Rare Case of Tegretol Overdose: A Case Report

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ABSTRACT

A 24-year-old Saudi female, known to be psychiatric, was brought to the Emergency Room with a suspicion of an accidental ingestion of unknown amount of tegretol (Carbamazepine) and Largactile (Phenothiazine) tablets. The patient was presented to the Emergency Room after more than four hours of ingestion. The stomach wash and repeated doses of activated charcoal was given. Although serum level of Carbamazepine was 31.5 ug/ml (Therapeutic range = 2-10 ug/ml) the patient vital signs were normal and she showed no signs of acute toxicity e.g., seizure, ventricular tachycardia, heart block, coma. One week later, she was discharged and referred to psychiatric treatment. This case report indicates that some patients unusually can tolerate even severe Carbamazepine poisoning.

(Key Words: Therapeutic Effect, Toxic Dose, Drug Overdose, Lethal Dose, Poisoning)

INTRODUCTION

Carbamazepine blocks voltage-gated sodium channels in a rate dependent manner. The CNS and cardiovascular effects observed in overdose may be related to the sodium channel blocking effect. Absorption is slow and erratic even in therapeutic doses with peak levels occurring between 4 to 8 hours. It distributes widely with a volume of distribution of 1.4 L/kg. It is about 75% bound to serum proteins. These factors are responsible for the significant increase in elimination with hemoperfusion.

The elimination half-life of Carbamazepine is 10 to 20 hours in chronic therapeutic doses. However, as there is considerable enzyme induction with chronic use, the half-life may be much longer in naive individuals. It is metabolized in the liver, primarily by the enzyme epoxide reductase to Carbamazepine 10-11 epoxide (Hardman et al, 1996). This metabolite is active as an anticonvulsant and also accounts for many of the adverse effects in therapeutic use.

Carbamazepine overdose leads to prolonged coma but is occasionally complicated by arrhythmias. The treatment is with aggressive gastrointestinal decontamination, even in late presenters, and respiratory support. Cardiac complications should be treated similar to those associated with Tricyclic Antidepressant poisoning. Clearance of Carbamazepine may be enhanced with repeated doses of activated charcoal (Bradbury & Vale, 1995) or charcoal hemoperfusion (Low et al, 1996).

DISCUSSION

After drug screening Carbamazepine was measured in patient serum. Its level was measured by TDX/FLX automated machine and was found to be 31.5 ug/ml. This was about three times the toxic dose. The therapeutic range is usually quoted as 2 to 10 ug/mL (Wallach J, 1998). At levels more than 10 and up to 20ug/mL nystagmus, ataxia and sedation occur. Concentrations of 20 to 40 ug/mL are associated with horizontal and vertical nystagmus, dysarthria, and the patient is generally unable to walk unaided, some patients may have delirium, coma, seizures and may require ventilation. Coma and profound respiratory depression are usual with levels greater than 40 ug/mL (170 umol/L) and life-threatening arrhythmias are much more common above such levels (Hojer et al, 1993; Spiller et al, 1990).

All other investigations of our patient were as follows: WBC = 9.6 THSD/CU MM, RBC = 4.09 MILL/CU MM, Hb = 12.2 GRAMS/DL, HCT = 34.9 %, PLT = 367 THSD/CU MM, Sugar = 6 mmol/L, Urea = 1.77 mmol/L, Creatinine = 62.6 umol/L, Na = 140 mmol/L, K= 3.6 mmol/L, CK = 58 U/L, LDH = 214 U/L, AST = 27, ALT = 42 U/L, ALP = 168 U/L, TBIL = 0.92 umol/L. All of these results are within normal limits.

CONCLUSIONS

According to the above results, electrolyte abnormality that may occur in Carbamazepine overdose was not seen. The patient was newly diagnosed epileptic and she took Carbamazepine for a week only. However the onset of enzyme induction by Carbamazepine is at about three days with maximum effect after one month, and there is a high interindividual variability to the extent to which induction occurs (Stan R, 1996). Furthermore, it is believed that Phenothiazines may potentiate the effect of Carbamazepine. Accordingly one should expect severe symptoms of toxicity in our patient. On the contrary our patient was asymptomatic which could be contributed to the inter-individual variation. She was kept under observation without any specific therapy. Five days later the patient was discharged and referred to the psychiatric evaluation and counseling.

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Cite As: Lebdeh, W.A.R.A., V.P. Gupta, and M.M.U. Kabiraj. 2001. Rare Case of Tegretol Overdose: A Case Report. *Greenwich Journal of Science and Technology*. 2(2):92-95.

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