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## Fatal cardiac failure after a single dose of doxorubicin in myeloma-associated cardiac amyloid

Sir,

While the cardiotoxic effects of cumulative doses of doxorubicin are well recognized,[1,2] the potential that this drug has for causing acute cardiac dysfunction is less well recognized. This is likely to be particularly important where there is pre-existing cardiac disease. We report a case where exposure to a single dose of doxorubicin resulted in fatal deterioration in cardiac function.

A 53 year old male presented with nephrotic syndrome and was found to have lambda light chain myeloma. Renal biopsy showed amyloid. Before treatment was started he developed acute pancreatitis which was complicated by renal impairment and severe fluid overload, with radiological evidence of pulmonary oedema. At this time blood pressure was 90/60 mmHg; electrocardiography showed low voltage complexes. Calculated echocardiographic ejection fraction was 47%; there was evidence of septal thickening (1.9 cm) but no chamber dilatation and no evidence of valvular disease or tamponade. Machine haemofiltration was instituted with correction of fluid overload and good biochemical control: there was no cardiovascular instability despite fluid removal of up to 3 kg per treatment. Five days later, treatment with doxorubicin 9 mg/m$^2$, vincristine 0.4 mg, and methylprednisolone 1 g/m$^2$ was given. Twenty-four hours after the initial dose he became hypotensive (70/50 mmHg) despite adequate filling pressures. Thermodilution cardiac output was 5.5 l/min despite treatment with dobutamine 28 μg/kg/min. Despite addition of adrenaline, dopamine, and enoximone there was continued haemodynamic deterioration and cardiac arrest occurred 4 h after transfer. At post mortem there was extensive amyloidosis involving the liver, spleen, kidneys and heart. The coronary arteries were patent and there was no evidence of infarction.

There are 3 previously published cases of fatal cardiac failure after low dose anthracycline administration; one case of fatal cardiac failure without evidence of coronary disease after 3 × 70 mg/m$^2$ daunorubicin (total dose 420 mg),[3] and 2 cases of fatal cardiac failure following 50 mg and 80 mg; one of these patients had pre-existing coronary disease.[4] In addition, there is one report of non-fatal myocardial infarction following each of 2 doses of doxorubicin[5] although this is not the usual mechanism of anthracycline cardiotoxicity. In the present case there was no evidence of ischaemic heart disease or sepsis and the patient was stable although hypotensive until the drug was administered. We conclude that doxorubicin toxicity, superimposed on pre-existing cardiac impairment caused by amyloidosis, was responsible for the patient's death. Caution is required in the administration of anthracyclines to patients where there is pre-existing myocardial disease. This has particular relevance to myeloma, where, as in this case, cardiac amyloid is a potential complication.

**Acknowledgements**

We thank Dr J. Feehally and Professor J. Walls for their permission to report on the clinical findings and Dr J. Mercer for her permission to report on the post mortem findings.

M.A.B. Devoy
C.R.V. Tomson
*Department of Nephrology,*
*Leicester General Hospital,*
*Gwendolen Road,*
*Leicester, LE5 4PW, UK.*

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## Intra-operative bumetanide in cadaveric renal transplants

Sir,

In cadaveric renal transplants, one aims to achieve primary function with diuresis on restoring the circulation, to make post-operative management much easier and obviate the need for dialysis. According to our protocol, a central venous pressure (CVP) line is placed in each recipient and intra-operatively they receive cyclosporine A infusion (2.5 mg/kg), methylprednisolone (15 mg/kg) and 100 ml of 10% mannitol. CVP is maintained between 10–15 cm of water. Following the revascularization of the graft, we hope to see diuresis within 5–10 min. If this does not occur, we have been encouraged by the use of intravenous bumetanide (2 mg) as a bolus. This has resulted in immediate diuresis and continued primary function of the graft in 6 patients.

Bumetanide is a potent 'high-ceiling' diuretic with a number of features which make it desirable for use in renal transplant patients. After intravenous injection, the diuresis starts within a few minutes with sharp peak and short duration of action.[1,2] The principal site of action is the thick ascending limb of Henle's loop where it inhibits the active reabsorption of $Na^+$ and $Cl^-$. There is evidence to suggest that it also has minor action at the proximal tubules via carbonic anhydrase inhibition. Bumetanide enhances glomerular filtration rate and renal plasma flow