Acute Digoxin Toxicity in a Neonate

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Digoxin an indispensible therapeutic agent has a narrow (0.9-2 ng/ml) therapeutic range(1) and low toxic level(3 ng/ml). Minor mistakes in dosage calculations and documentation such as a missed decimal point(2) can hence be lethal. This case reports our experience in the management of a neonate with acute digoxin overdosage.

Case Report

A 12-day-old term neonate weighing 2750 g presented with breathlessness and poor feeding. On examination she was in congestive heart failure with a systolic murmur Grade 3/6 at left sternal border. Associated anomalies noted were an anteposed anus and a rudimentary left thumb. *X*-ray chest and abdomen revealed cardiomegaly and 2 lumbar hemivertebrae. 2 D Echo revealed an atrioseptal and ventriculoseptal defect.

For rapid digitalization, a stat dose of injection digoxin (0.02 mg/kg) was given. 0.25 mg of digoxin was infused 12 hours later instead of 0.025 mg. An ECG done on realizing this oversight revealed a heart rate of 105/min, a prolonged PR interval (0.20 sec) with depressed ST segment(3-4 mm). Few hours later the baby vomited, developed a bradyarrhythmia and multifocal clonic convulsions.

Blood was collected for serum digoxin levels and 25 mg of IV phenytoin sodium was injected to combat the arrhythmia and convulsions. The serum K^+ level was 5.1 mEq/L and serum digoxin level 14.2 ng/ml (N=0.9 to 2 ng/ml). Six days after omitting digoxin, the repeat level was 2.2 ng/ml and oral digoxin liquid was restarted with proper dose monitoring.

Discussion

Digoxin has a low therapeutic index and hence the incidence of its toxicity ranges between 20-25%(3,4). High mortality rates correlate with serum levels >6 ng/ml(5). Due to inadvertent placement of a decimal point, ten times the advised dose was administered in our case. Morbidity and mortality associated with its toxicity has reduced considerably in recent years due to better understanding of its pharmacology and newer modes of treatment(4,6). Prematures and infants less than 3 months as also those with hypokalemia, hypomagnesemia and acid-base imbalance are at greater risk of toxicity.

Treatment of toxicity consists of omitting the drug and administration of K^+ if its serum levels are low, as is often noted with chronic digoxin overdosage. Serum K^+ levels may be increased in acute toxicity as digoxin inhibits ATP-ase pump causing a rise in serum K^+ levels, which may in itself be lethal. Nearly 35% of the drug is excreted by the kidneys and its half-life is 36 hours.

Thus digoxin must be omitted for 5-6 days before restarting therapy. For digoxin induced arrhythmia, parenterally admini-

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stered lignocaine (1 mg/kg) or phenytoin sodium (3.5 mg/kg over 5 minutes) can be used. Convulsions are rare, as in our case, and require higher doses of phenytoin. Anti digoxin antibody fragments (Fab) to the drug (digibind) is highly effective and is used in severe toxicity, especially if no response is noted to antiarrythemic agents(7). However, digibind is expensive and is not routinely available to us.

In conclusion, double checking of drug dose calculations should be practised, close monitoring and prompt correction of metabolic and ECG abnormalities while withholding the drug is sufficient in early cases. Whereas phenytoin sodium is effective in correcting the arrhythmia in most patients, digibind may be required in more severe cases.

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Nifedipine in Urticaria

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The efficacy of nifedipine, a calcium channel antagonist in the treatment of chronic idiopathic urticaria in adults is documented(1,2). This study was done to assess the efficacy and safety of nifedipine in children with giant urticaria and angioneurotic edema.

Subjects and Methods

Six children with giant urticaria and 2 children with angioneurotic edema were the subjects for this study. An informed oral consent was obtained from the parents/guardians of the child. A 5 mg, capsule of nifedipine was punctured with a needle and approximately half of its con-

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