Unusual presentation of more common disease/injury

Decompensated heart failure secondary to hypocalcaemia post coronary artery bypass grafting

Sheila Grecian, Mark Ainslie, James Liliker, Jaydeep Sarma

Department of Cardiology, Wythenshawe Hospital, Manchester, UK

Correspondence to Dr Mark Ainslie, markpeterainslie@hotmail.com

Summary

A man presented with an exacerbation of heart failure following coronary artery bypass grafting. He was found to be severely hypocalcaemic secondary to a combination of decreased parathyroid reserve and severe vitamin D deficiency. On treatment of his hypocalcaemia, all symptoms resolved. This case highlights the importance of recognising that metabolic derangement can affect cardiac function.

BACKGROUND

It is known that metabolic derangement can give rise to avoidable postoperative complications. Hypocalcaemia is a common problem with potentially serious consequences and in this case exacerbated postoperative heart failure. Of interest, this case additionally highlights important interactions between vitamin D and parathyroid hormone (PTH) in calcium metabolism.

CASE PRESENTATION

A 70-year-old house-bound man presented with breathlessness. He was 15 days post uncomplicated coronary artery bypass and 5 days postdischarge. On examination, signs suggested heart failure with bilateral leg oedema, a raised jugular venous pressure and bi-basal crackles. His medical history included type 2 diabetes mellitus, ischaemic heart disease, atrial fibrillation, chronic kidney disease (CKD) stage 3, chronic obstructive airways disease and thyroidectomy in 2004.

Chest x-ray confirmed pulmonary congestion, and bedside echo showed moderate left ventricular (LV) systolic impairment with a small pericardial effusion. He was treated with intravenous diuretics, and initially symptoms and signs improved.

However, he developed a progressive muscular weakness, resulting in reduced mobility, and became depressed. The symptoms were initially attributed to acute on chronic renal impairment as renal function worsened. Diuretics were stopped without clinical response.

It was noted that, on a background of longstanding hypocalcaemia, his calcium levels were decreasing (table 1). The worsening hypocalcaemia offered a potential cause of his symptoms and prompted further investigation.

DIFFERENTIAL DIAGNOSIS

We reviewed potential causes of worsening hypocalcaemia.

Acute exacerbation of hypocalcaemia secondary to electrolyte derangement was considered. It is known that hypomagnesaemia and hypermagnesaemia, hyperphosphataemia and alkalosis can lead to calcium derangement. Rhabdomyolysis is a well-known cause of hyperphosphataemia. This man's creatine kinase was elevated (2163 IU/l) with phosphate level 1.99 mmol/l. Statin therapy was considered the most likely culprit, and his statin was stopped. However, calcium levels and his clinical condition did not resolve. In addition, hypocalcaemia is normally only a feature in the acute stages of rhabdomyolysis, and thus its non-resolution suggests that, although a factor in his deterioration, this was unlikely to be the significant cause of his hypocalcaemia. In this gentleman's case, his magnesium levels were normal (0.7 mmol/l) and pH was normal on arterial blood gas.

Vitamin D deficiency secondary to lack of sunlight, poor diet, malabsorption or chronic renal failure is a common cause of hypocalcaemia. In this gentleman, both deficiency secondary to lack of sunlight and poor diet were possibilities given his social history. A coeliac screen was negative. This man's estimated glomerular filtration rate was 42, equivalent to CKD stage 3. Vitamin D deficiency secondary to renal failure is not normally seen until CKD stages 4 and 5.

Hypoparathyroidism is the third major cause of hypocalcaemia. This gentleman had had a thyroidectomy 6 years earlier. It had been presumed that iatrogenic hypoparathyroidism would have presented earlier, but on questioning he reported 3 years of leg cramps for which he had been

Table 1 Blood biochemistry

	Na (mmol/l)	K (mmol/l)	Urea (mmol/l)	Creatinine (μmol/l)	eGFR (MDRD)	Hb (g/l)	Ca (corr) (mmol/l)	PO ₄ (mmol/l)
Preop	138	4.5	8.3	106	64	130	1.92	1.13
Discharge Postsurgery	141	4.3	6.8	117	57	90	2.16	1.19
On admission	139	4.8	12.6	141	46	96	1.7	1.23
After diuretic treatment	132	4.8	20.7	152	42	98	1.39	1.99

eGFR, estimated glomerular filtration rate; MDRD, modification of diet in renal disease study equation.

prescribed quinine. This may in retrospect have been due to hypocalcaemia and would explain at least the baseline hypocalcaemia.

TREATMENT

Further tests revealed both a low PTH (35 pg/ml with calcium 1.53 mmol/l) and low vitamin D (25-OH-VitD3 <6) suggesting that hypocalcaemia was secondary to a combination of hypoparathyroidism and vitamin D deficiency.

He was treated with intravenous calcium infusion followed by oral calcichew D3 and 1 α calcidol. As calcium levels normalised, independent mobility returned and depression lifted. His cardiac function improved and repeat echo showed only mild LV systolic impairment.

OUTCOME AND FOLLOW-UP

Once his calcium levels normalised, he made a rapid recovery and was discharged with both cardiology and endocrine follow-up.

DISCUSSION

In this case, hypocalcaemia was a result of a combination of decreased parathyroid reserve post-thyroidectomy and vitamin D deficiency (diet, lack of sunlight and perhaps renal impairment). With no immediate preoperative 'calcium panel' bloods available, exactly what precipitated his acute decompensation is uncertain. However, the high phosphate (1.99 mmol/l) favours decreased parathyroid reserve as the dominant influence. It is postulated that decreased parathyroid reserve resulted in the parathyroid axis being unable to compensate for stresses placed upon it by vitamin D deficiency and acute renal failure induced by diuretics and rhabdomyolysis.

Hypocalcaemia certainly contributed to this gentleman's heart failure. It is possible that if this man's hypocalcaemia had been more fully investigated and corrected preoperatively, he may have avoided an admission with decompensated heart failure.

Of note, the role of vitamin D in the development of clinically significant hypocalcaemia in patients with decreased parathyroid reserve has been noted in other studies. Patients who undergo minimally invasive

parathyroidectomy for parathyroid adenomas have a significantly higher risk of developing hypocalcaemia if they have concurrent vitamin D deficiency.¹ Similarly, in the same patient group those who were vitamin D deficient had significantly higher preoperative PTH levels, indicating the need for PTH compensation in the face of vitamin D deficiency.¹ Hypoparathyroidism presenting many years after thyroid surgery has also been reported.²

Calcium is vital for cellular processes, none more so than in maintaining normal cardiac function. It is therefore important for levels to be normalised before any surgery but perhaps of particular importance in cardiac surgery.

Note: All calcium levels mentioned in this report are 'corrected'.

Learning points

- Complications of thyroidectomy may not be immediately apparent – the patient had enough PTH to cope on a day-to-day basis but failed to compensate in the face of severe vitamin D deficiency, worsening renal function and perioperative stress.
- Preoperative bloods indicated hypocalcaemia. Although asymptomatic at the time, as metabolic derangement can impact the perioperative course, further investigation and treatment would be recommended in future cases.
- Low calcium and other metabolic disturbances are recognised causes of heart failure exacerbation, and they should be identified and treated promptly, especially when heart failure is not responsive to conventional treatment.

Competing interests None.

Patient consent Obtained.

REFERENCES

1. **Lang BH, Lo CY.** Vitamin D3 deficiency is associated with late-onset hypocalcemia after minimally invasive parathyroidectomy in a vitamin D borderline area. *World J Surg* 2010;**34**:1350–55.
2. **Halperin I, Nubiola A, Vendrell J, et al.** Late-onset hypocalcemia appearing years after thyroid surgery. *J Endocrinol Invest* 1989;**12**:419–20.

This pdf has been created automatically from the final edited text and images.

Copyright 2011 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.

BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Please cite this article as follows (you will need to access the article online to obtain the date of publication).

Grecian S, Ainslie M, Liliker J, Sarma J. Decompensated heart failure secondary to hypocalcaemia post coronary artery bypass grafting. *BMJ Case Reports* 2011;10.1136/bcr.03.2011.4032, date of publication

Become a Fellow of BMJ Case Reports today and you can:

- Submit as many cases as you like
- Enjoy fast sympathetic peer review and rapid publication of accepted articles
- Access all the published articles
- Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow