Understanding the challenges of elderly collapse in emergency departments: metabolic and cardiovascular health: a complex relationship

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Case Report

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Understanding the challenges of elderly collapse in emergency departments: metabolic and cardiovascular health: a complex relationship

Abstract

Collapse is a significant and common presenting complaint in the accident and emergency departments globally, with varying incidence and prevalence influenced by regional health profiles and healthcare systems. It is leading to almost twenty percent of all admission to an acute hospital amongst the elderly population. It encompasses a wide range of underlying causes, from benign to life-threatening conditions that need to be excluded by Acute Physicians. This case highlights the importance of maintaining metabolic equilibrium and intricated relationship to cardiovascular health. It highlights impetus for adequate vitamin D levels for overall mineral balance and bone-cardiovascular health. It summarises the regulatory mechanisms of vitamin D and its interactions with key electrolytes and hormones such as calcium, phosphate and magnesium as well as parathyroid hormone. It is important that these metabolic changes can be recognised so that the appropriate treatment and preventative measures are taken.

Keywords: metabolic health, vitamin d (calcitriol), cardiovascular health, hormone

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Abbreviations: PTH, parathyroid hormone; UV, ultra-violet; CT, computer tomography; MRI, magnetic resonance imaging; ECG, electrocardiogram

Introduction

Across the world, emergency departments (EDs) are seeing a rising number of elderly patients presenting with collapse, which includes conditions like fainting (syncope), near-fainting, and general weakness. These incidents are alarming for both the individuals and their families. In their 60s, people start to face more chronic health issues, increasing the risk of collapse. In their 70s, about 20% of syncope-related ED visits are from this age group, often linked to cardiovascular issues. The 80s see a significant spike, with 3-5% of elderly ED visits due to syncope, caused by arrhythmias, blood pressure issues, and medication effects. For those in their 90s, collapse-related visits are even higher, up to 15-20%, with compounded health issues and increased frailty making them particularly vulnerable.

Hypocalcaemia is a life-threatening condition that is most commonly associated with vitamin D deficiency. Calcium is the most abundant mineral in the body and is needed for a variety of metabolic processes such as muscle contraction,[1] so it is important that adequate levels are maintained. Vitamin D is important for controlling calcium absorption in the gut therefore it's depletion can lead to decreased calcium levels in the bloodstream.[2] Hypocalcaemia secondary to vitamin D deficiency is prevalent in regions where there is low sunlight exposure and inadequate dietary vitamin D supply. Low calcium levels can cause life-threatening cardiac arrythmias that may present as a collapse.[3]

Case presentation

A 78-year-old man presented to hospital following a vacant episode. He recently had a right femoral-above-knee popliteal bypass surgery and has a past medical history of aortic valve disease, type 2 diabetes mellitus, hypertension, chronic obstructive pulmonary disease and peripheral vascular disease. The patient does not remember the episode and felt confused afterwards. His wife reported that he started to clutch at his chest; he went pale, then started to 'stare into a blank space' and did not respond to his name being called. The episode lasted for about one minute. His wife stated that he has had a similar episode about 5 years ago. He did not remember anything and denied feeling any chest pain. When asked if he was taking his calcium supplements, he said he was not sure and advised to ask his wife. A head CT was done and referenced to the patient's head MRI which was done previously. The head CT showed no acute haemorrhage, major vascular territory infarct or space occupying lesion. There was periventricular hypoattenuation which is likely secondary to chronic small vessel disease. There were generalised involuntary changes with compensatory dilation of ventricular spaces. Structures of the midline and posterior cranial fossa were normal and there was no skull vault fracture. Mastoid air cells were well aerated. As seen in the previous MRI, there was mucosal thickening of right maxillary, right ethmoid and right part of frontal sinus which suggests sinusitis or sinus disease. An ECG was performed which showed QTc prolongation of 477 and ST changes which was thought to be due to severe vitamin D deficiency leading to low calcium and magnesium. Once the electrolytes were normalised, QTc interval corrected immediately; patient reporting improvement in his symptoms (Figures1-3).

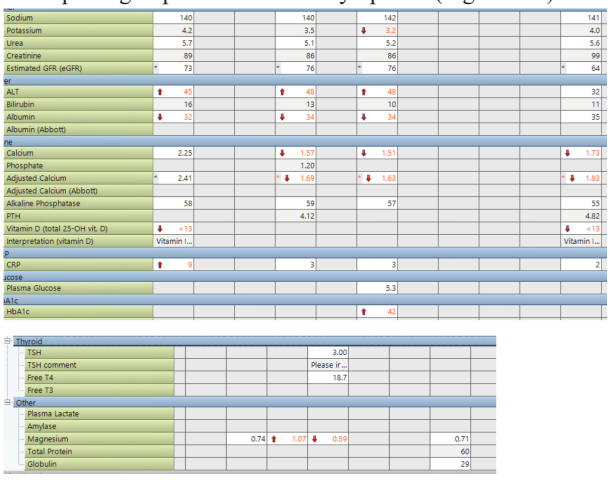

Figure 1 Investigations on admission with low serum calcium, Magnesium and Vitamin D levels.

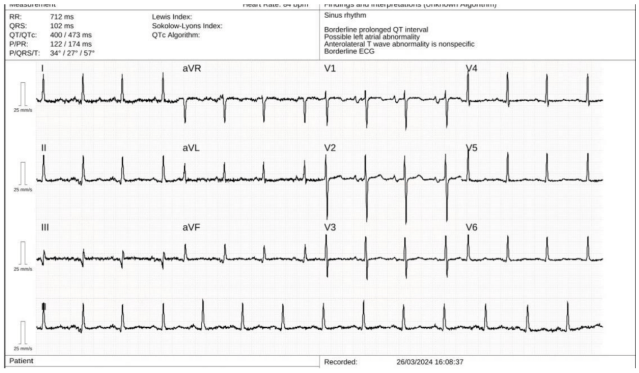

Figure 2 QTc prolonged 473 corresponding to low Calcium levels 1.67.

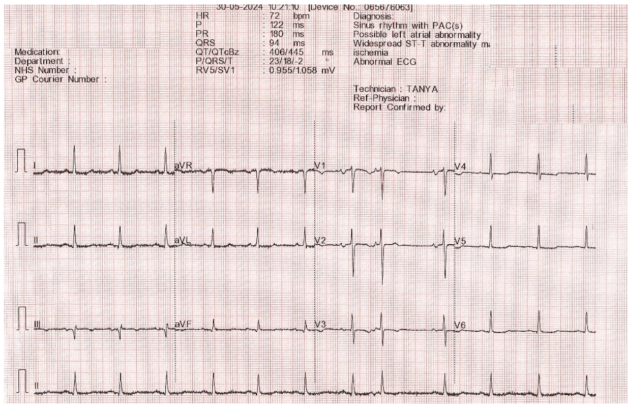

Figure 3 QTc interval graph.

Discussion

Calcium and mechanism of arrhythmias

A process known as excitation-contraction coupling enables the heart to convert electrical activation into mechanical force. This process relies on calcium ions which flow through calcium channels and trigger the release of more calcium ions from the sarcoplasmic reticulum.[4] The calcium ions bind to cardiac troponin thus causing myofilament contraction. The amount of calcium ions released from the sarcoplasmic reticulum determines the strength of systolic contraction.[4]

Electrolyte imbalances can alter ionic currents in cardiac cells which can cause arrhythmias.[1] Calcium has multiple roles which include the regulation of neural transmission and maintaining membrane stability, bone structure, blood coagulation and intracellular signalling.[1] When the serum calcium level drops below the lower limit of normal range (2.12mmol/L) this is defined as hypocalcaemia. Patients with severe hypocalcaemia may present with features of neuromuscular irritability. Depleted calcium levels can lead to ST segment modification; QT interval prolongation and T wave alterations, which can result in life-threatening ventricular arrhythmias.[5] Low calcium levels causes prolongation of the plateau phase of the cardiac action potential which causes calcium ion channels to remain open longer which allows a late calcium inflow causing tachycardia and ventricular arrythmias which are known as torsade de pointes.[6,7] Patients with hypocalcaemia can present with exertional syncope due to tachycardia.[8] Patients with congenital long QT syndrome will also have atrial arrhythmias along with ventricular ones, which are known as atrial torsade de pointes.[9] Other clinical features of hypocalcaemia are paraesthesia, muscle cramps and seizures of all types.

Vitamin D levels and calcium

The causes of hypocalcaemia can be classified into hypoparathyroid disorders and secondary hyperparathyroidism. Hypocalcaemia is most commonly associated with hypoparathyroidism, vitamin D deficiency, acute renal failure or hypomagnesia.[10] Vitamin D plays a pivotal role in calcium homeostasis. It is crucial for intestinal absorption of calcium and bone resorption and decreases renal excretion of calcium and phosphate.[2] When vitamin D levels are adequate, calcium absorption is efficient, therefore serum calcium levels are normal. Conversely, severe vitamin D deficiency leads to a decline in calcium and phosphorus absorption, resulting in hypocalcaemia.[2]

Parathyroid hormone (PTH) plays a crucial role in regulating calcium levels by promoting bone and renal calcium reabsorption. PTH affects calcium absorption indirectly through its ability to regulate renal vitamin D metabolism and control serum levels of the vitamin D hormone. When calcium levels decrease, serum PTH increases. This can occur due to vitamin D deficiency which causes hypocalcaemia and rapidly stimulates the secretion of PTH by the parathyroid glands which is known as secondary hyperparathyroidism.[11].

Phosphate levels are also impacted by vitamin D and PTH. Vitamin D increases serum phosphate mainly through enhancing intestinal phosphate absorption.[12] Increased PTH also reduces serum phosphate. Consequently, vitamin D deficiency can result in hypophosphatemia through increased excretion of phosphate in the kidneys.[12]

Magnesium is important for the metabolism of vitamin D as it acts as a cofactor in enzymatic reactions for vitamin D activation in the liver and kidneys. Increased vitamin D levels have been shown to increase serum magnesium levels,[13] therefore vitamin D deficiency can also be the cause of hypomagnesia.

The estimated prevalence of vitamin D deficiency in Europe was 13%.[14]However it is important to note that these estimates do not take into account the differences by ethnicity, as dark skinned eth nic groups within Europe are at much increased risk of vitamin D deficiency compared to their white counterparts.[14] Vitamin D deficiency is defined as a serum 25(OH)D concentration outside the range of 25-75 nmol/L.[15] The major causes of vitamin D deficiency are a combination of low ultraviolet B exposure and low dietary vitamin D. Studies show that 90% of adults and children in specific populations in Europe are at risk of vitamin D deficiency.[15] Furthermore, additional factors can increase the risk of vitamin D deficiency, such as age, obesity, inflammation, malabsorption syndromes and certain drugs that interact with vitamin D. Therefore due to the effects of low vitamin D levels on other electrolytes and hormones, these populations are potentially at much increased risk of hypocalcaemia and hypophosphatemia.[2, 12]

It is recommended that adults who are vitamin D deficient are given 20000 IU/d of vitamin D2 or vitamin D3 for 8 weeks followed by maintenance therapy of 1500-2000 IU/d. Higher doses are needed in high risk patients such as those with obesity, malabsorption syndromes or patients on vitamin D altering drugs.[16]

Conclusion

Vitamin D deficiency leads to a cascade of changes in electrolyte and hormone levels. Hypocalcaemia occurs due to impaired calcium absorption in the gut. Secondary hyperparathyroidism: caused by elevated PTH levels in response to low calcium levels. Hypophosphatemia: is a result of increased renal excretion of phosphate driven by elevated PTH. Hypomagnesia, as a result of low vitamin D levels needs to be addressed proactively for promoting metabolic cardiovascular health

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The patient presented with a collapse and QTc prolongation with ST changes. This case aims to recognise the clinical manifestations of hypocalcaemia secondary to vitamin D deficiency and understand the changes in electrolytes and hormone levels caused by vitamin D deficiency.

Early recognition of these dys-electrolyemia and biomarkers of metabolic well-being can be one of the remedial interventions for the elderly cohort of patients. Future research should be directed to offered bespoke treatments and preventative measures to patients at high risk to minimise burden on health care across the globe.

Learning Points

a) Metabolic and cardiovascular health are very intricately intertwined. Understand the factors at play is crucial to understand one of the most common presentation to Emergency departments. Hypocalcaemia can be a life-threatening condition that can present as a collapse, loss of consciousness or seizure.

b) Vitamin D deficiency is one of the most common causes of hypocalcaemia. Populations in low sunlight regions and of ethnic minorities are at a higher risk of vitamin D deficiency.

c) Vitamin D is a hormone that is involved in the metabolism of other electrolytes such as calcium, magnesium and phosphate. It is important to closely monitor and maintain adequate vitamin D levels to maintain metabolic homeostasis.

Acknowledgments

None.

Conflicts of interest

The authors declare there are no conflicts of interest.

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