Case Report

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Digoxin Toxicity in Infant Presenting as 1st degree Heart Block

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Abstract

Digoxin plays an important role in management of pediatric heart failure. Digoxin toxicity can occur even at thereupatic doses. We are reporting a case of 47 day old male baby who presented with vomiting, letharginess and first degree heart block on electrocardiogram. Toxicity was confirmed with serum level of 4.82 ng/mL. Baby was managed conservatively, by stopping digoxin and monitoring of heart rate. Baby improved on its own by day 6 of admission.

Keywords: Digoxin Toxicity; Heart Block.

Introduction

Digoxin plays an important role in management of pediatric heart failure. Despite the lack of data regarding its use in children, digoxin continues to be used by most clinicians in the management of pediatric heart failure due to various causes [1]. The widespread availability, low cost, and continued confidence in the usefulness of the drug based on long years of experience are some of the reasons for its continued use.

The major causes of digitalis toxicity in infantsand children are therapeutic administration and acute accidental overdose, the former being the more frequent modality. Digoxin increases intracellular calcium in myocardial cells indirectly, by inhibiting the sodium–potassium pump in the cell membrane. Increased intracellular calcium increases cardiac contractility, but also the risk of tachyarrhythmias [2]. Inhibition of this pump causes the hyperkalaemia commonly seen in toxicity. Digoxin also causes an increase in vagal activity, reducing activity in the sinus node and prolonging conduction in the atrioventricular node [3,4].

The elimination of digoxin is mainly by renal clearance and is prolonged in patients with renal impairment. Transport by P-glycoprotein also contributes to elimination. Consequently, a higher serum digoxin concentration for a given dose occurs in patients with renal impairment, lower body weight and in those taking amiodarone, verapamil, macrolides, azole antifungals and cyclosporin, which inhibit P-glycoprotein transport [2]. There is a narrow difference between the therapeutic and toxic range of digoxin.

The clinical features of toxicity are often non-specific. They commonly include lethargy, confusion and gastrointestinal symptoms (anorexia, nausea, vomiting, diarrhoea and abdominal pain). ECG changes in digoxin toxicity includes flattening or inversion of T wave, prolonged PR interval (1st degree heart block), 2nd or 3rd degree heart block due to inhibition of atrioventricular node [5-7]. Sinus arrest have also been reported [8].

Case

We are reporting a case of digoxin toxicity, presenting with first degree heart block. A 47 day male baby, weighing 3.7Kg, a known case of congenital acyanotic heart disease -moderate size

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Investigation	Observed Value	Unit	Biological Reference Interval
Digoxin (Lanoxin) (Serum,ECLIA)	4.82	ng/mL	Therapeutic range: 0.90-2.0

Medical Remarks: CORRELATE CLINICALLY

Interpretation :

- After oral Digoxin, the absorption, distribution and bioavailability of the drug depends on various factors including form of the drug, presence of naturally occurring enteric bacteria, strenuous physical activity, concomitant use of other drugs & Individual's ability to metabolize and respond to digoxin. Susceptibility to toxicity apparently increases with age.
- After oral administration there is an early rise in serum concentration. Equilibrium of serum and tissue levels requires 6-8 hours. For this reason blood specimens for digoxin analysis should be drawn at least 6-8 hours after drug administration

-- End of Report --

Fig. 1: Digoxin report2

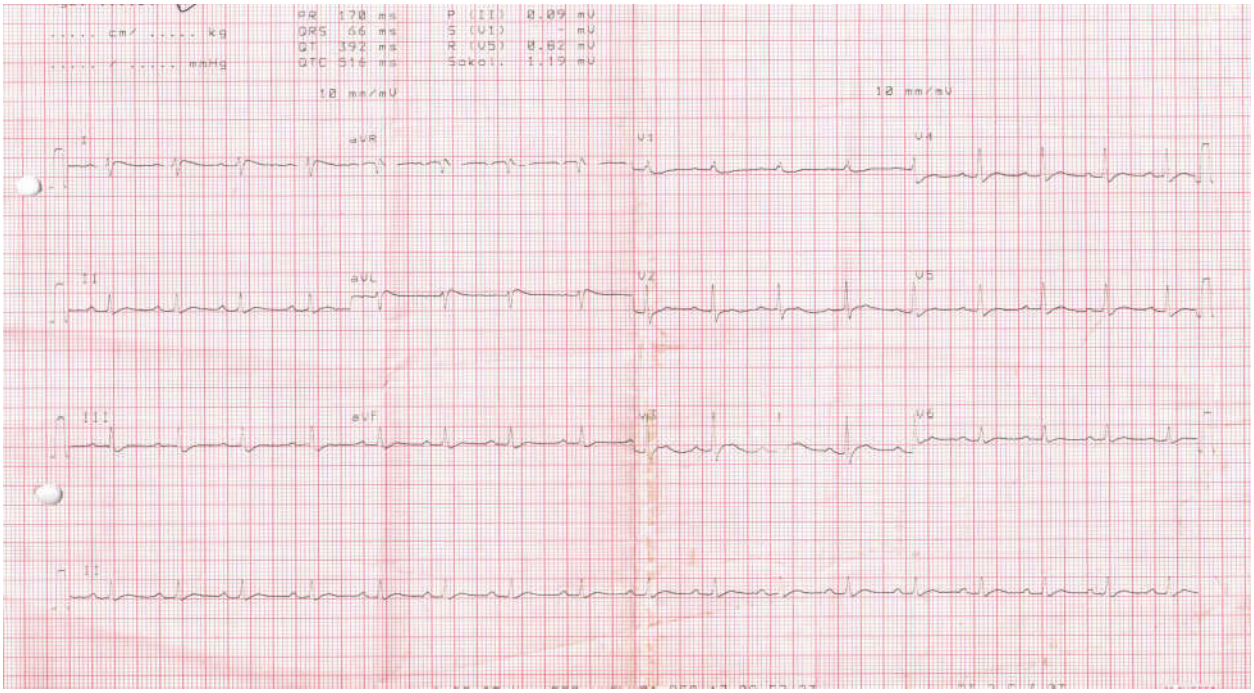

Fig. 2: ECG digoxin2

ASD and VSD (2mm) was diagnosed at age of day 20 of life. Baby was sent home, on oral digoxin at 10microgram/kg/day, furosemide 2mg/kg/day and enalapril 0.1mg/kg/day at day 28 of life. Baby came back after 20 days at day 47 of life, with complaints of vomiting, letharginess. Sepsis was ruled out with normal CBC CRP and blood culture. Baby was suspected to be a case of digoxin toxicity, in view of vomiting, letharginess and grade 1 heart block on ECG (PR interval 170 miliseconds) figure 1. Digoxin level was sent and was found to be raised to toxic level i.e 4.82 ng/mL. Figure 2 (Normal therapeutic level of digoxin 0.9-2.0 ng/mL). Serum potassium level of baby was 4.02 meq/l. Baby was having heart rate ranging from 90 to 120 beats/min. Digibind could not be given due to non availability. Plan was to start Isoprenaline if heart rate is less than 80 beats/min. Digoxin was stopped and child was managed conservatively for digoxin toxicity. Iv fluids were given for 3 days and gradually shifted to oral feeds. Baby's heart rate never reached below 80/min, at day 6 of admission ECG showed normal sinus rhythm with normalization of PR interval. Repeat digoxin level after 10 days was 0.9ng/mL which was in therapeutic range. During hospital admission, baby's condition improved gradually, was accepting and tolerating breast feed well, vitals were stable and no vomiting, so he was discharged.

Conclusion

Digoxin toxicity can occur even at thereupatic doses due to variable pharmacokinetics in neonates and children. High index of suspicion is required to diagnose digoxin toxicity and differentiate it from sepsis or electrolyte disturbances due to its similar presentation with vomiting, letharginess. ECG features may vary in digoxin toxicity. In our case ECG was suggestive of first degree heart block. Digoxin toxicity can be managed conservatively without digi-bind.

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