LEVOTHYROXINE - ? A TRIVIAL POISONING

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Objectives: There have been only case reports but no series of massive deliberate self poisoning of thyroxine in adults. Paediatric thyroxine poisoning were reported to cause symptoms in 5-27% of patients with mild toxicities.[1-3] We aimed to investigate the clinical syndrome in a series of deliberate large thyroxine overdoses.

Case series: There were three patients who presented with six episodes of acute thryoxine poisoning with a dose range of 6 to 35 mg (median: 15.5 mg). In all patients there was a delay in the onset of symtpoms of 12 to 48 hrs. The clinical manifestations included tachycardia (maximum HR median 118/min, range: 108-140), hyperthermia (median 37.6^{o}C, range: 37 – 40), borderline hypertension (max SBP median 130 mm Hg, range: 120-140), agitation and tremor. Maximum HR appeared to correlate well with thyroxine dose ingested (r = 0.86 CI: 0.17-0.98 P=0.03). One had possible seizures after ingesting 24 mg. In all six admissions the free T4 were elevated with all but one > 77.2 pmol/L (N: 12-25), and low TSH, median 0.03 mIUl/L (Range: 0.01-0.07) (N: 0.27-4.2). All patients were treated with supportive therapy including intravenous fluids, benzodiazpines, paracetamol and beta-blockers (mainly propranolol). Their symptoms improved over 2 to 7 days with complete revovery.

Discussion: Levo-thyroxine has a half-life of 7 days and large volume distribution of 10 L. The thyroid hormones (T3 and T4) accelerate cellular oxidation, increase energy expenditure, leading to increased heat production, oxygen consumption and potentiates the effects of catecholamine. T3 and T4 are both metabolically active, but T3 is 3-5 times more potent than T4. In our patients there was a delayed onset in toxicity, consistent with previous reports and likely due to the delay in the conversion of T4 to T3 and distribution of T3 into tissues. Despite the large doses, the majority of the symptoms were only moderately severe and responded mainly to symptomatic treatment. Treatment of hyperthermia with passive cooling and fluid replacement may be required. If patients present within a few hours of ingestion, decontamination with activated charcoal is reasonable. Propranolol inhibits the peripheral conversion of T4 to T3 and organ effects and is the main treatment of symptomatic patients.

Conclusions: Serious toxicity after levothyroxine ingestion is uncommon, but the onset of symptoms may be delayed. Initial therapy includes supportive treatment and activated charcoal. Hospitalisation is usually not indicated especially in asymptomatic patients. Close outpatient follow up for several days is necessary.