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Case Report

Organophosphorous intoxication and hyperthyroidism

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ABSTRACT

It is well established that transient thyroid dysfunction can occur in many non-thyroid illness. But abnormal thyroid function test in the range of hyperthyroidism is not reported many a times. Here we present two cases of chlorpyrifos intoxication presenting with hyperthyroidism and its implications in the management of the patient in acute illness. Role of atropine in antagonizing muscarinic effects of organophosphorus intoxication is well documented. Interplay between atropine, hyperthyroid state and tachycardia discussed.

Keywords: Organophosphorus intoxication, Hyperthyroidism, Atropinisation, Tachyarrhythmia

INTRODUCTION

When authors received the first case they were not aware that organophosphorus poisoning can precipitate thyrotoxicosis. As the patient having persistent tachycardia thyroid functions were asked for. It was presumed that finding a hyperthyroidism was incidental. But reviewing the literature it was found that abnormal thyroid functions can occur in Organophosphorus poising.[1] The second case confirmed the finding. We are presenting these two cases to inform the medical profession that this abnormality can occur.

CASE REPORT

First case:

A female 37 years old was admitted in casualty on 30th April 2015 with a history of consuming 150 ml of chlorpyrifos at her residence an hour ago. She was restless, confused and continuously retching. Pupils were small reacting to light and pulse rate 140/min, BP 130/80 mmHg, respiratory rate 20/ min, temperature 37.c, and 98% oxygen saturation on room air. She was given 2 gm. pralidoxime by slow infusion followed by 500 mg / hour infusion. She was put on oxygen supplementation via mask. She received atropine 2 mg IV bolus and followed by infusion 1-3 mg / hour. Intravenous normal saline 100ml per hour started and admitted to ICU. Patient was persistently tachycardia 140- 160 beats / minute though pupils were small. Lungs were clear. Though atropine infusion rate was down regulated tachycardia persisted. Repeated ECG and Echocardiography did not show any abnormality. Thyroid function test was asked for which showed increased T4, normal T3 and low TSH. Patient developed proximal muscle weakness and could not hold her neck in flexion. She was put on propranolol 40 mg through nasogastric tube three times a day and Neomercazole 10 mg three times a day. Her heart rate gradually settled down, permitting to increase atropinisation.

In spite above measures she developed hypoxemia and tachypnoea necessitating mechanical ventilation. She was on mechanical ventilator for three days and could be weaned off successfully. She was shifted to ward on 13th day and discharged home two days later. TSH was on the lower range at the time of discharge. A month later her

thyroid functions were normal and she was taken off beta-blockers and ant thyroid drugs.

Investigation:

At time of admission: Blood counts, Random blood sugar, Serum creatinine, LFT, ESR, and Electrolytes were normal.

Antithyroid antibody < 15.00 IU/ml (negative <60 IU/ml)

Thyroid uptake scan was normal.

Butyryl Cholinesterase: 100 U/L (4300-11500 U/L)
T3: 99.92ng/dl (87-200 ng/dl)
T4: 18.08 ng/dl (3.2- 12.6 ng/dl)
TSH: 0.15 IU/ml (0.4-5.5 IU/ml)

At time of discharge: TSH 0.621 IU (0.4-5.5 IU/ml)

One month later:

T3: 149.92 ng/dl (87-200 ng/dl)
T4: 10.5 ng/dl (3.2- 12.6 ng/dl)
TSH: 1.11 gm IU/ml (0.4-5.5 IU/ml)

Second case:

A 21 year old Female patient was admitted on 16 June 2015 with a history of ingestion of chlorpyrifos 100 ml 2 hours prior to admission. She was conscious, but restless, nauseated. BP 100/70 mm Hg, Temperature 37.C, respiratory rate 20/ min, oxygen saturation 98%, pulse rate 100-130/ mt.

She was resuscitated in casualty with oxygen supplementation, intravenous pralidoxime 2 gm. IV bolus followed by 500 mg / hour infusion. Atropine 2 gm. IV followed by 1-3 mg / hour infusion. ECG was showing tachycardia, her thyroid functions were checked which are hyperthyroid range. She was admitted to ICU and managed with atropine infusion to maintain heart rate 100-130 / mt. Pralidoxime 500 mg/ hour infusion continued. She made uneventful recovery and at the time of discharge her thyroid function test was normal.

INVESTIGATION: Complete blood picture, Random blood sugar, Urea, Serum creatinine, LFT, ESR, Electrolytes and U/scan of abdomen were normal.

Butyryl Cholinesterase: 626 U/L (4300-11500 U/L)
Antithyroid antibody: 33.6ng /ml (0.2-55.0 ng/ml)
Thyroid up take scan is normal.
T3: 211 ng/dl (82-200 ng/dl)
T4: 12.77 ng/dl (3.2- 12.6 ng/dl)
TSH: 0.150 IU/ml (0.4-5.5IU/ml)

DISSCUSION

Hyperthyroidism produced by organophosphorus intoxication may intervene with management of poisoning. Undue tachycardia produced by excessive release of thyroid hormonal may prevent adequate atropinisation. Surge of thyroid hormones in pre-existing diagnosed or undiagnosed hyper thyroid patient may precipitate thyroid storm which can be fatal.[2] Human thyroid has cholinergic innervation AChE positive nerve fibres are localised in the wall of thyroid artery. Glandular tissue is provided with cholinergic nerve fibres localised between and around thyroid follicles.[3] over stimulation these fibres by excessive acetylcholine in organophosphorus poisoning may increase thyroid hormone secretion leading to hyperthyroidism. Adequate atropinisation will block the release of thyroid hormones.[4] Atropinisation may be inadequate as the undue tachycardia produced by the acute release of thyroid hormones may prevent physician to use adequate doses confidently. Tachycardia in elderly patients and in patients with pre-existing cardiac disease may be deleterious.[5] It has to be further studied by using beta-blockers and anti-thyroid drugs if we can control heart rate and use atropine in adequate doses. Variation of thyroid function abnormalities in organophosphate intoxication may depend on peroxonase-1 enzymes (PONI) activity, the higher being less variability. Thus thyroid function abnormality due to organophosphate intoxication may vary genetically.[6]

CONCLUSION

Thyroid has cholinergic innervations and adequate atropinisation may prevent hyperthyroidism and thyroid storm produced by organophosphate intoxication.
It may be hypothesised that blocking thyroid hormone with antithyroid drugs and judicious use of beta blockers may facilitate adequate atropinisation in organophosphate poisoning presenting with hyperthyroidism bio chemically.

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REFERENCES

1. Tseng FY, Chen CS. Thyroid function tests in acute drug intoxication. J Formos Med Assoc. 1992;91 Suppl 1:S68-73.
2. Yuan YD, Seak CJ, Lin CC, Lin LJ. Thyroid storm precipitated by organophosphate intoxication. Am J Emerg Med. 2007;25(7):861.e1-3.
3. Amenta F, Caporuscio D, Ferrante F, Porcelli F, Zomparelli M. Cholinergic nerves in the thyroid gland. Cell Tissue Res. 1978;195(2):367-70.
4. Van Sande J, Dumont JE, Melander A, Sundler F. Presence and influence of cholinergic nerves in the human thyroid. J Clin Endocrinol Metab. 1980;51(3):500-2.
5. Eddleston M, Dawson A, Karalliedde L, et al. Early management after self-poisoning with an organophosphorus or carbamate pesticide – a

treatment protocol for junior doctors. Critical Care. 2004;8(6):R391-R397.

6. Lacasaña M, López-Flores I, Rodríguez-Barranco M, Aguilar-Garduño C, Blanco-Muñoz J, Pérez-Méndez O, et al. Interaction between organophosphate pesticide exposure and PON1 activity on thyroid function. Toxicol Appl Pharmacol. 2010;249(1):16-24.

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