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Endocrine Consequences of Childhood Poisoning

Author(s)

Murat Doğan[1], Mustafa Armut[2], Selim Kurtoğlu[3]

Affiliation(s)

[1]Kayseri City Hospital Pediatric Emergency Department, Kayseri, Turkey
[2]Memorial Hospital Pediatri Department, Kayseri, Turkey
[3]Memorial Kayseri Hospital, Kayseri, Turkey

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Abstract

Poisoning is reported as a major problem in the childhood period especially under five years. Acute or chronic exposure to toxic material occurs as a different clinical picture. Some cases have different endocrinological findings. Early diagnosis and management are required for careful clinical evaluation. In this article, endocrinological problems of childhood poisoning are reviewed.

Keywords: Poisoning, children, endocrine findings

Introduction

Poisoning is among the important medical problems that occur during the prenatal and postnatal periods.[1-4] 6.2% of the cases admitted to the pediatric emergency department is intoxication.[2] Poisoning occurs in the form of oral, dermal, ocular, intravenous, biting, sting, and inhalation. It is most commonly seen under 5 years of age and in adolescence[2-4] and reported that 88.6% of the cases are occurred at home, 92.1% by accident, 7.9% for suicide.[2] Poisonings can cause a various acute or chronic toxicological problems. Some poisonings result in a variety of endocrinological problems. In this article, the endocrinologic consequences of childhood poisoning are reviewed.

Endocrinological Problems Observed in Poisoning

1. Blood Glucose Changes

Hyperglycaemia: Hyperglycemia can be observed as a laboratory finding in some intoxications such as salicylate, salbutamol, theophylline, carbon monoxide, acetone band acetylene, and organophosphate poisoning (**Table 1**).[5-13] It is observed in 40-48% of cases with organophosphate poisoning[6,7] which may present with a clinical picture of diabetic ketoacidosis and not be diagnosed at the beginning. Therefore, clinicians should carefully evaluate the patient's pupils, halitosis, and conjunctivas

Correspondence: Selim Kurtoğlu, Memorial Kayseri Hospital, Kayseri, Turkey
E-mail: selimchief@gmail.com

in terms of miotic pupils, the smell of garlic in the mouth and conjunctival ciliary injection. If the patient has those findings, serum pseudocholinesterase level should be measured.[9]

Hypoglycemia: An important laboratory finding in childhood poisoning is hypoglycemia (**Table 2**).[5-7,14,15] In some poisonings such as iron and organophosphate, hyperglycemia may be observed first, followed by hypoglycemia. The frequency of hypoglycemia in anticholinesterase poisoning was found between 6.4-7.1%.[6,7] Hypoglycemia can cause abstinence, sweating, unconsciousness, and convulsions, and in severe cases, it can lead to irreversible neurological problems.[14]

2. Serum Calcium Changes

Hypercalcemia: Vitamin A and D intoxication are the most important intoxications that cause hypercalcemia. Vitamin A poisoning occurs as a result of taking high doses of vitamin preparations or consuming foods such as the chicken liver. Acute poisoning develops when taken at a dose of 25000 units/kg, while chronic poisoning occurs after 6-15 months of use at a dose of 4000 units/kg per day. In acute cases, symptoms include vomiting, restlessness, and increased intracranial pressure. Pseudotumor cerebri, bone pain, subfebrile fever, hypertension, polyuria, and polydipsia are observed in chronic poisoning cases.[5] Vitamin D intoxication results from an acute overdose, usually as a result of an overdose of vitamin preparations or fish oil consumption. Clinically, symptoms of pseudotumor cerebri, hypercalciuria, polyuria, and polydipsia are observed. The serum 25-OHD level was higher than 100 ng/ml.[16]

Hypocalcemia: Poisoning causing acute or chronic hypocalcemia is listed in **Table 3**. Clinically, patients present with tremors, jitteriness and convulsion. Besides, hyperphosphatemia may be observed.[5,17,18]

Tablo 1.
Poisoning causing hyperglycemia

• Salicylate	• Difenbahia
• Sodium phosphate enema	• Camel base
• Magnesium hydroxide	• Paramethoxy amphetamine
• Ethylene glycol	• Hydrofluoric acid
• Iskin	• Fluorine
• Colchicine	

Tablo 2.
Poisoning causing hypoglycemia

• Salicylate	
• Iron	• Ethyl Alcohol
• Insulin Metformin	• Venlafaxine
• Sulfonylurea group	• Citalopram
• Diazoxide	• Class 1 antiarrhythmics
• Valproic acid	• Mushroom
• Phenytoin	• Snake bite
• Opioids	• Paramethoxamphetamine
• Cocaine	

Highlight

- Poisoning is an accident occured unintentionally or deliberately at all ages, especially childhood. It can cause a several acute or chronic toxicological problems in human body.
- Acute and chronic endocrinological problems may be appeared in children presenting with poisoning based on the resource of toxic material.
- Therefore, acquiring a clinical history and closely monitorizaiton is required to prevent or decrease endocrinologic abnormalities in poisoned children.

3. Intoxications Affecting the Thyroid Gland

Thyrotoxicosis: Especially thyroxine preparations and iodine-containing drugs and amiodarone may cause thyrotoxicosis.[5,19-21] In the neonates, iodine overload may rarely cause hyperthyroidism.[22] Also, it has been reported that thyrotoxicosis may occur with tetrodotoxin, salicylate, and long term lithium use.[5]

Goiter and Hypothyroidism: Some drugs and chemicals can result in the development of goiter and hypothyroidism.[23-25] Excessive iodine intake during pregnancy, breastfeeding, neonatal period, and childhood can cause goiter and hypothyroidism. Use of antithyroid drugs, cyanide, florurethionamide, phenylbutazone, sulfamides, cobalt, epdantoin, aminoglutethimide, lithium in all ages may cause goiter and hypothyroidism.[25]

4. Drugs Poisoning with Antidiuretic Hormone (ADH) Release or ADH-Like Effect Chlorpropamide, vinblastine, tricyclic antidepressants, vincristine and carbamazepine derivatives may trigger water retention and lead to hyponatremia.[5]

Table 3.
Poisoning causing hypocalcemia

• SalicylateMethadone	• Teofillin
• Calcium channel blockers	• Acetone
• Endosulfan	• Carbon monoxide
• Organophosphates	• Salbutamol
• Phenylbutazone	• Acetylene
• Amitraz Phenytoin	• İsoniazid
• CyanideIron	• Aluminum phosphite

5. Intoxications Affecting Surrenal Gland Functions Some drugs and toxic substances may be a reason for surrenal insufficiency. Especially, the mother's exposure to steroids results in the life-threatening adrenal suppression in the fetus.[26] Oral or dermal application of corticoids in the postnatal period may lead to iatrogenic Cushing.[27] Hydrocortisone and prednisolone can be broken down in the placenta and 10% of prednisolone, 33% of betamethasone and 50% of dexamethasone cross the placenta. If prednisolone is used for a long time, the placental enzyme is saturated and prednisolone passes easily which can cause steroid toxicity.[26] The medications interfering with adrenal steroid synthesis such as Ketoconazole, fluconazole, etomidate, aminoglutethimide, trilostane may lead to adrenal suppression. Epdantoin, phenobarbital, rifampicin, topiramate and thyroxine may result in a marked increase in the rate of catabolism of cortisol. Mifepristone, chlorpromazine and imipramine can cause blockage in the glucocorticoid receptor.[28]

6. Endocrine Problems in Metal Poisoning

Lead poisoning is the most common heavy metal poisoning that causes endocrine disorders. Atabek et al. investigated the effect of cord blood lead level on the serum IGF-1 and neonatal anthropometric measurements.[29] They showed that the cord blood level above 10 micrograms/dl negatively affects birth weight without any effect on the IGF-1 level. Lead poisoning has been observed as a cause of important endocrine problems in person working in lead factories.[30] Lead accumulation in endocrine organs is thought to impair the cell functions. It has been demonstrated that lead poisoning affects the hypothalamus-pituitary axis, blunting the TRH, GNRH and GnRH response of the pituitary, but increasing the prolactin level. Although short-term exposure to lead can cause increased FSH and LH levels, testosterone level remains normal. However, in chronic intoxication, the hypothalamo-pituitary axis is disrupted. Lead accumulates in ovarian granulosa cells and negatively affects pubertal development and fertility. Lead may accumulate in seminiferous tubulus cells in men and negatively affect sperm count and motility. It decreases the level of stress-induced corticosterone by accumulation in the adrenal gland, and affects cytosolic and nucleer corticoid receptor binding. Besides its central effect on the thyroid gland, it may impair the function of binding proteins. It may also affect calcitriol levels and reduce calcium absorption from the intestines.[29,30]

Conclusion

Endocrine functions may be impaired as a result of intoxication in children. Patients presenting with poisoning should be investigated in terms of acute and chronic endocrinological problems and should be followed closely.

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