Unexpected outcome (positive or negative) including adverse drug reactions

Improvement in refractory heart failure by nephrectomy for renal tumour

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Summary

Hypoalbuminaemia and anaemia are conditions known to aggravate congestive cardiac failure (CCF). Renal cell carcinoma is often associated with hypoalbuminaemia and anaemia. The authors report an interesting case of a patient with severe refractory CCF who incidentally was found to have a massive renal cell tumour. Clinically, his heart failure was being aggravated by hypoalbuminaemia and anaemia. Despite aggressive diuretic therapy and multiple blood and albumin transfusions, there was no clinical improvement. He subsequently underwent nephrectomy of the renal tumour, with subsequent dramatic clinical improvement in his heart failure. His symptoms resolved completely and he did not require any further diuretic therapy.

BACKGROUND

Starling's hypothesis states that a decrease in the serum colloid osmotic pressure–pulmonary artery wedge pressure (COP–PAWP) gradient is the main cause of pulmonary oedema.¹ Significant decrease in the COP–PAWP gradient is usually related to a major increase in PAWP because of left ventricular failure. However, a substantial decline in COP can be an additional cause for the decrease of this gradient resulting in exacerbation of chronic heart failure (CHF).^{2–3} Hypoalbuminaemia, a common cause of decreased COP,^{4–5} is independently associated with increased mortality in patients admitted with congestive cardiac failure (CCF).^{6–7} In patients with CHF, even relatively mild degrees of anaemia are associated with worsening symptoms, functional status and survival.^{8–9} Renal cell carcinoma is known to cause both hypoalbuminaemia and anaemia and the severity is worse in advanced stages.¹⁰ Radical nephrectomy is an established treatment for renal tumour¹¹ but a relationship between radical nephrectomy and significant clinical improvement of refractory CHF is yet to be reported.

CASE PRESENTATION

Our patient, a 48-year-old man, had a background of ischaemic cardiomyopathy (ejection fraction (EF) of 35% on echocardiogram performed in 2006) following an anterolateral ST segment elevation myocardial infarction. He was admitted with symptoms of dyspnoea (New York Heart Association class III) along with worsening leg oedema. Physical examination showed decompensated CCF with jugular venous pressure elevated up to the earlobes, severe bilateral lower limb oedema up to the abdominal wall and scrotum. Chest auscultation revealed bilateral lower zone chest crepitations. Blood results showed iron deficiency anaemia (haemoglobin 7.9 g/dl (13–17 g/dl), ferritin 186.9 ng/ml (12–300 ng/ml), serum iron level 2.4 mcg/dl (60–170 mcg/dl)) and hypoalbuminaemia (serum albumin 18 g/l

(36–52 g/l)). A chest x-ray confirmed pulmonary congestion and an echocardiogram demonstrated deterioration in left ventricular function with an EF 21% (Modified Simpsons biplane technique). Pulmonary artery systolic pressure was calculated as 39 mm Hg. He had gained 21 kg of body weight (67 to 88 kg) over the preceding 3 months. Despite aggressive treatment with intravenous diuretic and nitrate infusions, blood transfusions and albumin replacement for 2 weeks, there was no clinical improvement.

A renal ultrasound was organised as the renal function showed slight deterioration. It showed a markedly enlarged left kidney raising a possibility of renal cell carcinoma. This was confirmed on a CT scan (figures 1 and 2)

Figure 1 CT abdomen confirmed the presence of massive left renal tumour.

Figure 2 3D reconstruction of CT images showing the size of left renal tumour.

which showed a massive left-sided renal tumour measuring 20×17 cm in diameter.

His symptoms proved refractory to aggressive medical treatment including frusemide infusions up to 300 mg/day. He received a total of 16 units of blood (8 litres of red cell concentrate) and 20 units of albumin (2 litres of 20% human albumin solution) over the course of 3 weeks with no significant improvement.

The outcome of a multi-disciplinary meeting between the cardiologists, urological surgeons and anaesthetists was to offer him a high-risk left-sided radical nephrectomy. The obvious risks and benefits were outlined to the patient who consented. Following surgery, there was a dramatic improvement in his symptoms of dyspnoea and peripheral oedema. There was complete resolution of signs of heart failure with return of body weight to baseline. Repeat echocardiogram postoperatively showed EF 29% and he was discharged home 15 days after surgery. Histology of the tumour revealed grade 4 clear cell and sarcomatoid type. He was readmitted soon afterwards with clinical and chemical dehydration due to frusemide 40 mg once daily which corrected on cessation of diuretics and thereafter did not require any further regular diuretic therapy.

DISCUSSION

Correction of anaemia is a proven treatment in patients with CHF.¹² Anaemia caused by heart failure is multifactorial but is usually mild. In cases of severe or refractory anaemia, other causes should be considered. Similarly, severe hypoalbuminaemia is unlikely to be due to CHF alone^{6 7} and should also be investigated and treated accordingly.

Following radical nephrectomy, both anaemia and hypoalbuminaemia have been shown to improve.^{13 14} Our patient received multiple blood and albumin transfusions with no significant response. Ultimately, only a high-risk radical nephrectomy proved beneficial.

Learning points

- ▶ Hypoalbuminaemia and anaemia are associated with some renal cell carcinomas.
- ▶ When a patient presents with heart failure we should always look for exacerbating causes.
- ▶ Definitive treatment of the underlying cause of illness (in this case a nephrectomy) often is the only approach that results in clinical improvement (supportive care with transfusions and albumin were ineffective).

Competing interests None.

Patient consent Obtained.

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

Shahzad A, Powell C, Etherington R, Somauroo J. Improvement in refractory heart failure by nephrectomy for renal tumour.

BMJ Case Reports 2012;10.1136/bcr.06.2011.4331, Published XXX

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