

Hypercalcemia with uric acid crystals

From other results it is also evident that:

HOSP #		WARD	Nephritic clinic
CONSULTANT	Dr. Heleen Vreede	DOB/AGE	49 y Female

Episode No	SA02784405	MRN	MRN78959694	Lab	Groote Schuur Laboratory		
Mrs Linda MEYER				F	49 y	24/06/1969	
Clotted blood; EDTA blood;							Urgent
F.N.	57495756	Alt		RN		Collection	20/02/2019 14:45
Hos	Groote Schuur Hospital wc GSH			021 404 9111		Received	20/02/2019 16:07
Wrld	Endocrine Clinic F58			404 5326		Registered	20/02/2019 17:44
Doc	[0DR] Doctor In Charge .					ePR	I Deta

Uric acid nephropathy with hypercalcaemia (Mrs. Linda Meyer)
MRN78959694

Abnormal Result

The calcium on 20/02/2019 on bloods taken 14h45 was 3.29 (2.15-2.50 mmol/L).

Presenting Complaint

The patient presented with pain "from loin to groin" which is the typical presentation of passing a renal stone.

History

The patient has chronic renal failure (first creatinine was 362 umol/L with eGFR of 12ml/min – MDRD) on 12 December 2017. Creatinines relatively unchanged since then.

Upon re-evaluation of the case in 2020 it was seen that the baseline creatinine has risen to ~445 umol/L indicating a worsening of the chronic renal failure eGFR now 9 ml/min – by both CKD-EPI and MDRD formulas.

Examination

N/A

Laboratory Investigations

The patient is known with Hyperuricemia, first result 0.50 (0.16-0.36mmol/L) on 16 February 2018. The response to treatment appears poor due to continuing rising serum uric acid levels (considering whether the patient is on allopurinol).

2. Regarding the hypercalcemia:

Episode	SA04315821	SA03552076	SA03535628	SA02816641	SA02784405	SA02622825	SA02369770	SA02123812	SA01901592
Date	11/11/2020	11/12/2019	04/12/2019	04/03/2019	20/02/2019	12/12/2018	04/09/2018	23/05/2018	16/02/2018
Time	09:44	10:22	17:03	15:48	17:44	17:11	10:31	16:25	15:28
Na			135 L		139	138	139.000	138.000	137.000
K	5,3 H	4,7	4,8		4,8	4,5	4.320	4.400	4.780
Urea			17,2 H		14,3 H	16,2 H	11,3 H	18,8 H	17,1 H
Creat	443 H	484 H	434 H	444 H	446 H	475 H	334 H	408 H	415 H
MDRD	9	8	9	9	9	8	13	10	10
CKD-EPI	9								
Ca	2,79 H		2,59 H	3,09 H	3,29 H	2,97 H	2.820 H	2.850 H	3,12 H
Mg			0.94		1,05	1.00		1.060 H	.980
Phos			1,02		1,25	1,33	.980	1.240	1.110
PTH			13,3 H		4,3	4,6			

Cumulative history of UEC and CMP with PTH.

From above results a consistent hypercalcemia with a single raised PTH result can be seen – see “Final Diagnosis” and “Take Home Message” below.

Other Investigations

Uric acid crystals were seen on the urine microscopy reflecting uric acid nephropathy – a possible cause of the chronic renal failure, but I could not find any biopsy result or alternative explanation for the renal failure and assume it is uric acid nephropathy. The patient also appears to have been for a procedure at Urology (? Renal stone removal).

A serum protein electrophoresis with immunofixation (13/09/2018) showed no monoclonal peaks.

Final Diagnosis

Uric acid nephropathy with renal stones.

Hypercalcemia likely due to tertiary hyperparathyroidism.

Take Home Message

Uric acid nephropathy appears to be an uncommon cause of chronic kidney disease (ref. [Up-to-date](#)).

It should however be emphasized that clinicians consider the cause on a differential, as it is a manageable cause.

Hypercalcemia sometimes occur in Chronic Kidney Disease patients due to tertiary hyperparathyroidism. This is due to persistent hyperphosphatemia with resulting hyperparathyroidism leading to hypercalcemia (as opposed to the more commonly occurring **hypocalcemia** is renal failure).

—Commentary by Nephrologist- Dr. Erika Jones—

WRT the Uric Acid

Difficult to say if it is cause or effect of CKD. We can only really make a diagnosis of uric acid nephropathy on kidney biopsy. But it is definitely a cause that we see on occasion.

The good news is that the creatinine has remained fairly stable in the last couple of years, unlike the UA, but as kidney function deteriorates it is expected the UA will increase.

According to our buff records she had staghorn calculi and that was labelled as the cause of her CKD.

Allopurinol in CKD is challenging as it accumulates with side effects. We have had two patients with full on Steven's Johnson Syndrome. So if she isn't symptomatic I wouldn't give it to her. She is recorded as having Sarcoidosis which explains the hypercalcaemia. I think this stage is too early to have tertiary hyperparathyroidism.

A case of hyperuricemia in the ICU

HOSP #		WARD	Surgical ICU
CONSULTANT	Heleen Vreede / George van der Watt	DOB/AGE	30 year Male

Abnormal Result

The result upon the query being raised by the reviewer was a uric acid of 0.95 mmol/L (0.21-0.43 mmol/L). Three days prior to this result, the patient had a uric acid serum concentration of 0.38 mmol/L.

Presenting Complaint

The patient presented to the hospital with a history of a swollen tonsil unilaterally. This worsened over few days to a severe infection (sepsis) as described below.

History

No significant history. Patient reported sober habits.

Examination

At initial presentation, the patient appeared to have a suppurative tonsillitis. The tonsillitis later developed into a retropharyngeal abscess and soon extended into the thorax, forming a pericardial abscess, which is what was found clinically at the time of admission to Groote Schuur Hospital.

Laboratory Investigations

Date: newest to oldest (only chemistry results included)

Test Set	Test Item	12/01/2019 04:53	11/01/2019 04:18	10/01/2019 04:40	09/01/2019 08:21	09/01/2019 06:57	08/01/2019 05:15	07/01/2019 04:18	06/01/2019 06:11	05/01/2019 15:45
Test Set	Test Item									
NA	Na				δ+ 145				δ+ 140	131 L
K	K				4,6				4,1	INVH
CL	Cl				δ+ 106				98	
UREA	Urea	25,7 H	23,5 H	31,3 H	δ+ 33,3 H	31,6 H	18,4 H	20,7 H	25,1 H	22.0 H
CRT	Creat	172 H	δ- 166 H	307 H	δ+ 396 H	324 H	87	δ- 110 H	198 H	194 H
CRT	MDRD	41	42	21	16	20	>60	>60	35	35
CA	Ca				δ- 1,72 L			δ+ 2,18	1,95 L	
MG	Mg	δ- 0.79	δ- 0.96	1,26 H	1,28 H	1,19 H		δ+ 1,26 H	0.80	
PO4	Phos	δ- 0.69 L	1,83 H	δ+ 1,67 H	0.83	0.70 L		δ- 1,07	2,79 H	
UA	Jric acid				δ+ 0.95 H				0.38	
TP	otal prot		CEGK		CEGK				61	
ALB	Alb		δ+ 16 L		δ- 14 L				26 L	
TBIL	otal bili		22 H		29 H				32 H	
CBIL	onj bili		22 H		29 H				30 H	
ALT	ALT		48 H		49 H				81 H	
AST	AST		130 H		183 H				223 H	
ALP	ALP		δ+ 129 H		73				68	
GGT	GGT		103 H		136 H				117 H	
LD	LD				415 H				390 H	

Other Investigations

CT scan: images to follow

Final Diagnosis

Retropharyngeal abscess progressing to a thoracic abscess and causing overt signs and symptoms of heart failure.

Patient required a thoracotomy and pericardial drainage of the

abscess.

Take Home Messages

- Do not take tonsillitis lightly. If not adequately managed, it may cause serious complications.
- Elevated Uric acid is a risk factor for acute kidney injury. This may be by means of acute gouty crystal deposition, but other crystal-independent roles has also been described.
- Uric acid concentration will rise significantly in severe infection, most likely due to the fast tempo of tissue or DNA turnover, both by bacteria and host tissue breakdown and repair. Uric acid is a product of the metabolic breakdown of purine nucleotides.
- Uric acid, being a heterocyclic compound, I thought could interfere in various assays, and I thought even in the Jaffe reaction for creatinine, but it doesn't seem to be a common interferent when doing a quick literature search.
- Uric acid appears to be the major anti-oxidant in human serum constituting around 61% of total anti-oxidant activity, evidenced by [Maxwell et al.](#):

Table 2. Comparison of antioxidant status in patients with IDDM and NIDDM uncomplicated by microvascular or macrovascular disease and groups of age-matched non-diabetic control subjects

	IDDM	Control	NIDDM	Control
AOA ($\mu\text{mol L}^{-1}$)	320.2 \pm 11.3***	427.5 \pm 19.2	433.8 \pm 25.4	473.9 \pm 30.2
Urate ($\mu\text{mol L}^{-1}$)	209.4 \pm 10.4***	297.1 \pm 16.7	299.5 \pm 19.4	324.8 \pm 21.4
Vitamin C ($\mu\text{mol L}^{-1}$)	63.6 \pm 6.0**	87.5 \pm 4.9	38.6 \pm 5.7*	58.5 \pm 5.3
Vitamin E Absolute ($\mu\text{mol L}^{-1}$)	25.2 \pm 1.4	27.5 \pm 1.4	32.0 \pm 1.8	33.3 \pm 3.3
Corrected	5.21 \pm 0.25	5.79 \pm 0.16	5.29 \pm 0.23	5.44 \pm 0.37
Vitamin A ($\mu\text{mol L}^{-1}$)	1.30 \pm 0.05***	1.94 \pm 0.10	2.23 \pm 0.14	2.23 \pm 0.18
Thiols ($\mu\text{mol L}^{-1}$)	458.9 \pm 8.0**	499.6 \pm 8.7	457.9 \pm 7.4*	424.0 \pm 15.2
Bilirubin ($\mu\text{mol L}^{-1}$)	8.7 \pm 0.9	9.7 \pm 0.7	7.5 \pm 0.6	7.4 \pm 0.5

All values are means \pm SE. Antioxidant activity (AOA) is measured in $\mu\text{mol L}^{-1}$ trolox equivalents. Corrected vitamin E values are absolute concentration ($\mu\text{mol L}^{-1}$) divided by cholesterol (mmol L^{-1}). Significant differences between the diabetic patients and their respective control groups are indicated by * $P < 0.05$, ** $P < 0.01$ and *** $P < 0.001$.

- Relative contribution to total serum anti-oxidant

activity in this study was: urate 65.1%, vitamin C 8.7%, vitamin E 10.6%, vitamin A 5.7%, thiols 7.8% (as in albumin) and bilirubin 1.9%.

- One immediately thinks that a patient with such a rapidly progressing infection has to be immunocompromised, the most common cause(s) in South Africa being HIV or diabetes mellitus. This patient however was HIV negative, according to HIV ELISA and did not have reported signs and symptoms of diabetes.