23. Urine sodium

Ward	Not stated	D.O.B/Age	10/06/1958
Consultant			

Urine sodium 224 mmol/L

Request form: hyponatraemia

Many patients present due to manifestations of other medical comorbidities, with hyponatremia being recognized only secondarily. Many medical illnesses, such as chronic heart failure, liver failure, renal failure, or pneumonia, may be associated with hyponatremia. Patients usually present with symptoms related to their primary illness.

Symptoms of hyponatremia range from nausea and malaise, with a mild reduction in the serum sodium, to lethargy, decreased level of consciousness, headache, and (if severe) seizures and coma. Overt neurologic symptoms most often are due to very low serum sodium levels (usually < 115 mmol/L), resulting in intracerebral osmotic fluid shifts and brain oedema.

Examination should include orthostatic vital signs and an accurate assessment of volume status. Volume status forms an integral part of assessment as it often guides assessment and treatment.

A full assessment for medical comorbidities is also essential, with particular attention to cardiopulmonary and neurologic components of the examination.

Authorised by Dr TA Gcingca on 05/01/2020 at 21:37

Urine sodium	224	mmol/L	

Authorised by Dr TA Gcingca on 05/01/2020 at 21:37

Urine osmolality	649	mmol/kg	50 - 1200

CT brain and CXR may be indicated if SIADH suspected.

True hyponatraemia.

Hyponatremia can be classified according to volume status, as follows:

- Hypovolemic hyponatremia: decrease in total body water with greater decrease in total body sodium
- Euvolemic hyponatremia: normal body sodium with increase in total body water
- Hypervolemic hyponatremia: increase in total body sodium with greater increase in total body water

Hyponatremia can be further subclassified according to effective osmolality, as follows:

- Hypotonic hyponatremia
- Isotonic hyponatremia
- Hypertonic hyponatremia

There are three essential laboratory tests in the evaluation of patients with hyponatremia that, together with the history and the physical examination, help to establish the primary underlying etiologic mechanism: urine osmolality, serum osmolality, and urinary sodium concentration.

1. Urine osmolality: essential to differentiate a deficiency in excreting free water vs primary polydipsia. Urine osmolality greater than 100 mOsm/kg

indicates impaired ability of the kidneys to dilute the urine.

2. Serum osmolality: differentiates between true hyponatremia and pseudohyponatremia. True hyponatraemia causes an decrease in serum osmolality.
3. Urinary sodium: helps to differentiate between hyponatremia secondary to hypovolemia and the syndrome of inappropriate ADH secretion (SIADH). With SIADH (and salt-wasting syndrome), the urine sodium is greater than 20-40 mmol/L. With hypovolemia, the urine sodium typically measures less than 25 mmol/L.

Ancillary testing may also help with differentiating SIADH from salt-wasting. Serum uric acid levels can be important supportive information (they are typically reduced in SIADH and also reduced in salt wasting). After correction of hyponatremia, the hypouricemia corrects in SIADH but remains with a salt-wasting process.

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7. EDTA contamination vs renal impairment

Ward	Surgical ICU	**D.O.B/Age**	17/04/1994
Consultant			

Potassium: 6.1 H mmol/L [3.5 – 5.1]

No diagnosis on request form, unable to get hold of clinician.

Authorised by Dr TA Gcingca on 27/11/2019 at 08:37

Sodium	137	mmol/L	136 - 145

Authorised by Dr TA Gcingca on 27/11/2019 at 08:37

Potassium	6.1 H	mmol/L	3.5 - 5.1

Authorised by Instrument on 27/11/2019 at 06:11

Chloride	106	mmol/L	98 - 107

Authorised by Dr TA Gcingca on 27/11/2019 at 08:37

Urea	19.7 H	mmol/L	2.1 - 7.1

Authorised by Instrument on 27/11/2019 at 06:11

Creatinine	198 H	umol/L	64 - 104
eGFR (MDRD formula)	38	mL/min/1.73 m²	

MDRD-derived estimation of GFR may significantly underestimate true GFR in patients with GFR > 60 mL/min/1.73m^2. It may also be unreliable in the case of: age <18 years or >70 years; pregnancy; serious co-morbid conditions; acute renal failure; extremes of body habitus/unusual diet; gross oedema. The MDRD-eGFR used here does not employ an ethnic factor for race.

Authorised by Dr TA Gcingca on 27/11/2019 at 08:37

Calcium	1.17 L	mmol/L	2.15 - 2.50

Authorised by Dr TA Gcingca on 27/11/2019 at 08:37

Magnesium	0.97	mmol/L	0.63 - 1.05

Authorised by Instrument on 27/11/2019 at 06:11

Inorganic phosphate	1.46 H	mmol/L	0.78 - 1.42

Authorised by Instrument on 27/11/2019 at 06:11

Indices in serum:

Haemoglobin index	Not detected
Bilirubin index	Trace
Lipaemia index	Not detected

Authorised by Instrument on 27/11/2019 at 05:44

White Cell Count	10.17	x 10^9/L	3.92 - 10.40
Red Cell Count	3.32 L	x 10^{12}/L	4.50 - 5.50
Haemoglobin	9.8 L	g/dL	13.0 - 17.0
Haematocrit	0.274 L	L/L	0.400 - 0.500
MCV	82.5 L	fL	83.1 - 101.6
MCH	29.5	pg	27.8 - 34.8
MCHC	35.8 H	g/dL	33.0 - 35.0
Red Cell Distribution Width	15.2	%	12.1 - 16.3
Platelet Count	116 L	x 10^9/L	171 - 388

Potassium ethylenediaminetetraacetic acid (EDTA) is a sample tube anticoagulant used for many laboratory analyses. Gross potassium EDTA contamination of blood samples is easily recognised by marked hyperkalaemia and hypocalcaemia. Subtle contamination is a relatively common, often unrecognised erroneous cause of spurious hyperkalaemia. In the case illustrated, it would be difficult to confidently exclude EDTA contamination based on these results alone. There is renal impairment which may explain the hyperkalaemia. The increased phosphate coupled with the renal impairment would also be an argument for the hypocalcaemia present.

In this instance, comparison with previous results was useful. The results are most likely due to renal impairment. As the patient had been admitted to the ward for a week, it was useful to be able to compare previous results. The gradual decline in renal function helped to explain the biochemical findings. As the samples were drawn of different days by different persons, the likelihood of EDTA contamination on all the days is relatively slim.

However, it is important to be cognisant that mild EDTA contamination may cause subtle shifts in results that may have negative consequences for the patient if erroneously acted on.

24. CoA trapping

Ward	Paeditric ICU	**D.O.B/Age**	11/03/2020
Consultant	Prof G. vd Watt		

Elevated propionic acid in the urine organic acid profile.

Fever with LRTI. ?COVID

Normal birth with no antenatal problems

#RVD exposed

Now:

#FTT

#LRTI. ?COVID

The patient presented with fever and LRTI which resolved after 3 -4 days of antibiotics. The patient then developed seizures with apnoeic attacks. The patient required intubation and ventilation and was transferred to ICU. The patient was noted to be having breakthrough seizures despite anticonvulsant therapy.

Further questioning revealed that the patient had become progressively drowsy with poor feeding.

Family history: No siblings noted to have had previous problem.

The patient was noted as not interacting with his environment.

CNS exam: Low GCS with upper motor neuron signs.

Other systems unremarkable.

pH	7.13 L		7.35 - 7.45
pCO2	2.99 L	kPa	4.66 - 6.38
pO2	19.90 H	kPa	11.04 - 14.36
Standard bicarbonate	9 L	mmol/L	22 - 26
Base excess	-21.6 L	mmol/L	-10.0 - -2.0
O2 saturation	100 H	%	94 - 98
Sodium	121 L	mmol/L	136 - 145
Potassium	4.4	mmol/L	3.5 - 4.5
Chloride	92 L	mmol/L	98 - 113
Glucose	13.3	mmol/L	
Ionised calcium	0.80	mmol/L	
Carboxyhaemoglobin	3.5	%	
Methaemoglobin	-1.7	%	

Authorised by NL Makhalima on 28/05/2020 at 16:42

Ammonia	1517 H	umol/L	40 - 80

Please note that preanalytical factors including a delay in sample reception and sample not transported on ice may cause raised ammonia results.

Trace lipaemia observed

Please repeate

Total cholesterol	1.90	mmol/L
Triglyceride	6.21	mmol/L
HDL cholesterol	0.18	mmol/L
LDL cholesterol	Triglyceride level too high [>4.5mmol/l] for LDL calculation	

CHOLESTEROL TREATMENT TARGETS (per CV Event Risk Category):

Risk Category:	TC target:	LDL-C target:
Low/Moderate Risk	<5.0 mmol/L	<3.0 mmol/L
High Risk	<4.5 mmol/L	<2.5 mmol/L

Authorised by KF Sephula on 28/05/2020 at 05:29

Calcium	1.24 L	mmol/L	2.12 - 2.64

Authorised by KF Sephula on 28/05/2020 at 05:29

Albumin	23 L	g/L	26 - 41

Authorised by NL Makhalima on 29/05/2020 at 14:07

Total bilirubin	5	umol/L	5 - 21

Authorised by NL Makhalima on 29/05/2020 at 14:07

Conjugated bilirubin (DBil)	2	umol/L	0 - 5

Authorised by NL Makhalima on 29/05/2020 at 17:52

Alanine transaminase (ALT)	106 H	U/L	1 - 25

Authorised by NL Makhalima on 29/05/2020 at 14:08

Aspartate transaminase (AST)	391 H	U/L	0 - 51

Authorised by NL Makhalima on 29/05/2020 at 14:08

Alkaline phosphatase (ALP)	382 H	U/L	75 - 316

Authorised by NL Makhalima on 29/05/2020 at 14:07

Gamma-glutamyl transferase (GGT)	44	U/L	12 - 122

Authorised by B Gool on 26/05/2020 at 16:35

CSF glucose	1.5	mmol/L	

CSF glucose reference range:

CSF glucose is normally 60 - 80% of plasma glucose, in samples taken within 15 minutes of each other.

Authorised by B Gool on 26/05/2020 at 16:35

CSF protein	1.62 H	g/L	0.20 - 0.80

Authorised by NL Makhalima on 26/05/2020 at 17:50

CSF adenosine deaminase	0.0	U/L

CSF ADA activity of > 6 U/L is suggestive of TB. However, other conditions such as bacterial or Cryptococcal meningitis may also produce elevated ADA levels.

CSF Analysis:

Appearance:

Clarity	Bloodstained	
Clots	Absent	

Cell Count:

Polymorphs	0	/uL
Lymphocytes	0	/uL
Erythrocytes	48	/uL

Authorised by NT Jikwana on 26/05/2020 at 14:53

Gram Stain:

Organisms	No bacteria observed

Authorised by MG Mpotje on 28/05/2020 at 09:07

Bacterial Culture:

No growth after 2 days

Authorised by NL Makhalima on 28/05/2020 at 16:45

White Cell Count	0.59 L	x 10^9/L	5.00 - 20.00
Red Cell Count	2.54 L	x 10^{12}/L	3.90 - 5.90
Haemoglobin	8.1 L	g/dL	12.0 - 21.8
Haematocrit	0.218 L	L/L	0.340 - 0.620
MCV	85.7 L	fL	88.0 - 126.0
MCH	31.7	pg	31.0 - 37.0
MCHC	37.0 H	g/dL	30.0 - 36.6
Red Cell Distribution Width	14.8	%	
Platelet Count	67 L	x 10^9/L	140 - 350
MPV	9.6	fL	7.0 - 11.4

Comment: Automated platelet count to be reviewed microscopically.

MCHC results may be affected by lipaemia

repeated tplateet = 71

FBC comment:

No clot detected in EDTA sample

Peripheral smear to be reviewed

CT brain may be useful in assess for organic neurological

cause.

Propionic acidaemia.

DDx: Biotinidase deficiency

Propionic acidaemia is an organic acidaemia characterized by deficiency of propionyl-CoA carboxylase. Propionyl-CoA carboxyalse converts propionyl-CoA to methylmalonyl-CoA. It is inherited in an autosomal recessive pattern. The metabolism of isoleucine, valine, threonine, and methionine produces propionyl-CoA. To a lesser degree, cholesterol and odd-chain fatty acids also contribute to propionyl-CoA levels. Affected individuals must follow a low-protein diet and early diagnosis improved prognosis.

The accumulation of propionyl-CoA results in significant mitochondrial CoA trapping and inhibited fatty acid oxidation. The enhanced anapleurosis of propionate and CoA trapping alters the pool sizes of tricarboxylic acid cycle (TCA) metabolites. This explains the marked hyperammonaemia that patients present with as well as potential hypoglycaemia

A high index of suspicion is required to diagnose inborn errors of metabolism (IEM). This case highlighted the importance of understanding key points in metabolic pathways. It also emphasized the correlation between catabolic stress being an initiating event in IEMs.