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Letters to the Editor

Artifical penile nodules

Sir,

A male 26 year old Filipino attended for non-specific urethritis. On examination of the genitalia, apart from urethritis, there were two nodules proximal to the glans penis which were soft in consistency. The patient related that these nodules were pieces of plastic which had been implanted one year before in Thailand. The patient was happy with them as they caused no discomfort.

Implantation of beads under the penile skin to enhance coital excitement and orgasm of the sexual partner is common in the Far East and South East Asia.[1] Foreign bodies such as stones, plastic beads, glass and pearls are used for this purpose. They are known by different names such as 'bulletus' and 'chagan balls'.[2] The number of objects implanted may vary from two to five and, usually, they cause no harm to the penis.

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P.M. Abdul Gaffoor
Department of Dermatology,
Hamad General Hospital,
Doha,
Qatar.

References

1. Lim, K.B., Seow, C.S., Tulip, T., Danial, M. & Vijayasingham, S.M. Artifical penile nodules: case reports. *Genitourin Med* 1986, **62:** 123–125.
2. Sugathan, P. Bulletus. *Int J Dermatol* 1987, **26:** 51.

Acute hepatic dysfunction following parenteral amiodarone administration

Sir,

We report a case in which intravenous administration of amiodarone was associated with acute deterioration of liver function.

A 59 year old man presented with a 2-week history of progressive congestive cardiac failure due to cardiomyopathy. On examination he was in uncontrolled atrial fibrillation, and had clinical and radiological signs of pulmonary congestion, and gross peripheral oedema. The liver was enlarged three fingerbreadths, and ultrasound examination confirmed hepatic congestion. His heart failure responded well to intravenous diuretics over the next 4 days, though his ventricular response rate remained high despite digoxin. Liver function tests were mildly deranged on admission due to hepatic congestion but initially improved.

His atrial fibrillation remained difficult to control and amiodarone was started on day 6. He received 450 mg intravenously for 5 days and 200 mg for a further 2 days. He became progressively more jaundiced following the administration of amiodarone despite a sustained improvement in the control of his heart failure. Liver function tests deteriorated with the aspartate transaminase rising to ten times the normal value (Figure 1). There was no evidence of haemolysis and repeat liver ultrasound showed mild hepatic congestion only with no signs of biliary obstruction. Heptatic serology and auto-antibody screen were negative. An adverse reaction to amiodarone was suspected, and liver function tests rapidly improved following its withdrawal. The patient's clinical state steadily improved.

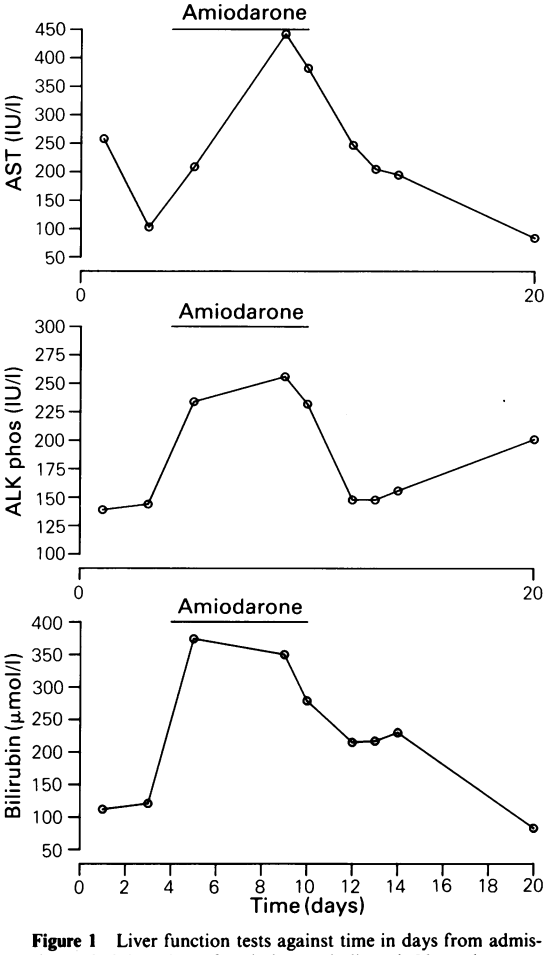

Figure 1 Liver function tests against time in days from admission: administration of amiodarone indicated. Normal ranges: AST 7–40 IU/1; alkaline phosphatase (Alk phos) 30–100 IU/1; bilirubin 2–14 μmol/1.

We believe that the clinical course suggests that the acute deterioration in liver function tests was a direct effect of amiodarone. Abnormal liver function tests associated with long term amiodarone therapy have been well documented,[1] and direct hepato-toxicity has been implicated. Liver biopsy reveals histological changes similar to alcoholic liver disease.[2] There have also been reports of fatal cirrhosis attributable to long term amiodarone therapy.[3,4]

Acute hepatic dysfunction following intravenous amiodarone (2 cases) has only recently been reported.[5] As in our case liver function returned to normal following withdrawal of amiodarone, suggesting direct hepatotoxicity. The liver is probably more susceptible to amiodarone toxicity when it is already compromised in congestive cardiac failure. Parenteral amiodarone is commonly used to control acute cardiac arrhythmias, and the general physician should be aware of this important adverse reaction.

R.N. Stevenson
T.H. Nayani
J.R. Davies
Whittington Hospital,
Highgate Hill,
London N19 5NF, UK.

References

1. Harris, L., McKenna, W.J., Rowland, E. *et al.* Side effects of long term amiodarone therapy. *Circulation* 1983, **67:** 45–51.
2. Rigas, B., Rosenfield, L.E., Barwick, K.W. *et al.* Amiodarone hepatotoxicity. A clinicopathologic study of five patients. *Ann Intern Med* 1986, **104:** 348–351.
3. Rinder, H.M., Love, J.C. & Wescler, R. Amiodarone hepatotoxicity. *N Engl J Med* 1986, **314:** 318–319.
4. Lim, P.K., Trewby, P.N., Storey, G.C. & Hold, D.W. Neuropathy and fatal hepatitis in a patient receiving amiodarone. *Br Med J* 1984, **288:** 1638–1639.
5. Pye, M., Northcote, R.J. & Cobbe, S.M. Acute hepatitis after parenteral amiodarone administration. *Br Heart J* 1988, **59:** 690–691.