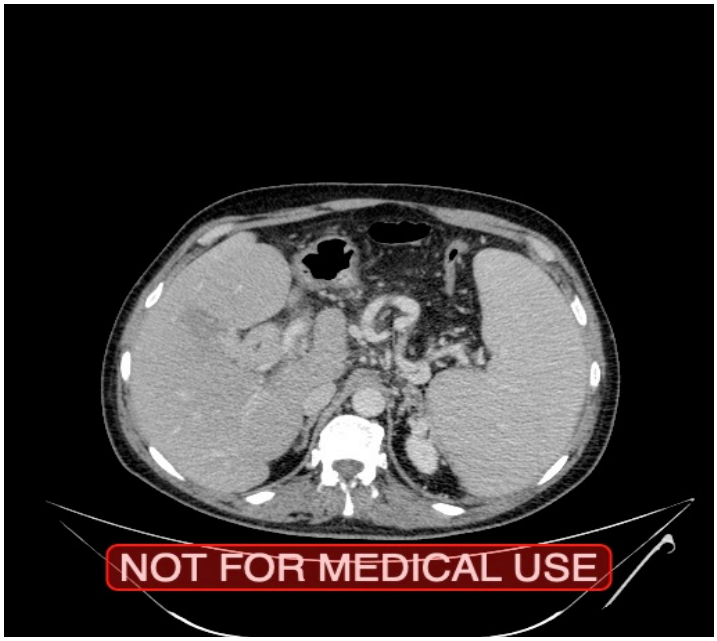
Figure 4: CT scan showing extensive embolism of the right lower main pulmonary artery.



In this study, 2.7% of the documented patients were diagnosed with an embolism or thrombosis. COVID 19virus induces a prothrombotic state through a variety of mechanisms. Firstly, a hypercoagulation state increases the risk of significant vessel thrombosis. This is due to the accompanying cytokine storm during the infection as the body attempts to fight the virus, with IL-6 being the most noteworthy [22, 23, 24, 25, 26]. Furthermore, several research focused on micro thrombosis caused by direct vascular or endothelial injury, as well as hemostatic regulation disruption. Endothelial damage promotes thrombus formation due to Von Willebrand factor(VWbF), stored in endothelial cells, which is a major contributor to platelet activation and consequently coagulation [27, 28]. This can manifest as inflammation caused by the cytokine storm, stimulation of the complement pathway, or renin-angiotensin-aldosterone system (RAAS) dysregulation [26]. Nucleocapsid proteins in SARS-COV-2 bind to Mannan-binding lectin serine protease 2 (MASP-2), triggering the lectin pathway, while spike proteins activate the alternative complement pathway [23]. Additionally, SARS-COV-2 is endocytosed when it binds to angiotensin-converting enzyme 2 (ACE2) receptors in large concentrations in organs such as the lungs, arteries, and veins. The levels of ACE2 receptors is reduced during this procedure, thereby affecting the RAAS system [26].

When angiotensin-converting enzyme 1 (ACE1) converts angiotensin 1(Ang1) into angiotensin 2 (Ang2), ACE2 counteracts the impact by degrading Ang2 into Ang 1 [1-7] which serves as a vasodilator and anti-inflammatory agent. When SARS-COV-2 decreases ACE2 concentrations, Ang 2 increases, resulting in a loop as it has pro-inflammatory and prothrombotic effects [23, 26, 28]. Finally, some studies suggest a possible imbalance between VWbF and its protease ADAMTS13, with the latter being overwhelmed and resulting in a pro-coagulation state [27]. Thrombosis of abdominal vessels, particularly the portal vein, happens in rare cases as shown in Fig 5.This can result in mesenteric ischemia and further issues. Given the challenge in diagnosing it, clinicians should not disregard this distinction when COVID patients also report severe abdominal pain [29, 24]. Anticoagulants such as unfractionated heparin and low molecular weight heparin are used to treat acute pulmonary embolism (PE). However, the use of anticoagulants as prophylaxis remains to be discussed. [25]

Figure 5: CT scan showing portal vein thrombosis.



3.4. Muscular Hematomas

Extravasated blood within a muscle, muscular hematomas caused by any kind of trauma offer little challenge in management. But, spontaneous muscular hematoma (SMH) can become a life threatening complication [30]. Although common, SMHsseverity factors aren't well known. Small SMH are the most common and can even resolve spontaneously. Voluminous SMH is less frequent, but can even cause hemodynamic instability and in many cases can be life threatening [31]. Risk factors for SMH are multiple: renal insufficiency, hemodialysis, hepatic insufficiency, cardiac insufficiency, disorder of coagulation, congenital collagen disease, and degenerative muscle disease [32, 33, 34, 35, 36]. With the basis of its pathophysiology as microangiopathy [31] that can be caused by any of these factors. COVID-19 as discussed earlier has been shown to cause endothelial damage, increased coagulation, thrombosis, and embolism. But a rare complication of hospitalized COVID-19 patients is SMH [37]. Cases of SMH COVID-19 complications have been coupled with the use of anticoagulation drugs such as heparin or low-molecular-weight heparin (LMWH) that lower the coagulation ability of the blood and make SMH possible [38, 39]. Within COVID-19 cases, the development of SMH was related to the prophylactic use of anticoagulants [39], bringing question to their relative risk to benefit ratio. In other studies, SMH has a 2.1% prevalence [37],in our data, 1 patient out of 222 (0.45%) hospitalized patients developed this complication.SMH was most commonly found

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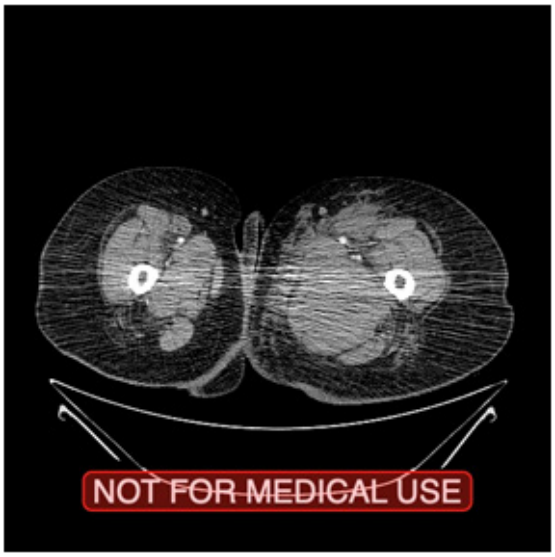
within the rectus sheath [33, 35] or within the ilio-psoas muscles [39]. In our data the case occurred within the thigh and specifically towards the adductor magnus muscle as is shown in Fig 6. Treatment of SMH in first steps consists of discontinuing anticoagulatory treatments. Then transfusion is initiated to counter the possible hypovolemic shock. More severe cases are treated with arterial embolization as it is preferred compared to the surgical option for its minimal invasion, and quick result [39]

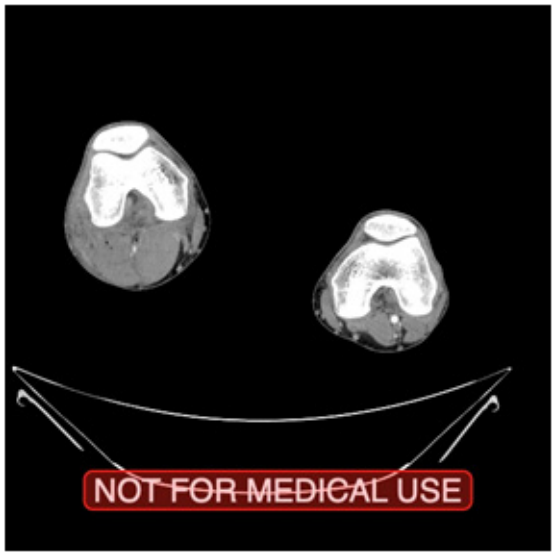
Figure 6: CT scan showing left adductor magnus hematoma.



3.5. Popliteal Artery Stenosis

Stenosis refers to the narrowing of blood vessels caused by an array of factors, which might result in ischemia along with other symptoms. Literature reporting popliteal artery stenosis is quite limited. However, there are documented events that may contribute to and explain such presentation. To begin, COVID has been associated with a cytokine storm that may generate a hypercoagulable state, leading to thrombosis in a variety of body locales and potentially vascular stenosis. Some studies have even shown that rarely affected tissues, such as the subcutaneous tissue around the outer ear, are impacted [40]. Thus, it is possible to extrapolate that to our case of popliteal stenosis, as shown in Fig 7. A different theory highlights the presence of vasculitis, which may result in vessel narrowing [41]. Giant cell arteritis (GCA)is also associated with an enhanced immune response and the release of IL-6 and IL-17. Studies have reported an increase in GCA cases during the outbreak of COVID-19 and recommends that it should be encompassed as part of the differential in individuals with lower limb ischemia who are not at a high risk of atherosclerosis. In the same way as the patient in our case study[42]. Finally, one explanation proposes that the stenosis and ischemia in our case study may be the result of an extrinsic cause, such as an abscess in the same leg as the stenosis. The literature mentions an incidence of such an occurrence in other parts of the body [43], but further research is needed to link COVID-19 to muscle abscess formation before it can be adopted.

Figure 7: Ct scan showing stenosis in the right popliteal artery.



3.6. Muscle Abscess

Muscle abscesses are divided into two subgroups: primary and secondary. Primary are caused by spread of an organism through the blood, while secondary muscle abscess has a multitude of origins from different organ systems and disorders causing the secondary muscle abscess. It can range from osteomyelitis to urinary tract infections [44]. In our data, 1 patient out of 222 (0.45%) hospitalized patients developed this complication. An abscess on the right leg is shown in Fig.8. This case presents a complication of gastrocnemius muscle abscess due to COVID. This complication has not yet been reported in literature, and thus no information about the mechanism of the complication. We can suggest multiple theories that can link COVID and the abscess formation. The first theory , the infected state of COVID patients makes them more prone to superimposed infections [45], leading to a hematologic bacterial infection, and its spread to the muscle causing an abscess. The second theory, COVID can cause vasculitis in the vessels [46] and the chronic inflammation can trigger the abscess formation. The third theory,there might be a direct link between COVID and muscle abscesses, but it has not been elucidated yet. More research is needed to find the mechanism of abscess formation in COVID patients.

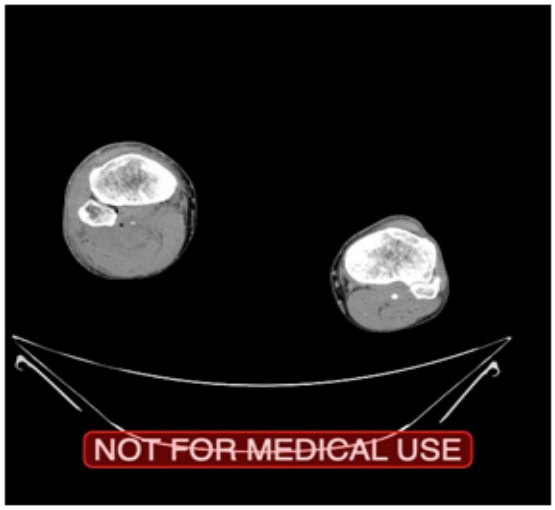


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Figure 8: CT scan showing muscle abscess in the right leg.

4. Conclusion

The rapidity of COVID-19 progression is correlated with extrapulmonary organ injuries and comorbidities. Thus, the identification of various factors that account for multi-organ injuries and preventive and protective measures must be taken into serious consideration. Furthermore, elucidating the mechanisms leading to these extrapulmonary manifestations is of particular importance to help implement proper treatment strategies with personalized approaches and restricting the risk of decompensation.

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