sequesters tissue-bound and albumin-bound digoxin. Decrease in plasma levels of free digoxin results in intravascular sequestration of tissue-bound digoxin leading to much higher concentration of plasma digoxin levels (Kramer, 1977; Rabetoy et al, 1990; Ujhelyi and Robert, 1995).

Digoxin measurement after administration of Digibind is of little value as the plasma digoxin is predominantly bound and inactive. In such patients, clinical improvement with electrocardiographic changes should be used as markers of improvement rather than plasma digoxin levels. Digibind–digoxin complex can be cleared from the blood through glomerular filtration or through hepatic metabolism.

Digibind has a relatively shorter half-life compared to digoxin leading to rebound increase in free plasma digoxin in 12–24 hours in patients with normal renal function. However, its elimination half-life is significantly prolonged in renal failure, and this delays the rebound by 12–130 hours in patients with renal insufficiency. This allows more time for renal as well as hepatic elimination of the complex. The free digoxin resulting from this dissociation can bind to circulating albumin, Digibind or diffuse into tissues. Hence, the rebound increase in plasma digoxin can have unpredictable effects (Valdes and Jortani, 1998).

The fear of delayed rebound in patients with end-stage renal disease necessitates longer periods of monitoring in these patients, and removal of the complex through plasma filtration if large amounts of digoxin are ingested (Peterson and Peterson, 1986). This patient was taking digoxin 0.125 mg daily. He required Digibind administration to sequester intracellular digoxin. This was followed by haemodialysis to remove excessive potassium. He was observed in the hospital for a few days. By the end of first week, his electrocardiogram returned to baseline and plasma digoxin returned to normal level.

Conclusions

Digoxin toxicity is not uncommon in patients with renal failure. Standard 12-lead electrocardiogram helps detect cases of digoxin toxicity early, and helps guide subsequent management. BJHM

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Computed tomography visualization of intracerebral haematoma 'pouring' directly into ventricular system

An 83-year-old woman was admitted to a university teaching hospital following an episode of collapse. She had a background history of atrial fibrillation, hypertension and ischaemic heart disease. She was on aspirin but no other anticoagulation medication. On admission, her Glasgow Coma Scale was 15/15 initially and when this deteriorated to 8/15, 1 day after admission, a computed tomography (CT) scan of the brain was done. The CT showed a 2 cm intraparenchymal haemorrhage within the right parietal lobe. Blood was seen to extend out of the haematoma and was seen to directly 'pour' into the adjacent lateral ventricle (*Figure 1*). Blood was also seen to lie dependently in the occipital horns of both lateral ventricles. The patient was treated conservatively and unfortunately deteriorated further and died the following day. BJHM

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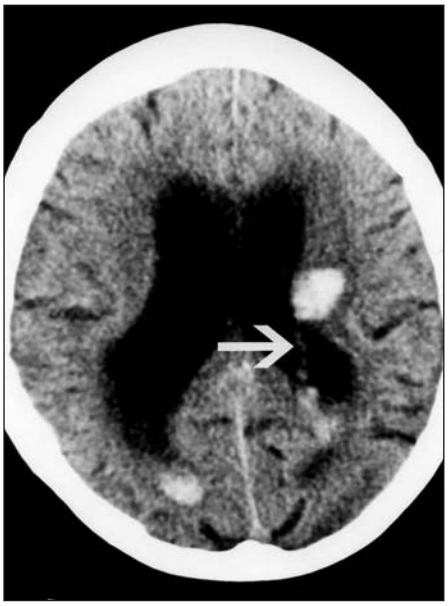

Figure 1. Axial non-contrast enhanced computed tomography scan of the brain demonstrates right parietal intracranial haemorrhage with direct extension into the adjacent lateral ventricle resulting in a 'pouring' effect.