COVID-19 associated severe rhabdomyolysis in a young male with Class III obesity: a case report

Rachel Smith[1,2], Daniel Morris[1], Santhana Kannan[1]

[1]Sandwell and West Birmingham NHS Trust, Birmingham, UK; [2]Department of Critical Care, Sandwell General Hospital, West Bromwich, UK

Contributions: (I) Conception and design: R Smith, S Kannan; (II) Administrative support: R Smith, D Morris; (III) Provision of study materials or patients: R Smith, S Kannan; (IV) Collection and assembly of data: R Smith, D Morris; (V) Data analysis and interpretation: All authors; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

Correspondence to: Dr. Rachel Smith, BMBS. Department of Critical Care, Sandwell General Hospital, Hallam Street, West Bromwich B71 4HJ, UK; Sandwell and West Birmingham NHS Trust, Birmingham, UK. Email: rachelhelensmith@yahoo.co.uk.

Background: Hospital admissions due to coronavirus disease 2019 (COVID-19) infection have reduced steadily as a consequence of vaccinations and weakened variant of the virus. However, patients continue to present with 'incidental COVID-19 infection' without features of pulmonary involvement. Rhabdomyolysis has been reported with COVID-19 infection though initial reports were associated with severe respiratory manifestations of the virus, mostly in older patients. We present this report of suspected, severe rhabdomyolysis induced acute kidney injury (AKI), secondary to COVID-19 infection but without signs of pneumonia, in a young patient with Class III obesity [body mass index (BMI): 66 kg/m^2].

Case Description: A male in his late twenties presented with a 5-day history of progressive breathlessness and feeling generally unwell. He was found to be COVID-19 positive but had no signs of pneumonia. He had high levels of creatine kinase (>426,700 IU/L) and severe AKI. Following hospital presentation, he deteriorated rapidly and required admission to intensive care. He went on to required renal replacement therapy (RRT) for 29 days. Despite the severity of his illness, he made a good recovery without the need for long term dialysis. There are no similar case reports in the literature.

Conclusions: This case highlights the possibility of rhabdomyolysis-induced severe AKI in incidental COVID-19 infection in a young patient with previously normal renal function. With a high index of suspicion and timely intervention, these patients can recover well.

Keywords: Rhabdomyolysis; coronavirus disease 2019 (COVID-19); obesity; case report

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Introduction

Hospital admissions due to coronavirus disease 2019 (COVID-19) infection have reduced steadily. This has been attributed to a combination of vaccination and weakened variant of the virus (1,2). Patients continue to present with 'incidental COVID-19 infection' without features of severe respiratory compromise. Although COVID-19 has been associated with rhabdomyolysis, most of the initial reports were in patients who had been admitted to hospital with severe COVID-19 infection features involving the lungs (3,4). We present this report of suspected, severe rhabdomyolysis induced acute kidney injury (AKI) secondary to COVID-19 infection but without signs of pneumonia, in a young patient with Class III obesity. Though there have been other reports of rhabdomyolysis without respiratory compromise (5-7), there are no previous known episodes of COVID-19 associated rhabdomyolysis in patient less than 30 years old leading to requirement of renal replacement therapy (RRT).

This aim of this report is to emphasise the importance of early recognition of rhabdomyolysis in incidental

COVID-19 infection, without evidence of pneumonia, and highlight that this can occur in patients of a young age. We present this case in accordance with the CARE reporting checklist (available at https://jeccm.amegroups.com/article/view/10.21037/jeccm-23-146/rc).

Case presentation

A male in his late twenties, 1.84 m tall and weighing 225 kg [body mass index (BMI): 66 kg/m^2], presented to the emergency department (ED) in the latter part of 2022 with a 5-day history of feeling unwell and progressive breathlessness. He had developed vomiting, diarrhoea and moderate intermittent abdominal pain which he attributed to a takeaway meal. He was vomiting about twice a day with episodes of diarrhoea described as 'infrequent'. There was no history of fever, cough, sore throat, headache, rash or blood in his stools. During the same period, his urine output had decreased significantly and had become concentrated. He reported pain in the anterior aspect of his left leg but there was no history of trauma. There was no other history of myalgia. The takeaway meal was not shared with friends or family, and they all had remained well. He had no other co-morbidities and was not on any regular medications.

Eighteen months prior to this admission, he had been treated for one episode of cellulitis of his right leg with oral antibiotics in the community. There was no history suggestive of obstructive sleep apnoea or obesity hypoventilation syndrome. He had not previously been admitted to hospital with similar symptoms. He had no allergies and there was no history of recent travel or exposure to pets. He was usually independently mobile requiring no aids and denied any limitation to his exercise tolerance. There was no history of falls. He had never smoked and did not consume alcohol. He was single, not in active relationship, lived with his family and worked full time. He had received two doses of Pfizer COVID-19 vaccination in 2021. He also reported two prior COVID-19 infections in early and mid-2022 confirmed by lateral flow test at home. He reported coryzal symptoms that self resolved within a week and did not require hospital admission for these infections.

On admission, he was fully conscious but breathless at rest. Vital signs showed a regular heart rate (HR) of 137 beats/min, respiratory rate (RR) 40 breaths/min, oxygen saturation (SpO_2) of 99% on room air, blood pressure (BP) 103/71 mmHg, and temperature 36.5 ℃. He had dry mucous membranes, but his capillary refill time was <2 seconds peripherally. His chest was clear on auscultation. There was an area of erythema about 15 cm wide anteriorly in his left leg extending from just distal to tibial tuberosity down to the ankle suggestive of cellulitis. The erythema was not circumferential. The left leg was mildly tender on palpation. There were no signs of underlying collection, crepitus or discharge. Both legs were symmetrical and right leg was normal on examination. There were no other areas of skin or pressure damage elsewhere in the body.

Results of blood investigations are summarised in *Table 1*. Of note, he had creatinine kinase (CK) of 202,921 IU/L, which rose to >426,700 IU/L (maximum measurable range) the following day. This CK value was repeated and confirmed to ensure no laboratory errors. Other results were suggestive of AKI. He did have abnormal ALT of 255 U/L (normal <41 U/L), and D-dimer 35.12 μgFEU/mL (range, 0.0–0.05 μgFEU/mL). Electrocardiogram (ECG) showed sinus tachycardia, chest X-ray was unremarkable and computed tomography (CT) pulmonary angiogram did not show pulmonary embolism or other pathology. CT scan of abdomen and pelvis did not show any abnormality. He was found to be COVID-19 positive on a lateral flow test. He had an echocardiogram which showed non dilated left heart with preserved left ventricular systolic and diastolic function, a non-dilated right heart with preserved right ventricular systolic function and no valvular abnormalities. Blood cultures on admission grew staphylococcus epidermidis

Highlight box

Key findings

- Severe rhabdomyolysis causing acute kidney injury (AKI) requiring renal replacement therapy can occur alongside incidental coronavirus disease 2019 (COVID-19) infection in young patients with no previous renal impairment. With treatment there can be full recovery of the patient and renal function.

What is known and what is new?

- It is known that rhabdomyolysis is an atypical feature of COVID-19 infection.
- It is known that COVID-19 associated rhabdomyolysis is associated with high mortality.
- Rhabdomyolysis causing severe acute kidney injury requiring renal replacement therapy can occur in cases of COVID-19 infection in young patients with mild or no respiratory manifestations.

What is the implication, and what should change now?

- A high index of suspicion is required in patients with AKI and COVID-19 infection, even if this may be an incidental finding.

Table 1 Laboratory results

Test	Reference range	Previous year results	On admission to hospital	Admission to ICU	Day 1 ICU admission	Day 5 ICU admission	Day 8 ICU admission	Day 13 ICU admission
Haemoglobin (g/L)	125–180	143	156	140	139	102	90	77
White cell count ($\times 10^9$/L)	4.0–11.0	13	12.5	12.3	13.8	15.9	17.6	9.6
C-reactive protein (mg/L)	<5	17	405	387	337	140	99	137
Sodium (mmol/L)	133–146	139	132	129	134	136	137	138
Potassium (mmol/L)	3.5–5.3	4.2	5.3	5.3	5.4	3.8	4.1	6.1
Creatinine (µmol/L)	44–133	74	356	560	628	589	280	463
Urea (mmol/L)	2.5–7.8	3.9	11	15.8	18	25.4	14.9	26.2
eGFR (mL/min/1.73 m^2)	Normal >60	>90	18	11	9	10	24	13
Creatinine kinase (IU/L)	40–320	–	202,921	–	>426,700*	77,969	1,228	–
Calcium (mmol/L)	2.2–2.6	–	1.47	–	1.55	2.16	2.32	2.06
Phosphate (mmol/L)	0.8–1.5	–	3.47	–	3.1	0.97	0.91	2.22
FiO_2	–	–	0.21	0.3	0.21	0.21	–	–
ABG pH	7.35–7.45	–	7.39	7.31	7.01	7.37	–	–
ABG PO_2 (kPa)	10–14	–	12.3	15	10	9.7	–	–
ABG pCO_2 (kPa)	4.5–6.4	–	2.29	1.92	9.87	4.57	–	–
ABG bicarbonate (mmol/L)	22–26	–	10.2	7.2	18.3	19.7	–	–
ABG base excess (mmol/L)	−2 to +2	–	−11.7	−16	−13.8	−4.8	–	–
ABG lactate (mmol/L)	<1.5	–	2.6	2	2.3	1.7	–	–

*, our laboratory could only process creatinine kinase levels up to 426,700 IU/L. ICU, intensive care unit; eGFR, estimated glomerular filtration rate; FiO_2, fraction of inspired oxygen; ABG, arterial blood gas; PO_2, partial pressure of oxygen; pCO_2, partial pressure of carbon dioxide.

which was a suspected contaminant. There was no growth from repeat blood cultures. Stool culture was negative. Blood film showed a reactive picture with no schistocytes or evidence of thrombotic thrombocytopenia purpura. His platelet count was normal and there was no evidence of haemolysis.

Despite fluid resuscitation, he remained oliguric. He was commenced on intravenous co-amoxiclav which was changed to piperacillin tazobactam. He was transferred to intensive care unit (ICU) due to lack of significant improvement. His ICU admission observations were RR 45 breaths/min, HR 125 beats/min, SpO_2 99% on air, BP 115/73 mmHg and temperature 38.3 ℃. Continuous veno-venous haemofiltration (CVVH) with citrate anticoagulation was commenced. He subsequently required noradrenaline infusion at 0.14 µg/kg/minute to maintain BP. His condition worsened on day one of ICU admission with respiratory failure [RR 50 breaths/min, arterial partial pressure of carbon dioxide (pCO_2) 9.87 kPa], increasing vasopressor requirement and alteration in consciousness level. He required endotracheal intubation and mechanical ventilation. Argipressin at 1.8 units/h and adrenaline 0.1 µg/kg/min infusions were added. Most of the clinical parameters rapidly improved and he was extubated 24 h later. CK levels and left leg erythema took a week to improve. He continued to require intermittent CVVH for another 29 days.

He was discharged from hospital 8 weeks later and he returned to his baseline state of health. About 6 weeks after discharge, he required readmission to hospital for progressive breathlessness and worsening cough for 3 weeks. There was no history of fever, diarrhoea or vomiting. His admission observations were HR 142 beats/min, RR 33 breaths/min, BP 116/64 mmHg and saturations 97% on fraction of inspired oxygen (FiO_2) of 0.28. His D-dimer was 8.66 µgFEU/mL (range, 0.0–0.05 µgFEU/mL) but there was no evidence of deep vein thrombosis. Lateral flow

test was negative for COVID-19. There were no signs of cellulitis or rhabdomyolysis and his kidney function was normal. He had a CT pulmonary angiogram which showed bilateral pulmonary embolism. Shortly after admission, his BP dropped to 80/50 mmHg. Echocardiogram showed right ventricular dilatation and impairment. Intravenous thrombolysis was administered using Alteplase 10 mg slow bolus followed by 90 mg as infusion. Systemic anticoagulation was maintained using unfractionated heparin infusion. His condition gradually improved and he did not require ICU admission. He was discharged home after 12 days on warfarin protocol. Further clinical course was uneventful.

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). As the case report involves routinely collected non-identifiable clinical data, no ethical approval was required under the UK policy framework for Health and Social Care. Written informed consent was obtained from the patient for publication of this case report. A copy of the written consent is available for review by the editorial office of this journal.

Discussion

Rhabdomyolysis presented with COVID-19 infection has most commonly been documented particularly in older males (8,9). Our case report demonstrates an episode of COVID-19 associated rhabdomyolysis in a young male patient. A recent literature review found that the average age in 38 patients with COVID-19 infection and rhabdomyolysis was 55.9 years. In this review there were only two patients younger than 30 years, neither with evidence of AKI (8). There is another report with a 28-year-old obese (BMI: 46 kg/m^2) female with AKI but did not require RRT (10). The three reports mentioned of patients less than 30 years had evidence of lobar pneumonia and presented with respiratory manifestations of COVID-19. Our patient did not have evidence of lung involvement on admission and only required invasive ventilation due to increased work of breathing and respiratory failure. Lung imaging in our patient had not shown pathology suggestive of COVID-19 and the fact that he was extubated 24 h following intubation also supports the lack of severe lung involvement due to COVID-19.

Evidence now supports that there is no direct correlation between the severity of COVID-19 illness and the incidence of rhabdomyolysis (8,11). There are other reports of COVID-19 associated rhabdomyolysis without lung manifestations (5-7). However, severe rhabdomyolysis requiring RRT in a young patient with a very high BMI has not been reported.

In some instances, the rhabdomyolysis in COVID-19 infection was delayed up to 2 weeks (12,13). There have also been reports of rhabdomyolysis after COVID-19 vaccination with the interval typically being 1–2 days (14-16). Our patient had two doses of vaccine and remained well following these. There is one report of COVID-19 associated rhabdomyolysis in a lady with history of recurrent rhabdomyolysis following influenza infection (8). Our patient did not have any rhabdomyolysis with previous COVID-19 or other viral infections.

The exact pathophysiological relationship between COVID-19 and the development of rhabdomyolysis is not clear (17-19). As CK levels increase, the probability of developing AKI becomes greater and increases the need of requiring RRT (20). Our patient had a CK 202,921 IU/L on arrival to ED which rose to >426,700 IU/L on day one of ICU admission accompanied by AKI [estimated glomerular filtration rate (eGFR) of 9 mL/min/1.73 m^2]. One study showed that 54% of patients with COVID-19 and AKI needed ongoing RRT at discharge (21). Another case series of 38 patients with COVID-19 associated rhabdomyolysis reported 47% mortality with 5% requiring long term hemodialysis (8). A meta-analysis showed the risk of death in patients with COVID-19 and AKI was significantly increased, with an odds ratio of 11.05 (22). In a retrospective observational study of 140 patients with COVID-19, rhabdomyolysis patients had a higher in-hospital mortality (9).

Class III obesity placed our patient at greater risk for development of rhabdomyolysis and may have been a factor which contributed to his need for RRT (1). There are multiple reports relating to rhabdomyolysis in obese patients post bariatric surgery, knee arthroplasty and urological surgery but no documentation of spontaneous rhabdomyolysis linked to obesity (23-25). In patients with rhabdomyolysis due to exertional heat stroke, raised D-dimer was a marker for AKI (26) which might explain the very high D-dimer levels in our patient.

Necrotising fasciitis was initially considered as a possible cause of the rhabdomyolysis. However, there were no other indicators. Inflammatory myopathies were considered but there was no history of muscle weakness or skin involvement. Haemolytic uraemic syndrome was considered due to his

presentation with diarrhoea, however, there was no evidence of haemolysis and stool culture was negative. Rhabdomyolysis has on occasion been linked to gastrointestinal (GI) infections. The most commonly reported incidence of GI infection induced rhabdomyolysis linked to salmonella (27) but our patient tested negative for this.

It is possible that diarrhoea and vomiting were features of COVID-19 infection in our patient (28) and that the takeaway meal prior to the onset of illness was a coincidence. This is difficult to confirm as the meal was not shared with friends or family. It is unlikely that the small number of episodes of vomiting and diarrhoea in our patient would have been sufficient to cause AKI in someone with previously good renal function although it may have been an aggravating factor for the renal injury due to other cause.

Similar to our patient, delayed pulmonary embolism following COVID-19 infection has been reported before (29) and COVID-19 increases the risk (30). High D-dimer levels have been proposed as a marker for pulmonary embolism (31). Hence a decision was taken to do a CT pulmonary angiogram even though oxygenation was not an issue (32). An increased risk of pulmonary embolism is known to be associated with RRT, particularly long-term hemodialysis (33). Though our patient had not required RRT for 10 weeks when he re-presented to hospital, it is an important contributing factor to consider.

Conclusions

In summary, this report highlights the rare instance of severe rhabdomyolysis-induced severe AKI associated with COVID-19 infection in a young patient with very high BMI of 66 kg/m^2, without evidence of pneumonia. This report adds to the literature that there is no correlation between respiratory manifestation of COVID-19 and severity of rhabdomyolysis. It emphasizes the need for a high index of suspicion in such patients as the risk of mortality is high. With timely detection and intervention, these patients can recover well.

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Footnote

Reporting Checklist: The authors have completed the CARE reporting checklist. Available at https://jeccm.amegroups.com/article/view/10.21037/jeccm-23-146/rc

Peer Review File: Available at https://jeccm.amegroups.com/article/view/10.21037/jeccm-23-146/prf

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://jeccm.amegroups.com/article/view/10.21037/jeccm-23-146/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). As the case report involves routinely collected non-identifiable clinical data, no ethical approval was required under the UK policy framework for Health and Social Care. Written informed consent was obtained from the patient for publication of this case report. A copy of the written consent is available for review by the editorial office of this journal.

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