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Original scientific paper

RABDOMYOLISIS AND ACUTE RENAL FAILURE IN A PATIENT WITH COVID-19

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Abstract

COVID-19 is characterized by a hyperactive immune reaction, causing a generalized systemic inflammatory response that most often manifests clinically as a febrile syndrome with respiratory symptoms, but there is a wide range of clinical concomitants that can equally appear as a consequence of Sars viral infection. CoV-2. Not sparing any organ system, Sars nCoV-2, in addition to the most common respiratory component of clinical presentation, equally affects other peripheral systems such as the cardiovascular system, the central nervous system, kidneys, muscles, etc. Even in the presence of the previous strains, cases of rhabdomyolysis and kidney infection were detected, but from the clinical experiences so far, with the appearance of the omicron variant in clinical practice, the frequency of patients whose clinical picture was dominated by myositis significantly increased, and in a certain group of older patients and rhabdomyolysis affecting kidney function. Rhabdomyolysis is a life-threatening disorder that presents with myalgia, fatigue, and pigmenturia, and may also present as acute renal failure. Precipitating factors for rhabdomyolysis include autoimmune myopathies, septicemia, electrolyte abnormalities, substance abuse, alcohol use, or infection. A viral infection, especially an infection with the influenza virus, can lead to rhabdomyolysis. Through this case report, the occurrence of rhabdomyolysis with acute real weakness in a patient with a severe form of COVID19 will be evaluated.

Keywords: COVID-19, immune reaction, inflammatory

Case report

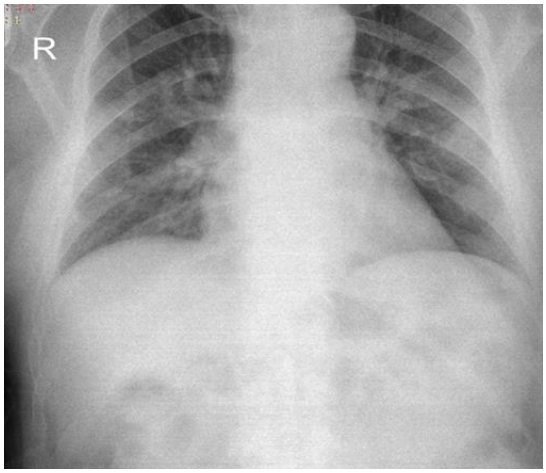
Introduction

COVID-19 is characterized by a hyperactive immune reaction, causing a generalized systemic inflammatory response that most often manifests clinically as a febrile syndrome with respiratory symptoms, but there is a wide range of clinical concomitants that can equally appear as a consequence of Sars viral infection. CoV-2. Not sparing any organ system, Sars nCoV-2, in addition to the most common respiratory component of clinical presentation, equally affects other peripheral systems such as the cardiovascular system, the central nervous system, kidneys, muscles, etc. Even in the presence of the previous strains, cases of rhabdomyolysis and kidney infection were detected, but from the clinical experiences so far, with the appearance of the omicron variant in clinical practice, the frequency of patients whose clinical picture was dominated by myositis significantly increased, and in a certain group of older patients and rhabdomyolysis affecting kidney function. Rhabdomyolysis is a life-threatening disorder that presents with myalgia, fatigue, and pigmenturia, and may also present as acute renal failure. Precipitating factors for rhabdomyolysis include autoimmune myopathies, septicemia, electrolyte abnormalities, substance abuse, alcohol use, or infection. A viral infection, especially an infection with the influenza virus, can lead to rhabdomyolysis. Through this case report, the occurrence of rhabdomyolysis with acute real weakness in a patient with a severe form of COVID19 will be evaluated.

Case presentation

A 76-year-old patient with confirmed Sars CoV-2 virus infection, unvaccinated for COVID19. With an anamnesis of the disease ten days before admission, weakness, musculoskeletal pain, decreased appetite, elevated body temperature (highest up to 38.5C), two days before admission with breathing difficulties. A patient with many accompanying chronic diseases such as DM2, HTA, BPH, CMP chr. On the day of admission to the clinic, the patient was febrile with T 38.4C, conscious, weak, brought to a wheelchair, with generalized muscle pain, tachycardic with a pulse of 120/min, normotensive, with borderline oxygen saturation of ambient air 92-93%, with auscultatory finding and x-ray of lungs in addition to covid pneumonia. Based on the initially performed laboratory biochemical analyses, high values of creatinine kinase were noted (CK – 26140, LDH- 879, creatinine- 214, urea- 14.8), a urinary catheter was immediately placed and placed in the ward. After admission, treatment was started with parenteral intravenous fluids, antimicrobial treatment with carbapenem (doses adjusted according to renal failure), antimycotic, corticosteroid therapy, anticoagulant therapy with low molecular weight heparin, diuretic, gastroprotective, probiotic, antiarrhythmic - beta brocator (prescribed by specialist cardiologist), other symptomatic, supportive substitution and regular chronic therapy. Electrolyte status was regularly monitored and adequate correction of electrolyte imbalance was performed. The glycemic profile was followed, after which, according to the indication of a nephrologist, the oral antidiabetic therapy was stopped, so that in consultation with a specialist endocrinologist, the high glycemic values were corrected with insulin therapy. During hospitalization, the patient was substituted with fresh frozen plasma and albumins on several occasions. Febrile during the first two days of hospitalization with a lytic drop in temperature and febrility until discharge. On the second day of the stay, due to a drop in oxygen saturation, the patient was placed on oxygen support via a nasal cannula on oxygen support. Due to the continuous increase of the degradation products in the initial days of the hospital stay, the motive was to consult a specialist nephrologist, regardless of the values of the degradation products, the patient had an orderly diuresis for the whole time, he was therapeutically placed on adequate parenteral hydration with intravenous fluids (the same distributed appropriately and to flow continuously, in a period of 24 hours, taking into account the heart failure), the doses of the diuretic and the antibiotic were also adjusted. Additional analysis showed a high value of Myoglobin. Due to pronounced tachyarrhythmia, during the second hospital day, the motive was to consult a specialist cardiologist, an electrocardiography with echocardiography was also performed, with findings in addition to AF as well as CMP chr, after which the patient was given an antiarrhythmic drug. A significant improvement in heart rate followed. In the remaining course of the hospitalization, there was a gradual improvement in the respiratory function, with a gradual reduction in the need for oxygen support and regression of the pulmonary auscultatory findings. The clinical improvement correlated with the improvement in the laboratory biochemical analyses, a significant drop in creatinine kinase was noted, a decrease in the values of the degradation products, with the maintenance of an orderly diuresis all the time, after which the patient with the exhaustive conservative treatment and regular consultations and monitoring of kidney function and from old age to spec. The nephrologist did not find the need for hemodialysis treatment. In the last week of the stay, the patient was no longer in need of oxygen support, with an improvement in lung auscultatory findings, regular diuresis and regular vital parameters. The patient ends his treatment at the clinic after an 18-day stay there, as cured and clinically cured, with instructions for further regular check-ups with a specialist nephrologist and cardiologist.

Analyses and diagnostic investigations



	18.07.22	20.07.22	21.07.22	22.07.22	23.07.22	25.07.22	01.08.22	03.08.22
pH	7.387	7,36	7.358	7,387	7.43	7,451		
pCO2	28.6	31,5	24.2	29,0	33.7	23,8		
PO2	55.3	65,0	81.0	73,7	53.5	63,5		
HCO3	17.4	18,4	13.7	17,6	22.7	16,7		
BE	-5.8	-5,5	-9.6	-5,7	-0.6	-5,5		
BEecf	-7.8	-7,3	-11.9	-7,6	-1.8	-7,4		
O2sat	88.6	92,0	95.7	94,7	88	93,5		
Na+	136	140	142	144	145	152		
K+	5.5	4,5	4.7	3,59	3.7	4,0		
Ca++	1.14	1,15	1.16	1,17	1.15	1,11		
GLU	8.7	10,1	20.5	12,0	14.0	18,6		
LAC	1.1	1,1	1.7	1,0	2.5	1,5		
HCT	36	30	31	31	25	25		
Cl -	107	111	116	116	119	122		
Troponin I	43.4 pg/ml	37.2 pg/ml						
CRP	175	156	124	60	46	37	49	22
Calcium	2.22	2,01	2.03	2.19	1.99	1.93		2.04
Potassium	5.3	3,8	3.7	4.1	3.0	3.6		3.7
Sodium	137	140	143	147	150	156		139
Globulins		28	29	28	25	25		
Albumins		39	38	37	35	34		
Total proteins		67	67	65	60	59		
Lipase		47						
(GGT)		10						
α-amylase		155						

(AP)		71						
(LDH)	879	438	365	406	415	514		320
CK-MB	226	111	69	48	31	31		17
(CK)	26140	10946	5878	2676	1065	491		101
(ALT)	57	63	64	60	52	51	30	30
(AST)	323	198	136	108	76	67	31	33
Glucose	8.8	6,5	18.7	9.4	15.7	15.5	9.8	5.1
Creatinine	214	338	401	439	428	376	167	135
Urea in Serum	14.8	21,6	25.4	25.4	26.1	30.2	13.1	9.3
Hb	141	117	121	118	106	102	94	100
Erit	4650	3890	3990	3910	3540	3350	3080	3360
Leuk	10.6	9,0	8.4	10.0	8.6	11.2	10.9	10.1
Trom	156	132	136	150	165	223	217	192
Hem	0.41	0,34	0.34	0.34	0.31	0.29	0.29	0.30
MCV	90.1	88	87	88	88	86	92	90
Newt	0.70	0,78	0.87	0.90	0.89	0.84	0.82	0.82
Lymph	0.21	0,19	0.10	0.05	0.03	0.07	0.11	0.11
Mono	0.09	0,03	0.03	0.05	0.08	0.09	0.06	0.01
Viro		NLR4,1	NLR=8.7	NLR=18.0	NLR=29.6	NLR=14.2	NLR=7.4	
Procalcitonin			8,58				0.07	
Myoglobin		1208.23						163.32

Discussion

Following the pandemic with the new disease COVID-19, new knowledge about the disease is constantly being discovered. Viral myositis and rhabdomyolysis are associated with several viruses, including viruses of: Influenza A and B, Coxsackie, Epstein-Barr, Herpes simplex, Parainfluenza, Adeno, Echo, Cytomegalo, Varicella zoster, Human immunodeficiency, Dengue viruses. Experiences in this group of viruses as a potential cause of myositis and rhabdomyolysis may also include the new Sars nCoV-2 coronavirus. The exact mechanism by which viruses cause muscle destruction has not been determined, but two possible mechanisms have been proposed: First - muscle necrosis related to the potential direct invasion of the virus into myocytes. Second - the toxic effect on myocytes caused by the host response (through the release of a range of cytokines and immune factors) Myoglobinuria is pathognomonic of rhabdomyolysis, and evidence suggests that myoglobin impairs glomerular filtration through several mechanisms, including: intrarenal vasoconstriction, ischemic tubular injury, and tubular obstruction. Treatment of rhabdomyolysis in patients with COVID-19 is delicate. On the one hand, hydration and stimulation of diuresis can limit kidney damage, but on the other hand, excessive hydration can prove detrimental to lung function and gas exchange, in patients with ARDS. It is precisely because of this that early detection, careful monitoring and rapid treatment with key, it involves early monitoring and possible prevention of progression of pulmonary findings, reduction of viral replication of the virus with specific antiviral drugs, inhibition of the cytokine storm in those patients with predictive parameters for the development of the same, and consequently a regular evaluation of the values of creatinine kinase, myoglobin, degradation products and diuresis in the patient. Current guidelines offer weak evidence-based treatment recommendations to prevent

rhabdomyolysis-induced AKI. The main recommendation of these guidelines includes early resuscitation with crystalloids.

Conclusion

The definitive etiology and pathophysiological mechanisms of rhabdomyolysis in patients with COVID-19 may not yet be clear, except for the previously proposed mechanisms of the host's response to the virus and the direct toxic effect of the virus on muscle cells, but there is no doubt that they are more in clinical practice we are evaluating rhabdomyolysis as a complication of COVID-19. Further research designed to better understand the pathophysiological mechanisms of rhabdomyolysis associated with COVID-19 would be of great clinical value, but until then regular evaluation of levels primarily of creatine kinase and myoglobin as predictive laboratory parameters should always be considered especially in patients with a severe clinical form of COVID19 in whom the risk of developing acute kidney failure is significantly higher. It is imperative that clinicians be aware of the potential for patients with COVID-19 to develop rhabdomyolysis and promptly initiate early treatment in order to minimize renal dysfunction.

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