Humber Teaching
NHS Foundation Trust

MANAGEMENT OF ELECTROLYTES IN ADULTS GUIDELINE

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VALIDITY – Guidelines should be accessed via the Trust intranet to ensure the current version is used.

CHANGE RECORD

Version	Date	Change details
1.0	*Aug-20*	*New Guideline*
2.0	*March-22*	*Reintrodution of hyperkaleamia guidance within the Electrolyte guidelines* *Taken through Clinical network Jan-22* *Approved DTG 31-Mar-22*
2.1	*July-22*	*Inclusion of sodium zirconium in the guideline for hyperkalaemia (for information only)*

Contents

1. INTRODUCTION

This guideline has been written in order to assist in the management of deranged electrolytes for adults in both hospital and community settings at Humber Teaching NHS Foundation Trust (HTFT). It does not replace clinical judgement. It also provides guidance as to when a senior review or an admission to the local Acute Trust may be required.

Prior to prescribing IV fluids or oral supplementation based on this guideline, please read the additional details / considerations listed for each state of electrolyte imbalance.

This guideline provides advice on the management of the following electrolyte disturbances:

- Hypokalaemia (low potassium)
- Hyperkalaemia (high potassium)
- Hyponatraemia (low sodium)
- Hypernatremia (high sodium)
- Hypomagnesaemia (low magnesium)
- Hypermagnesaemia (high magnesium)
- Hypophosphataemia (low phosphate)
- Hypocalcaemia (low calcium)
- Hypercalcaemia (high calcium)

These guidelines are **not** intended to provide comprehensive background knowledge for these conditions. They have been designed as a guide for clinical staff working in the Humber Teaching NHS Foundation Trust. The guideline focuses on assessment, monitoring and treatment which can be undertaken at the Trust (as we are not the Acute Trust). For greater detail on the management of these conditions please see a reputable source of information – such as: **NICE Evidence search**: https://www.evidence.nhs.uk/
or
NICE Clinical Knowledge Summaries: https://cks.nice.org.uk/

2. SCOPE

This guideline is aimed at doctors, pharmacists and nurses who may be involved in the assessment, prescribing, monitoring and administration of medications to correct electrolyte disturbances that do not require admission to the local Acute Trust.

3. PROCEDURES

3.1. Hypokalaemia (serum potassium <3.5mmol/L)[1][2]

Signs and Symptoms
Mild hypokalaemia may present with no symptoms at all.

Arrhythmias ● generalised weakness ● muscle cramps ● numbness/tingling ● constipation ● urinary retention may be present in more moderate hypokalaemia.

Severe hypokalaemia presents with muscle weakness ● muscle necrosis ● paralysis ● impaired respiratory function. This is a **medical emergency**.

Assessment / Monitoring
The most common causes of hypokalaemia include potassium depletion via renal losses (e.g. metabolic acidosis) or intestinal losses (e.g. diarrhoea).

- ECG monitoring is essential
- Confirm by urgent blood gas (if this facility is available)
- Serum magnesium (untreated hypomagnesaemia can cause refractory hypokalaemia)
- Serum bicarbonate
- Serum phosphate
- Renal function (see additional information, lower doses of potassium required in CKD)
- Liver function
- Digoxin toxicity if applicable (hypokalaemia can predispose a patient to digitalis toxicity)
- Arterial blood gases, if an acid-base cause is suspected, especially if the bicarbonate is abnormal.

General Management
Replace potassium losses (see Drug Therapy and Treatment below).

Identify and treat any underlying causes where possible, review:
Loop/thiazide diuretics ● Insulin or salbutamol treatment (e.g. long term nebulisation) ● Laxative abuse ● Glucocorticoids ● Vomiting and diarrhoea ● Cell damage - e.g. post-surgery or ischaemia (Acute Tubular Necrosis) ● Re-feeding syndrome ● Hypomagnesaemia (see below) ● Chronic alcoholism ● Mineralocorticoids excess syndromes e.g. Cushing's disease, Conn's disease.

Drug Therapy / Treatment
Oral potassium chloride can be utilised in mild to moderate hypokalaemia.

- Sando-K effervescent tablets contain 12mmol of potassium and 8mmol of chloride per tablet.
- Kay-Cee-L syrup contains 1mmol or potassium and 1mmol of chloride per ml. This should be used if patients cannot tolerate Sando K effervescent tablets.

Mild hypokalaemia: Serum K+ 3.0-3.5mmol/L (Approximate potassium deficit = 200mmol)
Sando-K - 2 tablets 3 times daily

- Monitor serum levels and review need for ongoing treatment after 3 days
- Monitor serum K+ twice weekly until stable
- Once serum K+ stable or greater than 4.5mmol/L reassess need for further supplementation

Moderate hypokalaemia: Serum K+ 2.5-2.9mmol/L (Approximate potassium deficit = 200 - 400mmol)
Sando-K - 3 tablets 3 times daily

- Monitor serum K+ daily until greater than 2.9mmol/L then manage as above.

Severe hypokalaemia: Serum K+ less than 2.5mmol/L or symptomatic hypokalaemia
IV potassium replacement with potassium chloride (KCl) usually required, refer to the local Acute Trust if unable to administer IV fluids.

If able to administer IV fluids:

- Approximate potassium deficit > 400mmol
- Assess the need to admit the patient for emergency treatment at the local Acute trust
- Follow NICE CG174: Intravenous fluid therapy in adults in hospital. Available from: https://www.nice.org.uk/guidance/cg174
- Sodium chloride 0.9% is the preferred infusion fluid as glucose 5% can cause transcellular shift of potassium into cells. However glucose 5% can be used in patients with sodium / fluid overload e.g. in ascites
- IV potassium replacement should not exceed 10mmol/hour.
- Potassium (KCl) concentration should not exceed 20mmol per 500ml
- Where possible use pre-made bags should be used e.g. 20mmol KCl in 500ml sodium chloride 0.9%
- Serum potassium should be monitored daily

If there are life threatening complications of hypokalaemia e.g. Arrhythmias, muscle weakness, respiratory failure, paralysis/paralytic ileus the patient will need transfer to the local Acute Trust for emergency treatment.

Additional Information / Considerations

- The dosage and duration of treatment depends on existing potassium deficit and whether there is continuing potassium loss.
- Potassium supplements should not be given in severe renal impairment and if serum K+ greater than 5mmol/L.
- Consider dietary measures and dietician referral
- Serum magnesium should be checked and replaced in severe hypokalaemia, unexplained hypokalaemia, refractory hypokalaemia and if hypokalaemia has been induced by diuretics. If serum magnesium is low, serum potassium is difficult to replace. (see section 3.5).
- Please note that lower doses of replacement therapy may be required for patients with renal disease due to reduced potassium clearance. Caution should be exercised in patients with CKD or those taking concomitant ACE inhibitors, potassium sparing diuretics and other medications associated with an increased risk of hyperkalaemia.
- Consider lower starting doses of potassium in patients with CKD e.g. Sando K 1 tablet 3 times daily in mild hypokalaemia

3.2. Hyperkalaemia (serum K+ > 5.5mmol/L)[1][3][20]

Signs and Symptoms

ECG changes associated with hyperkalaemia: tall tented T waves ● shortened QT interval ● prolonged PR interval ● a wide QRS complex ● Small/flattened P waves ● ventricular fibrillation ● finally a sine wave.

Patients may be asymptomatic up to the point of cardiac arrest. Despite this some patients may present with muscle weakness ● paraethesia ● palpitations ● chest pain ● nausea / vomiting.

Assessment / Monitoring

Hyperkalaemia is a **common life-threatening medical emergency that is treatable**. Patients may be completely asymptomatic even up to immediately prior to cardiac arrest, therefore the importance of early recognition and prompt treatment is paramount.

- The absolute level is important as well as rate of rise especially in the context of significantly reduced or no urine output.
- The effect of hyperkalaemia (≥6.5mmol/L) on the ECG is more important than the absolute serum potassium level.
- Serum potassium level result may be spurious. If the result is unexpected and there are no ECG changes, then repeat U&Es urgently and consider an urgent venous or arterial blood gas (if inpatient).
- **Do not delay treatment if there are associated ECG changes (if inpatient)**.
- **Review vital signs and medical history. Urgency of treatment will depend on patient's individual circumstances and medical history**
- Serum potassium
- Confirm by urgent blood gas (if this facility is available)
- ECG
- Renal function
- Serum bicarbonate (to assess for metabolic acidosis)
- Blood glucose

Serum digoxin (if on digoxin, to assess for digoxin toxicity.)

General Management

Exclude spurious hyperkalaemia (i.e. delay in analysis of sample (>6 hrs), sample contamination with EDTA, pseudohyperkalaemia secondary to haemolysis, thrombocytosis and leucocytosis. Check full blood count and corrected calcium. Consider magnesium and PTH (If available).

Check for ECG changes of hyperkalaemia. (ECG changes associated with hyperkalaemia is stated above and below in the 'Emergency Management of Hyperkalaemia in Adults' guideline produced by the Renal Association. **A 12 lead ECG is recommended to assess for cardiac toxicity and risk of arrhythmias. Where 12 lead ECG is not available or practicable then the patient should be referred to secondary care for immediate assessment. Where 12 lead ECG are undertaken the health care professional is required to be skilled in interpreting the results.**

Attach all patients to cardiac monitor for continuous ECG monitoring (minimum 3 lead ECG).
The patient's vital signs should be monitored using the NEWS2 tool and escalated in line with the Physical Health and Care of the Deteriorating Patient policy.

Identify and treat underlying cause where possible:

- Consider review of potential medications including: potassium supplements ● ACE inhibitors ● Angiotensin II receptor blockers ● potassium-sparing diuretics ● NSAIDs ● aldosterone antagonists should be discontinued
- Severe acidosis (pH < 7.1 to 7.2 based on underlying disease)
- Hypoaldosteronism e.g. Addison's disease
- In resistant hyperkalaemia or renal failure urgent referral and advice should be sought from the local acute Trust (e.g. Medical Registrar/ Renal team) as haemodialysis may be needed

Drug Therapy / Treatment

Follow the Renal.org "Emergency Management of Hyperkaemia in Adults" flow chart on next page.

If severe (serum K+ greater than or equal to 6.5mmol/L) with or without ECG changes: start treatment immediately if within inpatient unit or community wards and seek senior advice urgently (or seek advice from local acute Trust as appropriate). **This is a medical emergency. The patient will require admission to the local Acute trust. Dial 999.**

If confirmed serum K+ greater than or equal to 6mmol/L and/or ECG changes: start treatment immediately and seek senior advice urgently if within inpatient unit or community wards (or seek advice from local acute Trust as appropriate). **This is a medical emergency. The patient will require admission to the local Acute trust.**

Patients within a primary care setting with severe (serum K+ ≥6.5mmol/L) hyperkalaemia, with or without ECG changes, will require emergency transfer to secondary care for immediate assessment and treatment. Dial 999.

If serum K+ is confirmed to be mild (5.5-5.9mmol/L). Consider calcium resonium 15g four times a day in water (**NOT fruit juice**). Calcium resonium is associated with constipation and so Lactulose 15ml three times a day should be prescribed concomitantly to prevent constipation.

- Calcium resonium will not lower potassium acutely (onset of action up to 4 hours)
- Serum potassium should be monitored daily until less than 5.5mmol/L

All inpatient units and community wards will employ a robust local system to ensure that all patients with hyperkalaemia are immediately identified and the results are escalated to an appropriate health care professional. Each area is required to have a local standard operating procedure which outlines how blood results are received, processed, escalated, and actioned and who is responsible.

Additional Information / Considerations

If a patient has renal failure or severe CKD consider seeking advice from the renal team in the treatment of chronic hyperkalaemia.

If the patient is on digoxin, consider the possibility of digoxin toxicity in acute renal failure. **Patients with Digoxin toxicity will need transfer to the local acute Trust.**

Calcium gluconate undiluted has a high osmolarity and may cause venous irritation and tissue damage in cases of extravasation. If a central venous access device is unavailable, administer via a large peripheral vein, monitoring insertion site closely using a recognised phlebitis scoring tool. Resite cannula at first signs of inflammation.

Blood glucose should be monitored closely if IV treatment is initiated prior to transfer to the local acute Trust.

Preventing hyperkalaemia

Most cases of hyperkalaemia in the community occur in the context of treatment for hypertension, diabetes and or heart disease (UK Renal Association, 2014).

Renal function should be assessed prior to commencing any drug therapy that may cause hyperkalaemia for example renin-angiotensin drugs (ACE-inhibitors, angiotensin II receptor blockers, aliskiren), potassium sparing diuretics, and/or loop diuretics. This should be further monitored within the primary care setting following initiation, dose adjustments or during acute illness.

Patients should be advised to withhold these drugs during periods of acute illness for example diarrhoea, vomiting or sepsis.

The UK Renal Association recommends avoiding non-steroidal anti-inflammatory drugs and trimethoprim in patients with Chronic Kidney Disease 4 and 5 particular when in combination with renin-angiotensin blockade. Caution should also be taken in the elderly.

Further General Management in cases chronic hyperkalaemia

Consider review of medications associated with hyperkalaemia (under specialist advice where appropriate) e.g. calcineurin inhibitors, nicorandil, heparin, trimethoprim.

Mild to moderate hyperkalaemia may resolve following omission or reduction of drug therapies that may cause hyperkalaemia (renin-angiotensin drugs, potassium sparing diuretics, and/or loop diuretics). This requires appropriate levels of monitoring and follow-up.

According to NICE TA599

Sodium zirconium cyclosilicate is recommended as an option for treating hyperkalaemia in adults only if used:

- in emergency care for acute life-threatening hyperkalaemia alongside standard care or
- for people with persistent hyperkalaemia and chronic kidney disease stage 3b to 5 or heart failure, if they:
    - have a confirmed serum potassium level of at least 6.0 mmol/litre and
    - because of hyperkalaemia, are not taking an optimised dosage of renin-angiotensin-aldosterone system (RAAS) inhibitor and
    - are not on dialysis

NB: This is for information only as sodium Zirconium will not be initiated primary care

Consider dietetic advice and review of nutritional supplementation (e.g. nasogastric feeds or nutritional drinks) if dietary potassium is implicated in more chronic hyperkalaemia

Dietician referral may be beneficial if dietary advice required. Generally speaking, foods high in potassium include: Coffee, hot chocolate, malted drinks (Horlicks), fruit juices/smoothies, nuts, bananas, chips, crisps, soups, marmite and finally low sodium salt/salt substitutes e.g. Lo Salt

Patients who present unwell should be referred to secondary care for assessment.

Summary table

Assessing and monitoring of hyperkalaemia in Inpatient Units or Community Wards

Severity	Potassium Level	Action	Timescale	Follow Up bloods /escalation
Mild	5.5-5.9	Clinical Assessment Vital signs monitoring Medication and diet Review	Within 24 hours	Repeat sample and then follow-up again within 1 week
Moderate	**6.0-6.4**	Clinical Assessment Vital signs monitoring Medication and diet review 12 lead ECG.	Within 4-6 hours	Repeat sample urgently If no ECG changes and patient remains well follow-up bloods in 2-3 days. If patient unwell or ECG changes urgent transfer to secondary care
Severe	**≥6.5**	Urgent admission to secondary Care. **DIAL 999**	Immediate admission	As advised by secondary care on discharge

Assessing and monitoring of hyperkalaemia in Primary Care

Severity	Potassium Level	Action	Timescale	Follow Up bloods /escalation
Mild	**5.5-5.9**	Clinical Assessment Vital signs monitoring Medication and Diet Review	Within 1-2days	Within 1week No escalation required
Moderate	**6.0-6.4**	Clinical Assessment Vital signs monitoring Medication and Diet Review 12 lead ECG	Same day. If not possible refer to secondary care	If patient is well, medication and diet have been reviewed and ECG is normal follow-up bloods 2-3 days. No escalation required. If patient is unwell or there is ECG changes refer immediately to secondary care.
Severe	**≥6.5**	Urgent admission to secondary Care. **DIAL 999**	Emergency admission	As advised by secondary care on discharge

THE RENAL ASSOCIATION
founded 1950

Resuscitation Council (UK)

Emergency Management of Hyperkalaemia in Adults

NAME:
ADDRESS:
D.O.B.:
CHI:

Hyperkalaemia ($K^+ \geq 5.5$ mmol/L)
Consider initiating treatment if Hyperkalaemia suspected and K^+ unknown

Assess Patient

Airway Breathing Circulation Disability Exposure (ABCDE) Approach
Seek expert help if airway, breathing or circulation compromised

Perform 12-lead ECG

MILD	MODERATE	SEVERE
K^+ 5.5 - 5.9 mmol/L	K^+ 6.0 - 6.4 mmol/L	$K^+ \geq 6.5$ mmol/L
Consider cause and if treatment indicated	*Treatment guided by clinical scenario, ECG and rate of rise*	*Emergency treatment indicated*

Seek expert help!

Monitor ECG in high dependency area
Sick patient; *$K^+ \geq 6.5$ mmol/L; Acute ECG changes present*

Acute ECG changes present (*tick if present*)?
- ☐ Peaked T waves
- ☐ Absent or flattened P waves
- ☐ Broad QRS
- ☐ Sine wave
- ☐ Bradycardia
- ☐ VT

NO

YES

Protect the Heart

Calcium Chloride OR Calcium Gluconate IV
Repeat ECG and consider further dose after 5 min if ECG changes persists

Insulin–Glucose IV Infusion
Give in severe hyperkalaemia
Consider in moderate hyperkalaemia (*assess ECG and rate of rise*)

Shift K^+ into cells

Salbutamol 10-20 mg Nebulised
Give in severe hyperkalaemia
Consider in moderate hyperkalaemia (*assess ECG and rate of rise*)

Remove K^+ from body

Consider Calcium Resonium
15 g x4/day oral or 30 g x2/day PR

Consider Dialysis
Seek advice from Renal or ICU team; patient transfer may be required

$K \geq 6.5$ mmol/L despite medical therapy

Monitor K^+ and Blood Glucose

Monitor serum K^+ and blood glucose

Prevention

Consider cause of hyperkalaemia, prevent further rise and recurrence
Stop all nephrotoxic medication including ace-inhibitors, angiotensin II receptor blockers, potassium-sparing diuretics, NSAIDS and assess diet

K^+: potassium; Na^+: sodium; Creat: creatinine; IV: intravenous; min: minutes; PR: per rectum; EWS: early warning score; IHD: Ischaemic Heart Disease; NSAIDS: non-steroidal anti-inflammatory drugs

Date: ___/___/___ Time: ___:___

First 15-30 min

Na^+: _____	O_2 Sat: _____%
K^+: ___.__	RR: _____
Urea: ___.__	BP: ___/___
Creat: _____	Pulse: _____
Time: ___:___	EWS: _____

Check K^+
Send lithium-heparin sample to lab
Use blood gas analyser if available
Exclude pseudo-hyperkalaemia

Dialysis patient: Contact Renal Unit
Cardiac monitoring: YES/ NO
Call for senior help: YES/ NO
Renal or ICU referral: YES/ NO

IV Calcium (6.8 mmol)
10 ml 10% Calcium Chloride IV OR
30 ml 10% Calcium Gluconate IV
Use large vein
Give over 5-10 min

Next 30-60 min

Glucose (25 g) over 15 min
50 ml 50% Glucose OR
125 ml 20% Glucose,
WITH Soluble Insulin – 10 units

Salbutamol
Give 10 mg if history of IHD
Avoid if tachyarrhythmia present

Blood Monitoring:
Baseline Glucose __.__ K^+ ___.__
15 min Glucose __.__
30 min Glucose __.__
60 min Glucose __.__ K^+ ___.__

After 1st hour

Blood Monitoring:
90 min Glucose __.__
120 min Glucose __.__ K^+ ___.__
180 min Glucose __.__
240 min Glucose __.__ K^+ ___.__
360 min Glucose __.__ K^+ ___.__
24 hours K^+ ___.__

3.3. Hyponatraemia (serum sodium <135mmol/L) [8][14][15][16][17]

Signs and Symptoms
Patient may present with a wide range of symptoms or be asymptomatic. It is dependent on the severity and rate of sodium depletion but can include:
Reduced appetite ● nausea ● malaise ● headache ● irritability ● confusion ● weakness ● ataxia.
The severity of symptoms should be assessed before deciding on treatment. Severe symptoms such as **convulsions/seizure, reduced GCS and coma are a medical emergency** and require urgent treatment and transfer to the local Acute Trust.

Assessment / Monitoring
Initial assessment is to include:

- Any recent or past medical history which may explain: GI losses (vomiting, diarrhoea), heart failure, liver failure, malignancy, endocrine causes (hypothyroidism, adrenal insufficiency)
- Historic blood results to accurately establish whether acute or chronic hyponatraemia
- Speed of onset: Acute hyponatraemia develops <48hours, chronic hyponatraemia develops >48hours
- Determine fluid status i.e. hypovolemia, euvolemia, fluid overload?
- Are there any features of cerebral oedema?
- Review of medication which are associated with hyponatraemia (see below)
- Is the hyponatraemia - mild: 130-135mmol/mol, moderate: 125-129mmol/L or severe <125mmol/L.

Further assessment (see:

- Blood glucose and lipids (to exclude pseudohyponatraemia)
- Check serum and urine osmolality to confirm if patient has true hypotonic, hypo-osmolar hyponatraemia. (This will likely only be done in the acute Trust).

General Management
Identification and correction of the underlying cause is the first step in appropriate treatment of hyponatraemia.

Following this, fluid restriction should be the first intervention for patients with hypervolaemic or euvolaemic hyponatraemia with serum sodium (130-135mmol/L). Consider aiming for a negative fluid balance of around 500ml/day, however this will depend of level of hyponatraemia and severity of symptoms.

If this fails to correct hyponatraemia, symptoms become more severe or levels fall further, refer to the local Acute Trust as an endocrinology review will be needed. Follow guidance below:

- If serum sodium concentration is between 125-129mmol/L or if cause is unclear, refer to the medical registrar or an endocrinologist for advice and the need for admission
- Admit patients to the local Acute Trust if their serum sodium is severe (<125mmol/L), they are symptomatic or if they have signs of hypovolaemia.

Medications associated with Hyponatraemia (these lists are not exhaustive)
Thiazide diuretics: bendroflumethiazide ● indapamide ● hydrochlorothiazide
Loop diuretics: Furosemide ● bumetanide ● amiloride
ACE inhibitors: ramipril ● lisinopril ● enalapril ● perindopril ● captopril
ARBs: losartan ● candesartan ● olmesartan
SSRIs: sertraline ● fluoxetine ● citalopram ● paroxetine
Tricyclic anti-depressants (TCAs): amitriptyline ● nortriptyline
Anti-psychotics: olanzapine ● clozapine ● aripiprazole ● haloperidol ● risperidone ● quetiapine
Anti-epileptics/mood stabilisers: carbamazepine ● sodium valproate ● lamotrigine ● phenytoin
Proton pump inhibitors (PPIs): omeprazole ● lansoprazole ● pantoprazole ● esomeprazole ● rabeprazole
NSAIDs/COX-2 inhibitors: ibuprofen ● naproxen ● diclofenac ● celecoxib
Chemotherapeutic agents: vincristine ● vinblastine ●carboplatin ● cisplatin ● cyclophosphamide

Additional Information / Considerations

Hyponatraemia is the most common electrolyte imbalance and is classified as either acute or chronic (see below). Symptom severity depends on the speed of onset.

- Acute hyponatraemia develops in <48 hours.
- Chronic hyponatraemia develops over >48 hours.

The type of hyponatraemia should be ascertained in order to ensure that the correct treatment is followed. If it is unclear whether it is acute or chronic hyponatraemia; it should be assumed that it is chronic hyponatraemia unless otherwise evidenced.

When reviewing medications that can cause hyponatraemia please be aware that some medications such as anti-depressants and anti-convulsants should not be stopped without seeking specialist advice. Whereas antihypertensive therapies and diuretics can generally be stopped more readily.

The provision of guidance on management of hyponatraemia in a non-acute setting is limited as a number of further assessment steps are required to ensure that the appropriate treatment pathway is followed. These include:

- Serum osmolality
- Urine osmolality
- Urine sodium
- Blood glucose and lipids
- Cortisol
- TSH

Inpatient mental health wards, community hospitals and GP surgeries (for which this guidance has been primarily produced) may not have the appropriate facilities to conduct a number of these tests/measurements and the results need to be discussed with the Endocrinology team. Serum sodium will also need to be monitored very closely during the treatment period (e.g. at least every 6 hours and potentially more regularly in the initial treatment) which may not be feasible in these settings.

Because of this, it is recommended that Endocrinology (registrar or consultant) is contacted **early** if there are concerns surrounding hyponatraemia to ensure that the subsequent management is appropriate and guided by specialist advice.

3.4. Hypernatraemia (serum sodium>145mmol/L) - [1][13][18][19]

Signs and Symptoms

There are no specific clinical features of hypernatraemia. It is usually diagnosed incidentally on serum testing.

Symptoms depend on the degree of hypernatraemia and include thirst ● irritability ● headache ● nausea and vomiting ● nystagmus ● lethargy ● weakness ● muscle tremor and rigidity ● seizures.

When serum sodium >155 mmol/L, hypernatraemia can present with confusion and coma.

Furthermore, signs of dehydration: reduced skin turgor ● oliguria ● postural hypotension.

Assessment / Monitoring

Hypernatraemia (Serum sodium >145 mmol/L) is most commonly caused by dehydration. This can be a result of reduced water intake or where water losses are greater than sodium losses (e.g. watery diarrhoea).

Other examples include inappropriate fluid replacement (too much IV saline), diabetes insipidus or osmotic diuresis (e.g. in poorly controlled diabetes).

Hypernatraemia can also be caused by an excessive intake of sodium - usually iatrogenic which may be suggested by a normal urea level.

- Monitor BCP/U+Es
- Assess Renal function
- Blood glucose (to assess for hyperglycaemia)
- Assess for hypercalcaemia
- Assess hydration status and if patient is also hypovolaemic, then monitor urinary output and renal function

Identify underlying cause of hypernatraemia. If able, measure urine and serum osmolality.

- If urine osmolality is less than serum osmolality (Dilute urine); this suggests diabetes insipidus
- If urine osmolality is greater than serum osmolality (Concentrated urine); this suggests dehydration.
- If urine and serum osmolality are similar this suggests osmotic diuresis which is often caused by Hyperosmolar Hyperglycaemic State (HHS/HONK). This will be accompanied by very high blood glucose.

General Management

Treat underlying cause once identified. This is as important as treatment of hypernatraemia.

Review sodium content of medicines e.g. effervescent tablets, dispersible paracetamol. Medicines with a high sodium content may benefit from a review by pharmacy.

If a patient requires a feed via an enteral tube a dietician referral should be made as low sodium alternatives may be suggested. Nutritional supplements with a high sodium content may also benefit from review by dieticians

Consider calculating Free water deficit:

Free water deficit (L) = TBW × weight(kg) × ([current Na(mmol/L) / 140] – 1)

- Where % total body water (TBW) is, Adult male: use 0.6 in the equation, Adult female: 0.5, Elderly male: 0.5, Elderly female: 0.45, Child: 0.6.

Mild hypernatraemia
Replace missing body water with oral water (not electrolyte drinks).

If able to initiate IV fluids (e.g. on a Community ward) initiate glucose 5% slowly (for example 1L / 6 hours). Utilisation of the equation above may be helpful to guide fluid replacement (to provide an estimate of the amount of fluid required). Switching to oral fluids as soon as possible is recommended e.g. when serum sodium <145 mmol/L.

If it is felt the patient requires IV fluids (e.g. glucose 5%) the patient will need referral to the local Acute Trust if unable to administer.

Severe hypernatraemia
Severe cases of hypernatraemia (Na>160mmol/L): Refer to the local Acute Trust.

Additional Information / Considerations

If patient is hypovolaemic and hypernatraemic referral to the local Acute Trust is recommended for fluid resuscitation with sodium chloride 0.9% to restore circulatory volume. Further fluid replacement can be reviewed once circulatory volume is corrected.

- If able to administer IV fluids consider initiating IV sodium chloride 0.9% as per NICE CG174: Intravenous fluid therapy in adults in hospital (https://www.nice.org.uk/guidance/cg174) prior to transfer to local acute Trust.

The rate of reduction of serum sodium should not occur more rapidly than about 10mmol/L per day.

If the patient is symptomatic of hypernatraemia, investigations are suggestive of a cause that is not simple dehydration or the hypernatraemia is refractory, contact the Medical registrar or Endocrinology for advice.

3.5. Hypomagnesaemia (serum magnesium <0.7mmol/L)[1][4][5][6][8]

Signs and Symptoms

Muscle weakness ● muscle spasm or cramp ● fatigue ● tremors ● seizures ● cardiac arrhythmias ● tachycardia ● abnormal eye movements ● electrolyte disturbances (see below).

CNS disturbances include: confusion ● agitation ● apathy ● depression ● hallucinations.

Assessment / Monitoring

- Confirm with repeat urgent serum magnesium
- Check serum potassium, adjusted calcium and phosphate as frequently occurs with other electrolyte disorders
- Liver function tests / History of chronic alcoholism?
- Check for ECG changes and arrhythmias.
- Adults with normal renal function: Magnesium levels should be monitored daily, and the dose adjusted as necessary.
- Adults with renal impairment (CrCl <30 ml/minute): Patients with renal impairment should have the doses of magnesium halved as reduced urinary magnesium excretion puts the patient at risk of hypermagnesaemia.
- Establish and correct cause if possible.
    - GI - Malabsorption or excess loss.
    - Malnutrition and Refeeding risk
    - Renal - Renal tubular defects or osmotic diuresis (DM)

General Management

Review medication for drugs which may induce hypomagnesaemia:

- Proton Pump inhibitors (PPI) e.g. lansoprazole, omeprazole
- Thiazide diuretics (e.g. bendroflumethiazide) and loop diruetics (furosemide) – [rarely]
- Immunosupressants e.g. ciclosporin, tacrolimus (Only under specialist advice)
- Laxative abuse
- Also aminoglycosides, amphotericin, theophylline, cytotoxics (cisplatin) and salbutamol

Drug Therapy / Treatment

Magnesium aspartate sachets (Magnaspartate 243mg) contains 10mmol of Magnesium
Magnesium glycerophosphate tablets contain 4mmol of magnesium

Suggested doses for each of these formulations can be seen below. In a patient with normal renal function the aim is to replace 20mmol of magnesium / day.

Serum magnesium 0.3 – 0.7mmol/L AND the patient is non-symptomatic

1st line: Magnesium aspartate sachet 1-2 sachet daily in divided doses

2nd line: Magnesium glycerophosphate tablets 1-2 tablet three times daily

- Monitor serum levels and review need for ongoing treatment after 3 days
- Reduce dose if diarrhoea occurs (common side effect of oral magnesium)

Serum magnesium less than 0.3mmol/L OR the patient is showing signs of hypomagnesaemia:

If able to administer IV fluids: Prescribe IV magnesium sulphate: 20mmol per day for up to 5 days.

10mmol in 500ml sodium chloride 0.9% to be infused over 4-12 hours (2 bags per day)

20mmol in 100ml glucose 5% can be infused over 3 hours if IV access/fluid restricted

- Monitor serum levels daily

Assess for life threatening complications of hypomagnesaemia: dysrhythmia, muscle weakness, haemodynamic instability. If any of these are present the patient will need to be referred to the local Acute Trust. Consider initiating IV/oral therapy as described above whilst awaiting transfer.

If unable to administer IV fluids: refer to the local Acute Trust as IV magnesium is indicated.

Additional Information / Considerations

Magnesium is mainly an intracellular ion and so serum concentrations are not an exact measurement of total body stores

Hypomagnesaemia is often associated with other electrolyte disturbances such as reduced: potassium (K^+), calcium (Ca^{2+}), phosphate (PO_4^{3-}) or sodium (Na^+) may co-exist with low Magneisum (Mg^{2+}).

In renal impairment (Creatinine clearance <30ml/min) reduce dose of oral replacement AND IV replacement by 50%.

3.6. Hypermagnesaemia (serum magnesium >1mmol/L)[1][7][8]

Signs and Symptoms

Nausea ● vomiting ● flushing ● thirst ● hypotension ● drowsiness ● confusion ● absent relflexes (due to neuromuscular blockade) ● respiratory depression ● slurred speech ● diplopia ● muscle weakness ● arrhythmia ● coma ● cardiac arrest.

Assessment / Monitoring

Confirm with repeat serum magnesium urgently
Renal function
Blood glucose
Arterial blood gases (to assess respiratory depression)
Blood ketones
ECG (to assess for SA or AV node block)

General Management

Usually removing the source of magnesium and encouraging urine output is sufficient.

In addition to magnesium supplements magnesium may be found in antacids, laxatives and bowel cleansing preparations. Review if any of these are prescribed or bought regularly over the counter.

Close monitoring of magnesium levels may be required in patients with impaired renal function.

Additional Information / Considerations

Treatment of hypermagnesaemia is unlikely to be required unless the serum magnesium concentration exceeds 2.0mmol/L.

Magnesium serum concentration is >4 mmol/L or symptomatic (toxicity may occur if magnesium > 2.5 mmol/L):

Refer to the local Acute Trust for advice and/or admission.

- Diuretics may be recommended to maintain good urine output
- If able to give IV fluids – IV calcium gluconate may be indicated to temporarily antagonise the effect of magnesium on the cardiovascular system
- Dialysis may be required

3.7. Hypophosphataemia (serum phosphate <0.7mmol/L)[1][8][9]

Signs and Symptoms
Hypophosphataemia may be asymptomatic, but clinical symptoms usually become apparent when serum phosphate concentrations fall below 0.3 mmol/L.

Lethargy ● muscle weakness ● parathesia ● confusion ● delirium ● irritability ● dysrhythmia ● convulsions ● respiratory failure ● coma.

Usually asymptomatic - suspect in patients with current or history of alcohol excess or if patient malnourished

Assessment / Monitoring
- Check BCP daily in moderate to severe hypophosphatemia and adjust treatment as needed
- Vitamin D and calcium levels (correction of phosphate can worsen hypocalcaemia)
- Assess history of alcoholism
- Review treatment: Insulin (if appropriate, associated with intracellular shift in phosphate)
- Assess risk of refeeding syndrome. This is a **medical emergency,** refer to the local Acute Trust.
- Renal cause? E.g. dialysis, phosphate binders

Drug Therapy / Treatment
Phosphate sandoz tablets contain 16mmol of phosphate, 3 mmol of potassium and 20mmol of sodium.

A phosphate polyfusor contains 50mmol of phosphate/500ml (1mmol/10ml). It also contains 9.5mmol potassium and 81 mmol sodium/500ml, therefore monitor potassium levels.

Mild hypophosphataemia (0.6 – 0.69mmol/L)
No treatment required, address the likely cause.

Moderate hypophosphataemia (0.3 – 0.59mmol/L) AND asymptomatic
Phosphate Sandoz 1-2 tablets three times a day.

If the patient has a phosphate level of 0.3-0.5mmol/L and is symptomatic or is unlikely to absorb oral phosphate, consider referral to the local Acute Trust.

If able to administer IV phosphate replacement
Phosphate polyfusor 9mmol (90ml) over 12 hours (rate 7.5ml/hr for 12 hours), then discard remainder of the bottle.

- Monitor serum levels and review need for ongoing treatment after 3 days

Severe hypophosphataemia (<0.3mmol/L)
Treatment should be guided following a senior review as IV phosphate replacement is indicated.

- Check BCP daily and adjust treatment as needed

Normal renal function
Phosphate polyfusor 18mmol (180ml) over 12 hours (rate 15ml/hr for 12 hours), then discard remainder of the bottle.

Impaired renal function
Phosphate polyfusor 9mmol (90ml) over 12 hours (rate 7.5ml/hr for 12 hours), then discard remainder of the bottle.

Additional Information / Considerations
Diarrhoea is a common side effect of oral phosphate therapy and may necessitate a reduction in dose. Give in at least 120ml of water to reduce the risk of diarrhoea.

Hyperphosphataemia is commonly associated with CKD. Consider lower starting dose of phosphate supplementation in patients with impaired renal function.

The underlying cause must be identified and addressed, otherwise it is likely hypophosphatemia is likely to recur.

IV phosphate should not be infused via the same line as magnesium or calcium due incompatibility (risk of precipitation)

3.8. Hypocalcaemia (adjusted serum calcium <2.1mmol/L)[8][10][11][12]

Signs and Symptoms
Nausea ● Vomiting ● paraesthesia ● muscle cramps ● tremors ● muscle weakness ● tetany ● laryngospasm ● arrhythmias ● prolonged QTc ● hypotension ● seizures ● behavioural changes ● changes in mental state (pyschosis ● depression) ● Chvostek's sign ● Trousseau's sign.

Assessment / Monitoring

- Confirm with repeat serum calcium level or ionised calcium on blood gas (if this facility is available)
- Rule out iatrogenic causes – EDTA contamination
- Serum albumin (If low albumin, ionised calcium on arterial blood gas if available)
- ECG – arrhythmias, prolonged QTc
- Establish cause of hypocalcaemia and seek senior advice if necessary. (likely to recur if no cause identified)
- Assess whether patient is symptomatic (e.g. tetany)
- Serum magnesium (hypocalcaemia can be secondary to hypomagnesaemia and correction of hypomagnesaemia may correct hypocalcaemia)
- Serum phosphate
- Monitor renal function, metabolic acidosis and urea
- Parathyroid hormone (PTH) and vitamin D levels

Drug Therapy / Treatment
Oral calcium salts should be used in the event of hypocalcaemia. The recommended daily dose of elemental calcium is 1-3g (2.25-6.75mmol) daily,

Calvive 1000 effervescent tablets (formerly Sandocal) contain 2263 mg of calcium lactate gluconate and 1750 mg of calcium carbonate (equivalent to 1000 mg or 25 mmol of calcium).

Calcichew chewable tablets contain calcium carbonate equivalent to 500mg of elemental calcium.

Mild to Moderate Hypocalcaemia (adjusted serum calcium <2.1mmol/L) AND asymptomatic
Calvive 1000 effervescent tablets 1 tablet twice daily. (this is a suggested starting dose).

Calcichew tablets 2 tablets twice daily is an alternative option. (this is a suggested starting dose).

Once deficiency rectified adjust dose on an individual patient basis.

If oral replacement is ineffective after 2-3 days in asymptomatic patients, please seek endocrinology advice.

If calcium continues to fall despite treatment please refer the patient to the local Acute Trust.

Severe Hypocalcaemia (adjusted serum calcium <1.8mmol/L) OR if signs of hypercalcaemic tetany

This is a **medical emergency**. Refer to the local Acute Trust. IV calcium gluconate replacement is indicated.

Additional Information / Considerations

Hypocalcaemia may be due to deficiencies of calcium homeostatic mechanisms, secondary to high phosphate levels or other causes.

Hypocalcaemia may be related to vitamin D deficiency. See specific HERPC "**Clinical Guideline for testing and replacement of Vitamin D**" available at:
https://www.hey.nhs.uk/herpc/prescribing-guidelines/

Constipation and gastro-intestinal irritation is associated with calcium replacement therapy.

3.9. Hypercalcaemia (adjusted serum calcium > 2.6mmol/L)[1][8][19]

Signs and Symptoms

Abdominal pain ● vomiting ● constipation ● polyuria ● polydipsia ● depression ● weight loss ● weakness, tiredness ● hypertension ● confusion ● fatigue ● bone pain ● pyrexia ● short QT interval.

Assessment / Monitoring

- Serum calcium should be monitored regularly
- U&Es to assess hydration status and renal function
- Parathyroid hormone (PTH), thyroid function and alkaline phosphatase (if cause not known)
- Albumin and phosphate levels
- Vitamin D
- Lithium associated (can increase calcium reabsorption in the kidney)

General Management

Hypercalcaemia with low or supressed PTH:

- Most commonly caused by malignancy; however is also associated with hyperthyroidism/ thyrotoxicosis, Addison's disease, sarcoidosis, vitamin D deficiency and Lithium.
- Consider measuring vitamin D levels and a myeloma screen. Oncology or Haematology referral should be made based on results.
- Refer the patient to an endocrinologist if no apparent cause identified.

Hypercalcaemia with raised or inappropriately normal PTH

- Most commonly caused by primary hyperparathyroidism; also tertiary hyperparathyroidism, familial hypocalciuric hypercalcaemia. Medications such as thiazide diuretics are also implicated.
- Consider monitoring diet, assessing for prolonged inactivity, review drug chart for any causes e.g. calcium supplement/thiazide diuretics.
- Refer the patient to an endocrinologist.

Drug Therapy/Treatment

Moderate hypercalcaemia (If adjusted calcium is ≤3.4)

- Rehydration with IV sodium chloride 0.9% is indicated to correct volume deficit. 2-3 litres over the first 24 hours may be needed, taking into consideration fluid status, urine output, renal function and cardiac function. (I.e. more caution may be needed in heart failure, renal failure or elderly patients). Consider seeking advice from endocrinology.
- After 24 - 48 hours seek advice from/ make referral to endocrinology.
- If unable to administer IV fluids refer to the local Acute Trust for treatment.

Review medications and ensure any calcium supplements or thiazide diuretics are discontinued if appropriate. Recheck serum calcium levels 3 weeks later.

In life threatening hypercalcaemia (adjusted calcium > 3.4mmol/L)

- If able, initiate the patient on IV sodium chloride 1L over 4 hours if not contra-indicated **AND**
- An urgent transfer to the local Acute Trust will be required for emergency IV fluids, senior medical review. An endocrine referral and/or dialysis may be considered.

Additional Information / Considerations

Hypercalcaemia can be a medical emergency and severe hypercalcaemia requires an urgent senior medical review and/or referral to an endocrinologist.

Hypercalcaemia can be the presenting feature of serious disease such as malignancy.

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