

INSULINOMA: DIAGNOSTIC APPROACHES AND MEDICAL TACTICS

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INTRODUCTION

Insulinoma is the most common functioning pancreatic neuroendocrine tumor of β -cell origin, characterized by uncontrolled insulin production and in rare cases associated with MEN-1 syndrome. Insulinoma is a hormonally active tumor of β -cells of the islets of Langerhans, leading to the development of hypoglycemic attacks. In 1924, for the first time, the American doctor Seal Harris and the domestic surgeon V.A. Oppel simultaneously and independently described the syndrome of hyperinsulinism [1]. In the literature, there are different terms that imply an insulin-producing tumor: insulinoma, hypoglycemic disease, organic hypoglycemia, hyperinsulinism, insulin-secreting insulinoma. However, the term “insulinoma” is now generally accepted.

Relevance.

Insulinomas are rare: 1–4 cases per 1 million population per year in people 30–60 years old, up to 90% of cases are benign. This formation can be either sporadic (90%) or occur as part of the syndrome of multiple endocrine neoplasia type 1 (MEN-1) (10%), Wermer syndrome - hormonally active tumors of the parathyroid glands, adenohypophysis and tumors of the adrenal cortex (often hormonally inactive) [2]. Insulinoma is predominantly localized in the pancreas, but there are known cases of location outside the pancreas (splenic hilum, liver, duodenal wall, stomach wall, omentum, etc.) [3]. Hyperinsulinism can be both organic (insulinoma, adenomatosis, nesidioblastosis) and functional - as a result of fasting, surgical interventions on the stomach, the development of dumping syndrome, severe liver dysfunction, uncompensated hypothyroidism, uncompensated adrenal insufficiency, the initial stage of development of diabetes mellitus 2- type, autonomic dysfunction, etc. The clinical picture is caused by the release of contra-insular hormones (norepinephrine, glucagon, cortisol, somatotrophic hormone) in response to hypoglycemia, which develops as a result of uncontrolled secretion of insulin by pancreatic β -cells. Adrenergic symptoms

(tremor, anxiety, palpitations, nightmares) due to chronic hypoglycemia are replaced by glycopenic symptoms: weakness, drowsiness, dizziness, convulsions, decreased attention, impaired coordination of movements, neuropsychic disorders (hallucinations), symptoms of damage to the peripheral nervous system, etc. Prolonged and insufficient energy supply to the brain is initially accompanied by functional neurological disorders, and subsequently hypoglycemia leads to irreversible dystrophic changes in the central nervous system, making it difficult to verify the diagnosis in clinical practice. The diagnostic sign of insulinoma is Whipple's triad, which includes clinical manifestations of hypoglycemia on an empty stomach, a decrease in blood glucose level of 2.7 mmol/l or after 72 hours. If the patient has no symptoms of hypoglycemia and a decrease in glycemia within 72 hours.

PURPOSE OF THE STUDY

The purpose of the study was, based on a retrospective analysis, to evaluate diagnostic approaches and treatment tactics in patients with insulinomas.

RESEARCH METHODS

An analysis of the medical history data of 72 patients admitted with suspected organic hyperinsulinism (2022-2023) was carried out. The age of the patients ranged from 20 to 79 years. To confirm endogenous hyperinsulinism, patients underwent: a 72-hour fasting test, assessing the level of glucose, insulin, blood c-peptide, ultrasound and/or MSCT/MRI of the abdominal organs. Statistical data processing was performed using the Statistica 10.0 program

RESULTS

the diagnosis of organic hyperinsulinism was confirmed in 32 patients. Only 1 patient had insulinoma within the framework of MEN type 1 and was combined with the presence of primary hyperparathyroidism and a hormonally inactive pituitary microadenoma. When conducting a test with a 72-hour fast, hypoglycemia was achieved in 100% of cases within the first 48 hours from the start of the test. Analysis of the examination results in 50% of cases revealed the size of the pancreatic mass to be more than 1.4 cm. An inverse correlation was established between the size of the mass and the level of blood plasma glucose when hypoglycemia was achieved ($r=-0.45$, $p=0.02$). Thirty of the 32 patients underwent surgical treatment: enucleation of insulinoma was performed in 40% of cases, distal pancreatectomy in 60%. In 27 patients, the presence of insulinoma was confirmed, in three, according to histological examination, a diagnosis of "congenital organic hyperinsulinism" was established. In the early postoperative period, patients were discharged from the hospital on days 11-30. Only 12 (40%) patients had postoperative complications; in other cases, the postoperative period was uneventful and the duration of hospitalization was 13.0 ± 1.4 days ($p < 0.01$).

CONCLUSION

The data from the study confirm that for the successful management (diagnosis and treatment) of patients with endogenous hyperinsulinism, an integrated team approach is required, including a fasting test, the use of modern imaging methods and the use of high-tech treatment methods.

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