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General malaise in an elderly male patient

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Abstract

Patients with general malaise or feeling non-specifically unwell is a common scenario faced by all doctors. This case study reviews the made up case of an elderly male with these symptoms presenting to his primary care practitioner and his subsequent management in hospital. An evidence based clinical question and discussion of the ethical and personal impact of the case is also presented.

Note: This article is based on the Knol entitled "Common Cases: General malaise in an elderly male patient" accessed through http://knol.google.com/k/w-kent/common-cases-general-malaise-in-an/1blm6ty1i8a7z/6?collectionId=1bbsle13m97c0.391# on the 4th July 2011.

Presenting problem & summary of history

PC: General malaise for 3/12

HPC: This 73 year old male presented to his GP with undifferentiated symptoms and non-specific complaints of feeling of general malaise. He had been a frequent attendee to his GP since the recent tragic death of his sister-in-law. The GP had started Mr C on an antidepressant but also thought it prudent to request a blood screen, because the patient had not had a blood test for 3 years.

The blood tests revealed an elevated potassium level (7.2 mmol/l) and the GP organised admission to hospital. Despite this high potassium Mr C denied any symptoms of chest pain, palpitations, shortness of breath, nausea and vomiting, weakness, or urinary symptoms. He also denied dietary supplementation of potassium or use of any drugs with known hyperkalaemic side effects. A systems review revealed nil of note.

PMH: He had known:

1. Gout,
2. Hypertension, &
3. Depression.

DH: NKDA

On admission:

Drug	Dose/frequency	Indication	Prescriber
Allopurinol	200 mg / od	Gout prophylaxis	GP
Venaflaxine	75 mg / od	Depression	GP
Indomethacin	500 mg/ tds / (PRN)	Only during acute gout (none taken for >3/12)	GP
Inderal LA	160 mg / od	Hypertension	GP
Atorvastatin	40 mg / od	Elevated cholesterol	GP

FH: Nil of note.

SH: Mr C is a retired printer is independently mobile and lives with his wife in their privately owned house with 1 flight of stairs. He is an occasional drinker (<21 units/week) but denies ever smoking.

Examination findings

On examination Mr C was a well-built man who looked well, he was in no obvious discomfort and was alert (GCS 15/15) and orientated in time and space (AMT 10/10). His blood pressure was elevated at 191/91 mmHg but all his other vital observations were normal (apyrexial 37.6oC, heart rate 62 bpm, saturations 99% on air) and there were no clinically evident peripheral stigmata of disease (no jaundice, anaemia, clubbing, cyanosis, oedema, lymphadenopathy or goitre).

A cardiovascular examination revealed good perfusion, no signs of dehydration, normal heart sounds and peripheral pulses were palpable and no abdominal bruits were audible. A respiratory, gastrointestinal and neurological examination also failed to detect any abnormalities.

Summary: A retired 73-year-old male with known hypertension, gout and depression presented with a 3/12 history of general lethargy and malaise. Physical examination was normal apart from hypertension (191/91 mmHg). A screening blood test revealed hyperkalaemia (7.2 mmol/l).

Differential diagnoses

Causes of hyperkalaemia can be classified by pathophysiological mechanism into increased potassium intake, decreased potassium excretion or intracellular to extracellular shift of potassium.1 Decreased excretion is by far the most common underlying mechanism and the most common cause is the use of drugs affecting the renin-angiotensin system frequently combined with some degree of renal impairment, followed by other independent predictors of elevated serum potassium such as diabetes mellitus. Other unusual causes such as myeloma, increased ingestion of potassium, type IV renal tubular acidosis, and abnormalities of steroid metabolism should only be sought once more common causes are excluded.

Investigations

Bloods:

- Haematological: FBC (check for anaemia and type which may indicate or support underlying cause e.g. normocytic, normochromic in acute haemolysis or anaemia of chronic disease in chronic renal failure).

Biochemistry:

- U&Es & Cr level (assess renal function and degree of hyperkalaemia and to rule out a spurious result).
- Ca2+ level (in renal failure hypocalcaemia can exacerbate cardiac arrythmia potential).
- Glucose level to check for DM.

Urinalysis:

- Urine dip.
- Check volume and electrolytes.

Radiology:

- USS KUB (to screen for structural abnormalities and renal artery stenosis as a cause of impaired renal function).

ECG:

- Allow assessment of intracellular potassium & monitoring of patient.

Other tests:

- Fundoscopy (was performed to screen for end organ damage from hypertension).
- A "myeloma screen' (FBC, ESR, serum & urine electrophoresis, renal function tests & bone profile) was also carried out in this patient to rule out myeloma leading to renal dysfunction and subsequent hyperkalaemia.

The investigations confirmed the hyperkalaemia (7.1 mmol/l), and demonstrated impaired renal function (urea 23.5, Cr 494, eGFR 10) and marginal hypocalcaemia (2.14) as well as anaemia (Hb 9 g/dl). The urine dip was negative for infection but total urine protein was 2.46 g/l. fundoscopy revealed mild hypertensive retinopathy. The myeloma screen was negative but the ECG showed characteristic changes associated with hyperkalaemia (small p waves, broad QRS, and tall tented T waves). The voltage criteria for left ventricular hypertrophy was also fulfilled presumably due to the patients long standing hypertension. Interestingly the USS KUB reported a normal right kidney but the left kidney could not be identified and after discussion with the regional renal unit a renal MRA was ordered.

Management

Despite not having overt symptoms according to the hyperkalaemia guidelines a potassium levels of 7.1 mmol/l is classified as severe and there was ECG changes, both of which are independent indicators for urgent treatment. [1,2]

Urgent treatment involved placing the patient on telemetry and giving 10ml of 10% calcium gluconate over 2 minutes to protect the cardiac membrane and prevent fatal arrhythmia. The next step is to shift potassium into cells using followed by insulin-glucose IV (10 units Actrapid in 50ml of glucose 50%) infused over 30 minutes and salbutamol 10mg by nebuliser. The final stage was to remove potassium from the body using calcium polystyrene sulphonate resin (calcium resonium) with regular lactulose to remove potassium from the GI tract.

Clinical decision making

Assessment of the patient involves[1,2]:

Questions	This case
1. Confirm true hyperkalaemia	Yes (by repeat bloods)
2. How severe is the hyperkalaemia?	Severe (>7mmol/l)
3. Is urgent treatment required?	Yes (>7mmol/l & ECG changes)
4. Why has this patient got hyperkalaemia	Full history, examination & investigation (Acute on chronic renal failure, secondary to hypertension)

Evidence base

What is the risk of a 73 year-old nondiabetic male with hypertensive chronic kidney disease (CKD) developing hyperkalaemia?

The reported incidence of hyperkalaemia (K+ > 5.5 mmol/l) in hospitals varies between 1-10%, and it is most prevalent at the extremes of life.1,2 It is an important diagnosis to consider and make because it is potentially life threatening, mortality rates can be as high as 67% if severe hyperkalaemia is not rapidly treated.[3]

In a study[4] randomly allocating 1094 male nondiabetic participants with hypertensive CKD to ACE inhibitor (ACEi), Beta blocker (BB), or dihydropyridine calcium channel blocker (CCB) therapy it was found that the risk of hyperkalaemia was small, especially if the GFR was higher than 40mL/min/1.73m^2. During the study 80 events (K+ >5.5 mmol/l) in 51 participants were recorded. The risk of hyperkalaemia was higher in the ACEi group compared with those taking CCB (HR, 7.00; 95% CI, 2.29-21.39 [P < .001]) or BB (HR, 2.85; 95% CI, 1.50-5.42 [P = .001]). The hazard ratio (HR) for hyperkalemia also varied with GFR. In patients with a GFR between 31 and 40 mL/min/1.73 m(2) and a GFR lower than 30 mL/min/1.73 m(2) was 3.61 (95% confidence interval [CI], 1.42-9.18 [P = .007]) and 6.81 (95% CI, 2.67-17.35 [P < .001]), respectively. However, there was no increased risk of hyperkalemia if GFR was 41 to 50 mL/min/1.73 m(2). Interestingly a secondary finding was that concomitant dieuretic therapy was associated with a 59% reduction of hyperkalaemia.

These results suggest that the risk of hyperkalaemia is low and the use of diuretic therapy deserves further investigation in the management of nondiabetic men with hypertensive CKD.

Effect of illness episode on patient and family/carers

The patient was cheerful throughout his admission and was very interested in his management and the possible reasons for his hyperkalaemia, especially as he did not feel unwell. He was well supported by his wife and after a few days in hospital for investigation he was very keen to return home. Although he was intrigued to find out more about his absent left kidney.

Ethical / governance issues:

Is this case the GP ordered blood tests without a definite question in mind for the tests to answer. The use of investigations in this manner has been critisised as potentially wasteful because it does not follow guidelines.[5] However, the results were potentially life saving for this individual, who had a marked changes on his ECG secondary to servere hyperkalaemia. Thus, the use of blood tests in this situation was justified and shows the value of doctors acting on experience and clinical suspicion.

Summary

The urgent management of hyperkalaemia is an important topic for physicians because of the risk of mortality from cardiac arrythmias associated with the condition. This case study presents both population level guideline evidence and evidence based practice focused on the individual patient. It is hoped that by reading this article medical students are more confident in the initial assessment and management of hyperkalaemia.

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