Rheumatology International (2022) 42:905–912
https://doi.org/10.1007/s00296-022-05106-3

Rheumatology
INTERNATIONAL

CASE BASED REVIEW

Concurrent myopathy and inflammatory cardiac disease in COVID-19 patients: a case series and literature review

Ophir Freund[1] · Tali Eviatar[2] · Gil Bornstein[1]

Received: 31 December 2021 / Accepted: 25 February 2022 / Published online: 11 March 2022

Abstract

Adult COVID-19 patients can present with acute muscle and/or cardiac involvement. Our study aims to describe the incidence and characteristics of patients with the co-occurrence of COVID-19 myopathy and inflammatory cardiac disease. We retrospectively reviewed all COVID-19 patients admitted to a large tertiary center to assess the co-occurrence of myopathy and inflammatory cardiac disease. We conducted a literature review of prior relevant case reports. There were three COVID-19 patients with concurrent involvement from our center and five cases in the published literature. Overall, mean age was 57.7 ± 16, four were females (50%) and only two patients (25%) had major relevant comorbidities. Muscle involvement included rhabdomyolysis or myositis and cardiac involvement included myocarditis or pericarditis. Most patients (75%) had no respiratory COVID-19 symptoms. Troponin and creatine phosphokinase levels were higher than twofold of the upper limit of normal for all patients. Steroids were used in the treatment of most patients (75%). All patients had a resolution or improvement of their extra-pulmonary involvement while two (25%) deteriorated due to COVID-19 pneumonia. The incidence for this co-occurrence is 0.07% among hospitalized COVID-19 patients. Patients with these rare COVID-19 simultaneous manifestations have distinct features. They are generally younger, present with extra-pulmonary symptoms and do not have severe respiratory compromise. An underdiagnosis causing treatment delay is possible. Further study is needed.

Keywords COVID-19 · Myopathy · Myositis · Cardiac inflammatory disease · Myocarditis

Introduction

Severe acute respiratory syndrome (COVID-19) caused by the novel coronavirus SARS-CoV-2 is known to cause a wide variety of extra-pulmonary manifestations [1–4]. These can be either related to a specific organ or mimic a systemic inflammatory disease. Cardiac involvement secondary to COVID-19 occur in over 50% of patients with different presentations, manifestations and complications [2, 5, 6]. Up to 16% of these patients can also be asymptomatic [7, 8]. Skeletal muscle myopathy is an additional extra-pulmonary manifestation of COVID-19, occurring in up to one-third of symptomatic patients and ranging from limited myalgia to myositis or rhabdomyolysis, depending on the authors' definition [9, 10]. While involvement of each system separately is well described among COVID-19 patients, their concurrent appearance is not; only a few cases are reported in the literature. Here we report a case series of patients with concurrent myopathy and inflammatory cardiac disease secondary to active COVID-19 infection and review the literature of such cases.

Methods

This is a retrospective case series review in Tel Aviv Sourasky Medical center, a large tertiary center in Israel. Cases were identified from the electronic hospital registry system using the MDClone software [11]. The inclusion process is presented in Fig. 1. We searched the registry for relevant cases among all patients admitted between March 2020 and August 2021 with proven COVID-19 by oropharyngeal SARS-CoV-2 polymerase chain reaction (PCR) swab. Our

✉ Ophir Freund
ophir068@gmail.com

[1] Internal Medicine B, Tel-Aviv Sourasky Medical Center and Sackler Faculty of Medicine, Tel Aviv University, Tel Aviv-Yafo, Israel

[2] Rheumatology Department, Tel Aviv Sourasky Medical Center and Sackler Faculty of Medicine, Tel Aviv University, Tel Aviv-Yafo, Israel

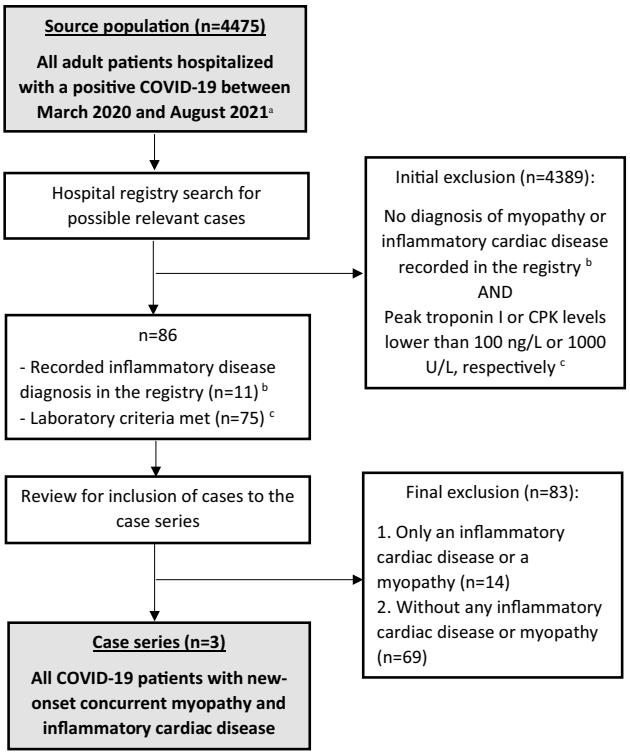

Fig. 1 Flow chart of the inclusion process. *CPK* creatine phosphokinase. [a]Diagnosed by positive oropharyngeal SARS-CoV-2 polymerase chain reaction (PCR) swab. [b]Diagnoses recorded by the treating physician during admission, including myositis, myasthenia gravis, rhabdomyolysis, myopathy, pericarditis, myocarditis, or cardiomyopathy. [c]Regarded as the laboratory criteria

initial search parameters included adults over the age of 18 with a recorded diagnosis of myopathy or inflammatory cardiac disease during admission (including myocarditis, pericarditis, cardiac tamponade, myositis, myasthenia gravis, and rhabdomyolysis). To avoid the loss of other relevant cases, we also searched for patients that had a peak creatine phosphokinase (CPK) level of at least 1000 U/L (normal 35–145 U/L) with a peak troponin I level of at least 100 ng/L (normal < 50 ng/L). Exclusion criteria were prior diagnosis of any chronic myopathy or cardiac inflammatory disease. Medical records of all cases found using the method above were then reviewed to verify the diagnoses and co-occurrence of myopathy and inflammatory cardiac disease. Only patients diagnosed with new onset myopathy and inflammatory cardiac disease during admission were included in the final series. For this purpose, we reviewed the hospital admission note, previous hospitalizations, clinic visits, and laboratory and imaging results. Diagnoses of myopathy or inflammatory cardiac disease were made based on the presence of relevant symptoms, physical examination findings and either laboratory or imaging findings. We defined these diagnostic criteria for a minimum of a probable confidence diagnosis. A probable diagnosis of Idiopathic Inflammatory Myopathies (IIM) relies mainly on patient complaints, physical examination, and laboratory findings and is considered enough to classify patient as having IIM [12]. The study was approved by the institutional review board (TLV-0546-21).

Search strategy

A case-based search was conducted according to the review strategies recommended in the literature [13]. Using the MEDLINE/PubMed, DOAJ and Scopus databases, we searched the literature for reported cases between January 2020 and October 2021, with the following strategy: ((myositis) OR (myasthenia gravis) OR (rhabdomyolysis) OR (myopathy)) AND ((pericarditis) OR (myocarditis) OR (cardiomyopathy)) AND ((Covid-19) OR (SARS-CoV-2)). Twenty-eighth articles were retrieved from MEDLINE/PubMed, 23 from DOAJ and 52 from Scopus. The titles, abstracts, and full texts of all case series / case reports or other studies with case presentation were reviewed. Inclusion criteria of cases were as follows: (1) reports in English describing patients aged ≥ 18 years, (2) detailed case presentation, (3) diagnosis of COVID-19 using a nasopharyngeal swab test or serological examination, (4) new onset concurrent occurrence of myopathy and inflammatory cardiac disease during active COVID-19 disease, and (5) no prior myopathy or inflammatory cardiac disease.

Results

Case series

A total of 4475 patients with COVID-19 diagnosis were admitted in our facility during the specified period and appeared in the MDClone database. Eighty-six cases were found through the hospital registry search (Fig. 1). After review, three cases met our inclusion criteria for the case series. Clinical details of these cases are outlined below and summarized in Table 1.

Case 1: Perimyocarditis, cardiac tamponade and myositis

A 47-year-old female presented with fatigue, myalgia, and fever 5 days after being detected positive for SARS-CoV-2 by oropharyngeal PCR swab. Her medical history included past invasive ductal carcinoma of her left breast (2011) and fibromyalgia. Vital signs included tachycardia of 120 beats per minute, blood pressure 103/87 mmHg with normal O2 saturation at room air and no fever. Bilateral lower limb tenderness on palpation was noted on physical examination. Initial troponin level was 396 ng/L. After several hours, a sudden deterioration occurred with O2 de-saturation of 83% at room air, hypotension of 77/40 mmHg and metabolic

Table 1 Characteristics of COVID-19 patients included in the case-series

	Case 1	Case 2	Case 3
Age	47	41	91
Gender	Female	Female	Male
Comorbidities	Previous breast cancer, fibromyalgia	Polycystic kidney disease with CKD (creatinine 6 mg/dL)	Mild cognitive impairment, Atrial fibrillation, HTN, dyslipidemia, CKD (creatinine 1.5 mg/dL), past smoker (40 pack/years)
Inflammatory cardiac disease	Perimyocarditis, cardiac tamponade	Myocarditis	Pericarditis
Myopathy	Myositis	Myositis	Rhabdomyolysis
Worst ventilatory support	Nasal cannula	None	High-flow nasal cannula
Hospitalization duration (days)	8	18	Death
Max troponin (ng/L)	1625	399*	1057
Max CPK (U/L)	16820	1150*	8575
Max CRP (mg/L)	62.8	151	154
Max LDH (U/L)	1350	1134	1277
Max WBC (K/uL)	14.3	13.7	7.2
Max D-Dimer (mg/L)	1.61	6.3	3.28
Treatment	Dexamethasone, Remdesivir, Tocilizumab	Dexamethasone, Casirivimab and Imedevimab	Dexamethasone

CKD, chronic kidney disease; *HTN*, hypertension; *Max*, maximum; *CPK*, creatine phosphor-kinase; *CRP*, C-Reactive Protein; *LDH*, lactate dehydrogenase; *WBC*, white blood cells

*Tests were taken after two dialysis sessions

acidosis with pH of 6.9. The patient was treated with intravenous fluids and noradrenalin. A bed-side trans-thoracic echocardiogram (TTE) was performed, revealing a moderate amount of pericardial fluid with mitral valve respiratory variation. An urgent pericardiocentesis was carried out with drainage of 350CC serous-bloody fluid and restoration of hemodynamic and respiratory stability. On day 2, second TTE showed minimal residual pericardial fluid and a follow-up ECG showed a QRS complex enlargement in the precordial leads (Fig. 2). Bacterial cultures, acid-fast stain, adenosine deaminase, cytology for malignant cells and PCR of COVID-19 from the pericardial fluid were negative. During the same day, a proximal weakness of upper and lower limbs appeared with worsening of myalgia. On physical examination, strength of bilateral iliopsoas was 2\5 and bilateral deltoid 3\5 with tenderness to palpation. Lab results showed an increase in serum CPK to 9000 U/L peaking at 16,000 on day 3. Testing of other infectious etiologies were negative for viral respiratory panel (Adeno virus, Influenza A and B, Respiratory syncytial virus, human metapneumovirus and parainfluenza), hepatitis C and B (negative Hepatitis B surface antigen and antibody, total core antibody and antibodies for Hepatitis C), Human immunodeficiency virus, cytomegalovirus (negative IgM antibodies) and Epstein-Barr virus (negative IgM antibodies). Laboratory testing for primary rheumatologic causes were negative for rheumatoid factor, antinuclear antibody, low complement C3 and C4 levels and for specific and associated idiopathic inflammatory myopathies antibodies (Mi-2 alpha and beta, TIF1 gamma, MDA5, NXP2, SAE1, Ku, PM-Scl100, PM-Scl75, JO-1, SRP, PL-7, PL-12, EJ, OJ and Ro-52). A computed tomography (CT) scan showed bilateral lung ground-glass opacities consistent with COVID-19, mild pericardial thickening consistent with pericarditis, and edema of the proximal lower limb musculature consistent with myositis (Fig. 3). Treatment with Remdesivir, dexamethasone and tocilizumab were given in accepted dosages per-institutional protocol for severe COVID-19 [14–16]. General improvement was noted on day 5 with decreased myalgia, increase of iliopsoas strength to 4\5 and resolution of upper limb weakness.

Case 2: New left ventricular global functional impairment as a presentation of myocarditis and myositis

A 41-year-old female presented with bilateral lower limb weakness, cough, and fever for 2 days. She was detected positive for SARS-CoV-2 by oropharyngeal PCR swab upon arrival. Her medical history includes chronic kidney disease secondary to polycystic kidney disease with a baseline creatinine of 6 mg/dL. Vital signs were within normal range, bilateral proximal lower limb weakness was noted upon medical examination. Initial laboratory results showed a creatinine of 18.5 mg/dL without any electrolyte abnormalities, venous blood gases with pH of 7.14 and bicarbonate of

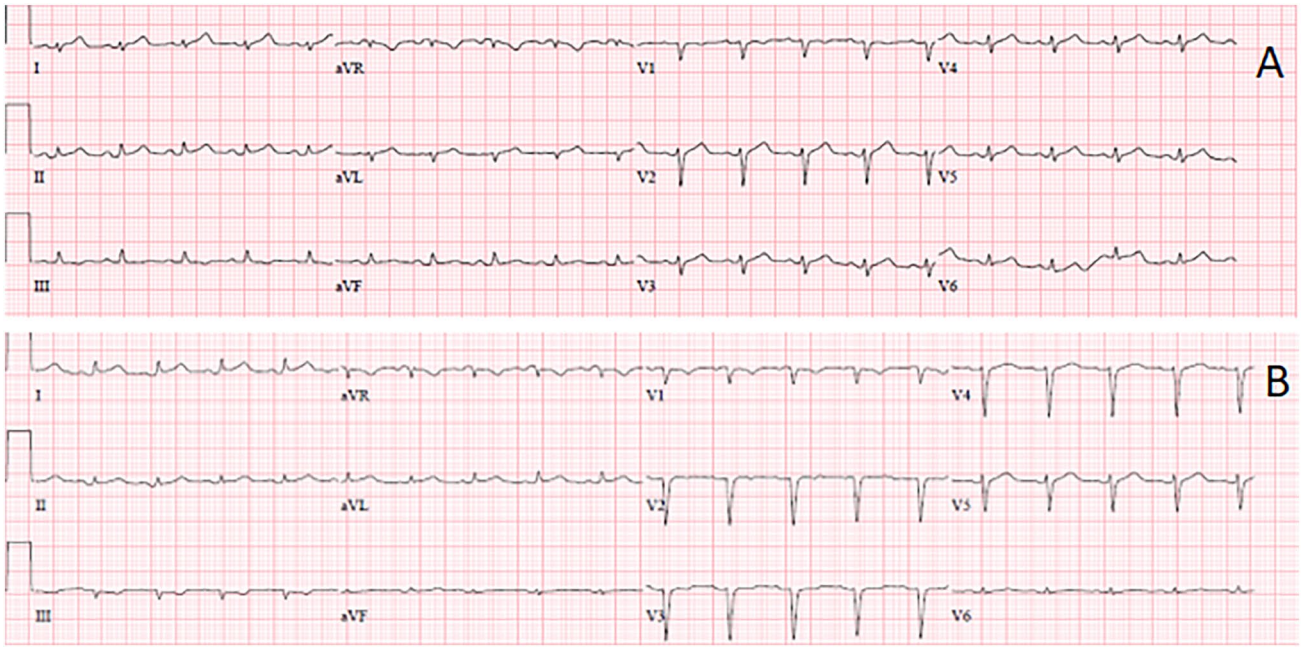

Fig. 2 Electrocardiograms from case 1, upon arrival (**A**) showing low QRS voltage and at day 2 (**B**) showing enlargement of QRS in precordial leads

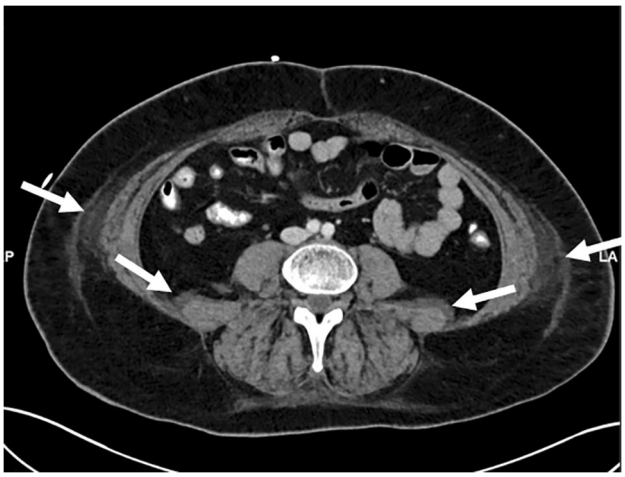

Fig. 3 Computed tomography from case 1, symmetric bilateral edema of the proximal lower limb musculature (white arrows), consistent with myositis

5.7 mmol/L. An urgent dialysis was performed with resolution of acidosis. Treatment with dexamethasone was initiated and a single dose of monoclonal anti-SARS-CoV-2 antibodies (Casirivimab and Imedevimab antibodies) was given due to low anti-SARS-CoV-2 spike IgG levels [17]. The patient completed second dialysis session 12 h after admission to the COVID-19 department. First CPK level, taken on day 2, was 1150 U/L with improvement of lower limbs weakness on day 3. Upon arrival to the COVID-19 department, the patient complained of dyspnea which appeared with minimal effort combined with generalized fatigue. First troponin level taken on day 2 was 399 ng/L. TTE showed severe global LV systolic dysfunction with ejection fraction (EF) of 30%, which was a new finding in comparison with a TEE performed in December 2019. The patient continued dialysis with improvement of symptoms and was transferred to a non-COVID-19 internal medicine department after recovery from COVID-19. Two weeks later, coronary angiography showed a non-significant coronary artery disease and a follow-up TTE showed improvement of global systolic dysfunction with an EF of 40%.

Case 3: Pericarditis and rhabdomyolysis

A 91-year-old male presented with 2 days of extreme bilateral lower limb weakness ("could not stand") and shortness of breath. He was detected positive for SARS-CoV-2 by oropharyngeal PCR swab upon arrival. His medical history included a mild cognitive impairment, paroxysmal atrial fibrillation, hypertension, dyslipidemia, chronic kidney disease with baseline creatinine of 1.5 mg/dL and prior smoking (more than 20 years ago) with 40 pack years. Upon admission abnormal vital signs included fever of 39.2 degrees Celsius and oxygen saturation of 86% at room air elevated to 95% while using oxygen nasal cannula. Blood pressure and heart rate were within normal range. Physical examination was noticeable for bilateral proximal lower limb weakness without signs of

dyspnea or tachypnea. Chest radiograph (CXR) showed an enlarged heart silhouette (Fig. 4). Bedside TTE showed moderate pericardial effusion. Initial lab results included elevated troponin level of 121 ng/L and a CPK level of 5011 U/L, both peaked the following day at 1057 ng/dL and 8575 U/L, respectively. Treatment with Dexamethasone was initiated. Two days after admission, lower limb weakness has improved, and the patient was able to walk with assistance. However, the following day a respiratory deterioration occurred and was attributed to worsening of COVID-19 pneumonia. Oxygen desaturation continued to worsen, and the patient required high flow nasal cannula. Three days later the patient died due to respiratory failure.

Literature review

We identified 26 articles with a case description, and after reviewing, only 5 were included in our case series using the above inclusion criteria. We excluded eight cases of patients with only myopathy or inflammatory cardiac disease, five cases with a known myopathy or inflammatory cardiac disease, four cases of patients without COVID-19, three cases describing the effect of COVID-19 vaccine, and one case of a patient under 18 years. The characteristics of the included cases are summarized in Table 2 [18–22]. Median age was 50 (39–78) years and only one patient had a history of prior cardiac disease. The presenting symptom in all patients was related to one of the extra-pulmonary systems and 80% had no COVID-19-related respiratory symptoms. Steroids were part of the treatment regimen of three patients, colchicine for two patients and hydroxychloroquine for one.

Discussion

In this article, we present the first case series of COVID-19 patients with concurrent myopathy and inflammatory cardiac disease. Our study sample included two young patients, in accordance with the relatively young age of the patients from the reviewed cases (overall median 49, range 39–91). Previous reports showed a wide age variety of COVID-19 patients with any of these manifestations [2, 3]. Overall, only one patient (12.5%) had multiple cardiovascular or neuromuscular comorbidities [18]. In addition, only two patients (25%) did not recover and suffered from further deterioration. These findings are in contrary to the higher rates of cardiovascular comorbidities and worse outcomes as seen in previous studies among COVID-19 patients with cardiac complications [1, 5]. A reasonable explanation might be the exclusion of patients from our study with any vascular complications such as myocardial infraction or stroke or any complication secondary to sepsis or shock. At least one of the extra-pulmonary manifestations occurred for all patients early during the course of the disease, in line with previous findings on COVID-19 related myositis or myocarditis [2].

COVID19 related myopathy and cardiac complications are known to have a strong inflammatory component [10, 23, 24]. Concomitant involvement can also occur in inflammatory myopathies or as a side effect of drugs such as checkpoint inhibitors which further highlight the possible inflammatory (as opposed to infectious) mechanism for this association [25, 26]. In our case series considered together with cases from the literature review and other, most patients did not have a major respiratory complication and only two had critical COVID-19 pneumonia [4, 22]. There might be a different mechanism leading to the involvement of each system in COVID-19, inflammatory or else, which can explain this finding.

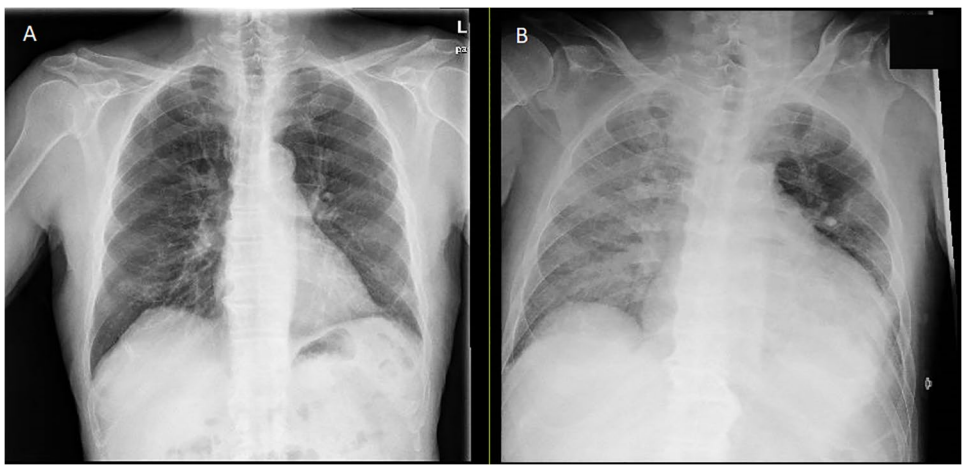

Fig. 4 Chest radiograph from case 3, from 2018 (**A**) and upon arrival (**B**) with enlarged heart silhouette due to pericardial effusion

Table 2 Characteristics of patients from case reports included in our literature review

References	Shabbir et al. [18]	Legrand et al. [19]	Fadiran, O [20]	Arai et al. [21]	Murillo et al. [22]
Age/gender	50 F	39 M	78F	61 M	48 M
Comorbidities	Hypertension, reactive arthritis, prior myopericarditis	None	Major depressive disorder, hypothyroidism, bipolar disorder, dyslipidemia, mild cognitive impairment	Hypertension	None
Inflammatory cardiac disease	Myopericarditis	Myopericarditis	Myocarditis	Pericarditis mimicking fulminant myocarditis	Probable myocarditis
Myopathy	Myositis	Rhabdomyolysis	Rhabdomyolysis	Rhabdomyolysis	Rhabdomyolysis
Max troponin (ng/L)	128	25000	1144	9630	168
Max CPK (U/L)	32230	17070	10650	8318	10768
Main Complaint	Pleuritic central chest pain	Chest pain and dyspnea	Weakness	Not specified	General malaise, myalgias, dry cough, progressive dyspnea
Worst ventilatory support	None	None	None	*ECMO	Mechanical ventilation
Treatment	Ibuprofen, prednisolone, colchicine	Colchicine	Remdesivir, dexamethasone	Cortico-steroids, dantrolene	Ceftriaxone, azithromycin, oseltamivir, and hydroxychloroquine

CPK creatine phosphor-kinase, *ECMO* extracorporeal membrane oxygenation

*Used due to cardiogenic shock and not because of COVID-19 pneumonia

Other infectious agents, including viruses, bacteria and parasites, are known to trigger autoimmunity, which may also play a key role in the development of extra-pulmonary COVID-19 disease [27]. Molecular mimicry is a possible mechanism causing autoimmunity in COVID-19 disease, with known shared epitopes between human and SARS-COV-2 proteins [2, 23, 28]. COVID-19 has also been associated with activation of the innate immunity, which may transform into autoimmunity [27]. Another proposed mechanism for autoimmunity is the persistence of SARS-COV-2 in the host long before it is detected, leading to prolong innate immunity activation [3].

Myopathy and inflammatory cardiac disease in covid-19 are common, while their concurrent involvement is rare, and its incidence is yet to be described. Romero-Sánchez et al. demonstrated that among hospitalized patients with COVID-19, muscle damage was the most prominent neuromuscular manifestation, with myopathy and rhabdomyolysis at incidence of 3.1% and 1.1%, respectively [30]. Another study that summarized COVID-19 patients who undergone echocardiography found myocarditis and cardiac tamponade in 3% and 1% of the patients, respectively [31]. Based on our analysis among all COVID-19 patients in our medical center, the incidence of patients with concurrent involvement was 0.07%, although there may have been underdiagnosis of such cases, effecting early treatment and outcomes.

Cases with simultaneous muscle and cardiac involvement might present a diagnostic challenge. Myositis can be overlooked unless its diagnosis is supported by laboratory or directed imaging and functional tests. In comparison with the reviewed cases and previous studies, patients in our case series had atypical cardiac complaints, further delaying their diagnosis [24, 32]. Troponin and CPK levels were high among all patients and TTE was effective in demonstrating the cardiac pathology, all of which seem to be reliable and basic diagnostic tools that are available in most clinical settings. While further investigations with modalities such as magnetic resonance imaging (MRI) are needed to establish a diagnosis, they are usually not available for most COVID-19 patients due to logistical or resources issues.

Therapeutic dilemmas may appear as well. Non-steroidal anti-inflammatory drugs (NSAIDs) and colchicine are considered first-line treatments for acute pericarditis [33] while glucocorticoids are selected as second-line treatment (due to a higher recurrence rate during steroids tapering down). However, no specific treatment was proven for myocarditis. In addition, a treatment which is suitable for one system might have an adverse effect on the other. NSAIDS usage for pericardial involvement in patients otherwise healthy might worsen kidney function if a concurrent rhabdomyolysis exists. In our report, dexamethasone was the most frequent drug of choice, as it was used for all cases in our series and in three others from the review. Steroids are widely used in severe COVID-19

and are accepted as a first-line therapy in cases of myositis attributed to other etiologies [15, 34]. It seems reasonable to use dexamethasone for such cases, as already described for COVID-19-related myocarditis [35]. No evidence of adverse effects was noted for any of the treatments probably due to short-term use and immediate quit from treatment.

Our report has several limitations. First, our initial inclusion criteria and retrospective nature of this study may fail to recognize other patients with milder signs and symptoms, or without a declared diagnosis, but we have tried to overcome this by adding the laboratory criteria. Second, diagnosis of any extrapulmonary involvement was based mostly on clinical and laboratory findings without histopathologic, electromyographic or MRI confirmation. To ascertain the relationship between COVID-19 disease and the time onset of the inflammatory disease, we reviewed medical records prior to admission. Still, this relationship may be interrupted by a sub-clinical disease or unreported symptoms that were present before COVID-19 disease onset. The follow-up time is short and restricted to the hospitalization period which renders our study unable to assess for recurrence of symptoms and signs and late prognosis. We also acknowledge that our study design and the small sample size was not suitable to assess the efficacy of the different treatments.

In conclusion, COVID-19 can manifest a myriad of inflammatory complications, amongst myopathy and inflammatory cardiac disease; their concurrent involvement seems to be rare yet probably underdiagnosed. Our study raises awareness to the unique characteristics of these cases and may shed light on the shared pathophysiology of these COVID-19 complications. Larger studies and case series are needed to define the best treatment strategy for this myopathic complication and its long-term prognosis.

Funding This study was not funded by any source.

Declarations

Conflict of interest Ophir Freund, Tali Eviatar, and Gil Bornstein declare that they have no conflict of interest.

Ethical approval The study was approved by the Sourasky medical center institutional review board (TLV-0546-21). The study has been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. This is a retrospective study and details that might disclose the identity of the subjects under study was omitted as possible.

References

1. Gupta A, Madhavan MV, Sehgal K et al (2020) Extrapulmonary manifestations of COVID-19. Nat Med 26(7):1017–1032. https://doi.org/10.1038/s41591-020-0968-3
2. Ramos-Casals M, Brito-Zerón P, Mariette X (2021) Systemic and organ-specific immune-related manifestations of COVID-19. Nat Rev Rheumatol 17(6):315–332. https://doi.org/10.1038/s41584-021-00608-z
3. Ahmed S, Zimba O, Gasparyan AY (2021) COVID-19 and the clinical course of rheumatic manifestations. Clin Rheumatol 40(7):2611–2619. https://doi.org/10.1007/s10067-021-05691-x
4. Kocyigit BF, Akyol A (2021) Reactive arthritis after COVID-19: a case-based review. Rheumatol Int 41(11):2031–2039. https://doi.org/10.1007/s00296-021-04998-x
5. Aghagoli G, Gallo Marin B, Soliman LB, Sellke FW (2020) Cardiac involvement in COVID-19 patients: risk factors, predictors, and complications : a review. J Card Surg 35(6):1302–1305. https://doi.org/10.1111/jocs.14538
6. Long B, Brady WJ, Koyfman A, Gottlieb M (2020) Cardiovascular complications in COVID-19. Am J Emerg Med 38(7):1504–1507. https://doi.org/10.1016/j.ajem.2020.04.048
7. Erdol MA, Ozbay MB, Yayla C et al (2021) Cardiac involvement in MRI in young population after COVID-19: a single tertiary center experience. Echocardiography 38(8):1327–1335. https://doi.org/10.1111/echo.15160
8. Puntmann VO, Carerj ML, Wieters I et al (2020) Outcomes of cardiovascular magnetic resonance imaging in patients recently recovered from coronavirus disease 2019 (COVID-19). JAMA Cardiol 5(11):1265–1273. https://doi.org/10.1001/jamacardio.2020.3557
9. Rivas-García S, Bernal J, Bachiller-Corral J (2020) Rhabdomyolysis as the main manifestation of coronavirus disease 2019. Rheumatology 59(8):2174–2176. https://doi.org/10.1093/rheumatology/keaa351
10. Saud A, Naveen R, Aggarwal R, Gupta L (2021) COVID-19 and Myositis: What We Know So Far. Curr Rheumatol Rep 23(8):63. https://doi.org/10.1007/s11926-021-01023-9
11. MdClone - Dynamic Data Exploration. https://www.mdclone.com/. Accessed 12 Dec 2021
12. Lundberg IE, Tjärnlund A, Bottai M et al (2017) EULAR/ACR classification criteria for adult and juvenile idiopathic inflammatory myopathies and their major subgroups HHS public access. Ann Rheum Dis 76(12):1955–1964. https://doi.org/10.1136/annrheumdis-2017-211468.EULAR/ACR
13. Gasparyan AY, Ayvazyan L, Blackmore H, Kitas GD (2011) Writing a narrative biomedical review: considerations for authors, peer reviewers, and editors. Rheumatol Int 31(11):1409–1417. https://doi.org/10.1007/s00296-011-1999-3
14. Abani O, Abbas A, Abbas F et al (2021) Tocilizumab in patients admitted to hospital with COVID-19 (RECOVERY): a randomised, controlled, open-label, platform trial. Lancet 397(10285):1637–1645. https://doi.org/10.1016/S0140-6736(21)00676-0
15. Group RC (2021) Dexamethasone in hospitalized patients with Covid-19. N Engl J Med 384(8):693–704. https://doi.org/10.1056/nejmoa2021436
16. Beigel JH, Tomashek KM, Dodd LE et al (2020) Remdesivir for the treatment of Covid-19—final report. N Engl J Med 383(19):1813–1826. https://doi.org/10.1056/nejmoa2007764
17. O'Brien MP, Forleo-Neto E, Musser BJ et al (2021) Subcutaneous REGEN-COV antibody combination to prevent Covid-19. N Engl J Med 385(13):1184–1195. https://doi.org/10.1056/nejmoa2109682
18. Shabbir A, Camm CF, Elkington A et al (2020) Myopericarditis and myositis in a patient with COVID-19: a case report. Eur Hear J Case Rep 4(6):1–6. https://doi.org/10.1093/ehjcr/ytaa370
19. Legrand F, Chong-Nguyen C, Ghanem N (2020) Myopericarditis, rhabdomyolysis, and acute hepatic injury: sole expression of a SARS-CoV-2 infection. Circ Cardiovasc Imaging 13(7):1–3. https://doi.org/10.1161/CIRCIMAGING.120.010907

20. Fadiran O (2021) An atypical case of COVID-19 induced pancytopenia rhabdomyolysis and myocarditis. Cureus 13(1):3–8. https://doi.org/10.7759/cureus.12455
21. Arai R, Murata N, Yamada A et al (2021) Multimodal findings of COVID-19-related rhabdomyolysis complicated with pericarditis mimicking fulminant myocarditis. Circ Rep 3(7):419–420. https://doi.org/10.1253/circrep.cr-21-0055
22. Murillo F, Ramos G, Pozo JLD, Luis Manuel Valdez JZ (2020) SARS-CoV-2 infection with associated rhabdomyolysis and probable myocarditis. Eur J Case Reports Intern Med. 7(9):001867. https://doi.org/10.12890/2020_001867
23. Shah S, Danda D, Kavadichanda C, Das S, Adarsh MB, Negi VS (2020) Autoimmune and rheumatic musculoskeletal diseases as a consequence of SARS-CoV-2 infection and its treatment. Rheumatol Int 40(10):1539–1554. https://doi.org/10.1007/s00296-020-04639-9
24. Hakmi H, Sohail A, Brathwaite C, Ray B, Abrol S (2020) Cardiac tamponade in COVID-19 patients: Management and outcomes. J Card Surg 35(11):3183–3190. https://doi.org/10.1111/jocs.14925
25. Johnson DB, Balko JM, Compton ML et al (2016) Fulminant myocarditis with combination immune checkpoint blockade. N Engl J Med 375(18):1749–1755. https://doi.org/10.1056/nejmoa1609214
26. Kurth BJ, Wagler V, Keith M (2016) Cardiac tamponade as a manifestation of severe dermatomyositis. BMJ Case Rep. https://doi.org/10.1136/bcr-2016-216860 (**Published online**)
27. Sener AG, Afsar I (2012) Infection and autoimmune disease. Rheumatol Int 32(11):3331–3338. https://doi.org/10.1007/s00296-012-2451-z
28. Angileri F, Legare S, Marino Gammazza A, Conway de Macario E, Macario A Jl, Cappello F (2020) Molecular mimicry may explain multi-organ damage in COVID-19. Autoimmun Rev 19(8):102591. https://doi.org/10.1016/j.autrev.2020.102591
29. Birra D, Benucci M, Landolfi L et al (2020) COVID 19: a clue from innate immunity. Immunol Res 68(3):161–168. https://doi.org/10.1007/s12026-020-09137-5
30. Romero-Sánchez CM, Díaz-Maroto I, Fernández-Díaz E et al (2020) Neurologic manifestations in hospitalized patients with COVID-19: the ALBACOVID registry. Neurology 95(8):e1060–e1070. https://doi.org/10.1212/WNL.0000000000009937
31. Dweck MR, Bularga A, Hahn RT et al (2020) Global evaluation of echocardiography in patients with COVID-19. Eur Heart J Cardiovasc Imaging 21(9):949–958. https://doi.org/10.1093/ehjci/jeaa178
32. Chen C, Zhou Y, Wang DW (2020) SARS-CoV-2: a potential novel etiology of fulminant myocarditis. Herz 45(3):230–232. https://doi.org/10.1007/s00059-020-04909-z
33. Adler Y, Charron P, Imazio M et al (2015) 2015 ESC Guidelines for the diagnosis and management of pericardial diseases. Eur Heart J 36(42):2921–2964. https://doi.org/10.1093/eurheartj/ehv318
34. Oddis CV (2016) Update on the pharmacological treatment of adult myositis. J Intern Med 280(1):63–74. https://doi.org/10.1111/joim.12511
35. Kamarullah W, Nurcahyani, Mary Josephine C, Bill Multazam R, Ghaezany Nawing A, Dharma S (2021) Corticosteroid therapy in management of myocarditis associated with COVID-19; a systematic review of current evidence. Arch Acad Emerg Med. 9(1):32. https://doi.org/10.22037/aaem.v9i1.1153