COURSE AND TREATMENT OF HYPERPARATHYROID CRISIS IN CHILDREN SUFFERING FROM UROLITHIASIS

Hyperparathyroid crisis (HPC) in children suffering from urine stone disease is developed because of fast and acute raise of calcium content in blood. Diagnostics and treatment process of children with urine stone disease and further developed HPC was examined. The crisis developed due to a sharp increase in concentration of parathyroid hormone in blood serum. Due to successful diagnostics and treatment, two patents were cured, and, because of late entering and HPC complications with comatose state, the taken treatment was not successful.

**Keywords:** Urolithiasis, hyperathyroidism, children.

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**Introduction**

Hyperparathyroid crisis (HPC) is a serious, life-threatening condition. It develops as a result of the rapid and sharp increase in levels of parathyroid hormone in blood. Parathyroid hormone, affecting bone resorption, renal reabsorption and intestinal absorption increases the level of calcium in blood (Popovtzer et al., 1997; Wong et al., 2001). Acute course, early complications and difficulties in diagnosing hyper parathyroid in children with urolithiasis determined the relevance of finding new methods of diagnosis and treatment (Bielec et al., 2005; Harjit et al., 2007).

In this article we develop some comments on the clinical course and treatment of hyper parathyroid crisis in children with urolithiasis.

**Material and methods**

Of 305 children with urolithiasis, in 82 was observed acute calculus occlusion of the urinary system. These children needed urgent restoration of urodynamics. In the early postoperative period after pielo-and-ureterolithotomy in 3 (5.6%) children hyperparathyroid crisis developed.

**Results and discussions**

One patient was hospitalized in the state of hyper parathyroid coma. Hyper parathyroid crisis manifested itself suddenly, with anxiety and pain behind the sternum and in epigastrium. Pain in joints, bones, femur and tibia were sustained. There was a sharp muscular weakness, drowsiness, impaired memory. Anxiety and pain were accompanied by indomitable vomiting (10-12 times), which was not connected with in-taking food, body temperature rose to 39-40 °C. Later the anxiety of patients slowed down, they became shock stilled or change of mental status, psychosis. Amid exsicosis decreased urine output. The pulse became more frequent - 140-150 beats per minute, heart sounds damp down, observed hypertension (up to 150/100 Hg.cmm). The ECG demonstrated reduction in the interval Q - T, arrhythmia and depression of segment ST.

The following patient was admitted to the hospital with a diagnosis of urolithiasis. Multiple stones in kidneys, uremia, oliguria. Acute renal failure in a state of hyperparathyroid coma (level of serum calcium 5.0 mmol/liter). On examination the child inhibited (soporous state), does not contact, does not react to the examination, blood pressure 70/30 mm Hg, tachycardia with a violation of rhythm (beats) of the heart, moderate leukocytosis (10.5x10⁹), elevated ESR (19 mm/h). Levels of urea (16.9 mmol/liter) and creatinine (220.3 mkmol/liter) gradually raised. Content of general calcium raised in two patients to 4.5 mmol/liter (norm is 2.26 mmol/liter), in patient with hyperparathyroid coma it raised up to 5.0 mmol/liter. The level of parathyroid hormone
(PTH) raised up to 181 pg/ml (norm is 65.15 pg/ml), alkaline phosphatase activity raised to 4.5 mmol/liter (norm is 0.82 mmol/liter). Potassium, sodium, chlorine, general protein and its fractions were in terms of normal values.

In order to reduce calcium intoxication, the patients underwent intensive medical therapy: glomerular excretion of calcium was increased by using of 0,9% sodium chloride solution (150-300 ml/h/v) under the control of central venous pressure, furosemide (20-40 mg / IV) under the control of diuresis and levels of electrolytes (K, Mg, PO₄) in the blood.

Forced diuresis was taken before the reduction of calcium level in blood. In order to reduce calcium absorption in the intestine and increasing its excretion in urine was used prednisolone (30 mg per day, IM), as a calcium antagonist was used a solution of magnesium sulfate (20% solution of 5-10 ml, IV). To reduce calcium release and to improve its fixation in bones calcitonin was administered (4 units per day in 300 ml of isotonic solution NaCl). The content of total calcium was determined every 2-3 hours.

Conservative therapy provided short-term effect, which forced to think about the possibility of re-development of hyper parathyroid crisis, which defined indications for dialysis.). After the removal of patients from the state of hyper parathyroid crisis parathyroidectomy surgery was urgently performed. Deleted parathyroid adenomatous glands were changed up to 2 cm. Postoperative course was smooth, calcium level and PTH normalized during the first nights after parathyroidectomy; urea, creatinine and alkaline phosphatase content gradually reduced; and by the end of the second night were normal. The patient admitted to the hospital in the state of hyper parathyroid coma, despite the treatment with dialysis, could not be brought out of coma. The patient died. At post-mortem examination was revealed a solitary adenoma of parathyroid gland of the size 2.5 cm

Thus, timely diagnosis and implementation of adequate therapy with dialysis can successfully remove patients from the state of HPC. Radical method of treatment is parathyroidectomy. At late admittance of patients and comatose complication of HPC considerably reduce success of conservative therapy.

References


