COVID-19 and Low Uric Acid Levels

COVID-19 ve Düşük Ürik Asit Seviyeleri

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Editöre Mektup Letter to Editor

Dear Editor

Low serum uric acid predicts severely ill patients, the earliest laboratory indicator of sepsis ⁽¹⁾.

Novel coronovirus (COVID-19) infection is the one of the most fearful pandemics in recent decades and is associated with increased rates of mortality with well known risk factors including diabetes mellitus, ischemic hearth disease and cancer ⁽²⁾.

The relationship between serum uric acid (SUA) and prognosis in COVİD-19 patients is unclear. Our aim was investigate whether baseline and ongoing serum uric acid (SUA) levels was an independent predictor of disease severity in patients who were treated for COVİD-19 infection. Favipravir, a novel pseudo-purine analog, is potent and effective for treating COVİD-19 infection (3).

Thus; we also aimed to evaluate the effect of oral favipravir on SUA levels.

We describe a MD patient with Covid-19 whom had prior nephrectomy. Severe acute respiratory syndrome in coronavirus 2 (SARSCoV-2) infection was confirmed by computed tomography of toraks, reverse-transcriptase-polymerase-chain-reaction (RT-PCR) with assay.

Further diagnostic work-up also revealed lymphopenia, higher CRP levels, and elevated levels of D-dimer. Urinalysis did not reveal hemoproteinuria and cellular casts. Urine protein/creatinine was also in normal ranges. Renal ultrasound showed unilateral nephrectomy with normal seized remnant kidney. In the absence of another etiology, we concluded he had pneumonia possibly associated with COVID-19 infection, and started on hydroxychloroquine (HCQ) 400 mg per day and azitromycin 500 mg once a day. Due to higher D Dimer levels, low molecular weight heparin 4000 unit per day subcutaneously was also added to his treatment regimen. Five days later he developed dyspnea, shortness of breath, repetative cough, with fever. In this setting, his CRP level rose to 50 U/L from a baseline of 16 U/IL, and laboratory tests later showed lactic ascidosis consistent with SIRS. Thus, HCQ has been

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Table 1.

	Case 1	Case 2	Case 3
White-cell count (per mm3)	8060	5790	31900
Total leukocyte count			
Differential count (per mm3)			
Total neutrophils			
Total fleuti opinis	4440	4390	26800
	1410	840	2900
Total lymphocytes			
	2100	490	2000
Total monocytes			
Platelet count (per mm3)	213000	201000	22200
Hemoglobin (g/liter)	12,7	12,3	11,5
Albumin (g/liter)	41,9	42,5	31,7
Albumin (g/nter)	41,9	42,5	31,7
Alanine aminotransferase (U/liter)	80	22	25
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Aspartate aminotransferase (U/liter)	51	20	59
Lactate dehydrogenase (U/liter)	262	164	209
Creatinine (mg/dl)	1,18	0,75	0,82
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Creatine kinase (U/liter)	572	99	186
High-sensitivity cardiac troponin I (μg/L)	0,006	0,005	0,014
mgn sensitivity cardiae tropomin (μg/ μ)	0,000	0,003	0,014
Prothrombin time (sec)	10,6	10,4	13
(411)	1,1	-,	
Activated partial-thromboplastin time (sec)	27,5	24	30,0
Fibrinogen (mg/dL)	506.0	282,7	717,73
D-dimer (ng/ml)	552	153	1691
Serum ferritin (µg/liter)	1006.00	90.69	FFF FO
Octum territin (µg/itter)	1096,00	80,68	555,50
Procalcitonin (ng/ml)	0,08	0,05	1,40
			,
C-reactiveprotein (mg/liter)	84,27	3,24	238,64
Uric acid(mg/dl) day one	5,23	4,18	4,30

continued for 5 days. At the sixth day of ongoig treatment, patient was ultimately treated with favipravir and Tazobactam, which resulted in improvement in clinical symptoms. Laboratory parameters including SUA, CRP, lipid parameters became normal and lymphopenia resolved at the end of sevnth day of favipravir treatment. Trajectories of SUA levels of the patients were depicted in Table 1.

Further laboratory details are summarized in Table 2. He had no medical histrory of pulmonary diseases, hypertension, diabetes mellitus, malignancy and atrial fibrillation. Since the SUA level is affected by the renal dysfunction, we selected that cases who

had without chronic kidney disease (CKD) for searching the association between the SUA level and the disease progression. Two other patients with similar findings were seen at the specialized hospital unit for patients with Covid-19 at our hospital.

Serum uric acid, hematologic parameters ,simple biochemical tests, D dimer levels, serum ferritin and C-reactive protein (CRP) were also measured at baseline and at the end of favipravir treatment. All of our patients switched to favipravir from HCQ treatment due to lack of clinical and radiologic response. At the end of additional seventh days of the favipravir treatment all patients's SUA levels were normali-

Table 2. Case 1



Case 2



Case 3



zed in line with their rapid clinical and radiologic improvements.

In conclusion, HCQ administration significantly reduced the serum uric acid levels in patients with COVID-19 infection when compared with baseline SUA levels. No significant difference in the changes in serum creatinin and sodium, was identified in the presented cases. Intensive SUA lowering with COVID-19 infection was also correlated with disease severity. Rapid clinical detoriation was also observed among those patients with hypouricemia without any effect to renal functions. A larger study is underway to determine which patient groups might affect

most from COVID-19 infection during favipravir treatment.

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