

## INTRODUCTION

Terms:

AKI: Acute Kidney Injury, defined by increase in creatinine SIADH: Syndrome of Inappropriate Secretion of Antidiuretic Hormone CXR: Chest X-ray CMP: Complete metabolic panel, includes liver panel BMP: Basic metabolic panel, excludes liver panel BID: Twice a day NS: Normal saline IV solution

Coronavirus disease 2019 (COVID-19) increases the likelihood of organ failure in healthy individuals. COVID-19 has been shown to be associated with multiorgan failure, including acute kidney disease. The mechanism of how it causes acute kidney disease is still unknown. More patients are presenting with severe kidney injury in the setting of mild COVID-19 symptoms. In some cases if left untreated, these injuries can lead to severe complications which increase the risk of morbidity and mortality in previously otherwise healthy individuals. The aim of this study is to demonstrate a possible type of kidney injury that can occur in the setting of COVID-19.

# CASE REPORT

60 y.o. Caucasian female with history significant for migraines and HTN who presented to the ED with vomiting and diarrhea and poor oral intake for a 4 day duration prior to presentation. She carried no prior history of renal disease. Laboratory evaluation noted her to be severely hyponatremic (Na 116) in the ED, resulting in admission to the ICU. Patient tested positive for COVID-19 but chest X-ray did not demonstrate pneumonia. There were no acute abnormalities noted on CT of the head. Serum and urine osmolality was tested. The patient's Sodium was checked every 4 hours, and Intravenous Normal saline was provided in boluses as required. Later, the patient was treated with fluid restriction and Urea BID, 3% NS at 100 mL, and Tolvaptan QAM for 2 days.



CT head without acute abnormalities or anatomical changes



Chest X-ray without acute abnormalities

# A Case Report On Acute Kidney Disease In the Setting of COVID

Kethia Phelizor, D.O., Daniel Baux, D.O., Erik Polan D.O., FACOI

### **RESULTS & DISCUSSION**

With initial normal saline treatment, the patient's Sodium only improved to 119, then decreased to 116 again. Additional pertinent labs include: serum osmolality 252, urine osmolality 405, urine Na 117, uric acid 2.5, TSH 0.59, and cortisol 27.4. These study results confirmed SIADH while ruling out other common causes of hyponatremia. Patient's sodium did correct with the aforementioned therapy. Following fluid restriction, the patient's labs were: urine osmolality 691, urine Na 53, urine K 59, and a serum sodium of 116 again. After utilizing Urea twice, 3% NS infusion at 100ml/hr and Tolvaptan once, the Sodium started to improve. In the course of 48 hours, the patient's sodium increased from 116 to 131. In addition the Urine Na and osmolality were 32 and 677, respectively. On the final day of treatment, no follow up urine electrolytes were obtained. During the patient's entire stay, her creatinine maintained her baseline of 0.8.

COVID-19 can present with symptomatic hyponatremia even without other classic symptoms of the disease. Considering the patient did not have any other comorbidities or causes of SIADH, it was determined that COVID-19 infection caused it. More studies need to be conducted to determine incidence rates and pathogenesis of SIADH in patients infected with COVID-19 to help with future treatment plans and identification.

	14	- Harris	15		16		17	18		19		20		21	22	1
	10/5/2021 0643		10/5/2021 0243		10/4/2021 1853		10/4/2021 1450	10/4/2021 1403		10/4/2021 0730		10/3/2021 2148		10/3/2021 1328	10/3/2021 1123	
GENERAL CHEMISTRY																
Sodium	119	*	119	¥	116	¥		117	¥	120	+	119	¥		119	¥
Potassium	4.0		3.2	+	3.8			3.7		4.0		3.9			3.3	-
Chloride	83	-	84	+	81			82	+	84	+	84	*		82	-
C02	27.3		25.1		27.8			25.6		25.8		26.0			26.1	119
Anion Gap	9	144	10		7			9		10		9			11	
BUN	16		18		20	-		23	^	8		7			8	9)198
CREATININE	0.64	nt.	0.59		0.64			0.62		0.59		0.60			0.69	11661
eGFR (MDRD)	94.66*		103.97 *		94.66 *			98.19 *		103.97 *		101.97 *			86.79 *	
Glucose	121	+	136	-	136	-		119	1	125	1	114	1		131	-
Calcium	8.6		8.4	+	8.4	+		8.6		9.0		8.2	+		8.8	
Phosphorus			2.9	184						2.4	+					
Magnesium			1.8				2.0			1.9						
Alk Phos		1.5													76	
Albumin									1101						3.8	
Total Protein															8.2	
Uric Acid						114						2.3	*	2.5	-	
Lipase				1.18											108	
AST										ALL ALL					21	1111
ALT				1.1		221									21	
Bilirubin Direct		11				1.11			1						0.6	
Bilirubin Total															0.0	Partie I
LD						12							The second		10	11110
Lactic Acid		1398							1						7.0	

	1		2	創橋	3		4	AIR	5		6		7		a			
	10/8/2021 0458		10/7/2021 1140		10/6/2021 2159		10/6/2021 1843		10/6/2021 1417	Part of the	10/6/2021 0952		, 10/6/2021 0610		8 10/6/2021 0221		9 10/5/2021 2202	
<b>GENERAL CHEMISTRY</b>	THEFT	ARAD	ALC: NOT	AU							A DESCRIPTION		A STATISTICS OF A			SEP	ALC DA	
Sodium	131	AUT	130	ATT.	131		132		129		123		124	Constant Constant	126		121	
Potassium	4.3		4.5		4.8		4.5		4.5		42		47	×	120	*	121	-
Chloride	95	1.1	93	T.	95		94		93		88		89		91		88	
CO2	25.0	ATT	24.5		25.5		29.6		27.3		27.8		24.8		28.3		27.6	*
B/ Anion Gap	11		13	-	11		8	AT I	9	樹	7		10		7	133	5	
BUN	40	-	35	+	31	*	34	+	26	•	25	-	26	*	30	*	31 *	
CREATININE	0.69	ABILITY	0.74	MIT .	0.78		0.85		0.76	相關	0.82	織物	0.69		0.76		0.74	
eGFR (MDRD)	86.79 *	anav	80.05 *	山間	75.34 *		68.22 *		77.63 *	aw	71.11 *		86.79 *	ATT	77.63 *		80.05 *	1995
Glucose	126	+	110	-	134	-	136	-	153	-	174	•	107	-	106	AV	112	-
Calcium	9.0		9.2		9.4		9.7		9.6		8.6		8.5	ANS I	8.7	MA	8.5	SSI
Phosphorus	4.3		4.1	(SH)						1115		ATT	3.1			NT IV	ALLOW STREET	
Magnesium	2.2		2.4					All				119	2.3	AURY	VIALAN SUMMER	AN N	18 COLORIS	
Alk Phos	75		78									AR	70		A STATISTICAL AND A STATISTICA	ANY	AN IN THE R	
Albumin	3.9		3.9					Alle		ARE			3.5			(SBY	AS UN STREET	
Total Protein	7.5		7.7	115	Sela Sela			AND					7.3	ALS	S. D. BERNAN	A ST	ALL	120
UricAcid		120				110							2.9				ANTI SUSID	
Lipase								1313									63000000000000	
AST	24	ABU	20							ALL ST			17			200	ENGLANDING ST	
ALT	32	MIT	25									and a	19					
Bilirubin Direct	0.1		0.1										0.7					
Bilirubin Total	0.4		0.4										170					
LD	199		187										119					
LacticAcid		ANI																

Kidney disease and injury can take the forms of acute kidney injury (AKI), hematuria, or proteinuria in patients with COVID-19, depending on the specific pathophysiology. Some patients, especially African Americans, present with focal segmental glomerulosclerosis. Up to half of the patient's diagnosed with COVID-19 in the hospital also present with AKI. Nephrological complications are associated with increased mortality in patients with COVID-19. Patient's with COVID-19 also have a higher incidence of AKI compared to patients with similar comorbidities. In this particular case, the patient presents with SIADH or Syndrome of Inappropriate Secretion of Antidiuretic Hormone. SIADH features euvolemic hyponatremia, hypo-urea, high urine osmolality, and low serum osmolality and otherwise normal renal function. There have been dozens of case studies reporting SIADH and severe hyponatremia as the initial and isolated symptom of COVID-19. Usually, SIADH is caused by central nervous system disorders, pneumonia, endocrine diseases, paraneoplastic syndromes, and various drugs but our patient had none of these. Previous studies showed that COVID-19 may be associated with SIADH in the presence of fever and lung disease but sometimes hyponatremia could be the only defining symptom.

Moledina DG, Simonov M, Yamamoto Y, et al. The Association of COVID-19 With Acute Kidney Injury Independent of Severity of Illness: A Multicenter Cohort Study. Am J Kidney Dis 2021; 77:490.

Habib, Mhd Baraa et al. "Acute symptomatic hyponatremia in setting of SIADH as an isolated presentation of COVID-19." *IDCases* vol. 21 e00859. 1 Jun. 2020, doi:10.1016/j.idcr.2020.e00859

Yousaf Z., Al-Shokri S.D., Al-Soub H., Mohamed M.F.H. Covid-19 associated SIADH; a clue in the times of pandemic! Am J Physiol Endocrinol Metab. 2020 doi: 10.1152/ajpendo.00178.2020.

Transitional Year Residency, Philadelphia College of Osteopathic Medicine, Philadelphia, PA
Department of Internal Medicine, Philadelphia College of Osteopathic Medicine, Philadelphia, PA
Division of Internal Medicine, Chestnut Hill Hospital, Philadelphia, PA

### CONCLUSION

# REFERENCES

### ACKNOWLEDGEMENTS