PSIXONEVROLOGIK BUZILISHLAR BILAN NAMOYON BO'LGAN SHMIDT SINDROMI: KLINIK HOLAT

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Kirish: Shmidt sindromi - autoimmun poliglandulyar sindrom II turi (APS II) ning eng ko'p uchraydigan shakli bo'lib, buyrak usti bezi va qalqonsimon bezning autoimmun zararlanishi bilan namoyon bo'ladi. Bemorlarda kasallikning to'liq korreksiya etilmasligi va turli psixonevrologik buzilishlarning vujudga kelishi orasida o'zaro bog'liqlik bo'lishi mumkin.

Maqsad: Shmidt sindromida dismetabolik psixoz mavjud bemorning klinik tahlili.

Klinik holat tavsifi: 1991-yilda tug'ilgan ayol umumiy holsizlik, ko'ngil aynishi, 3-5 martagacha qayt qilish, qon bosimining pasayishi, sho'r mahsulotlarga ehtiyojning ortishi, uyqusizlik hamda ruhiyatidagi o`zgarish (sababsiz yig'lash, tez jahl chiqishi, xavotir, suidsidal fikrlar, diqqatni jamlay olmaslik) shikoyatlari bilan RIEIATM ga murojaat qilib keldi. Anamnezida bemorda shikoyatlar 2013-yilda paydo bo'lishni boshlagan, 2015-yilda birlamchi surunkali buyrak usti bezi yetishmovchiligi va 2018-yilda autoimmun tireoidit tashxislari qayd etilgan bo'lib, doimiy ravishda gidrokortizon 30 mg/sutkasiga va levotiroksin 100 mkg/sutkasiga qabul qilib kelgan. Lekin so'nggi vaqtlarda gidrokortizon preparatini topa olmaganligi sababli, oxirgi 6 oy davomida prednizolon 10 mg dan noregulyar ravishda qabul qilgan. Klinik va laborator belgilar asosida bemorda adrenal kriz rivojlanish xavfi mavjudligi tahmin qilindi.

Natijalar: Obyektiv ko'rikda bemorning umumiy holati o'rta og'ir, xushi o'zida, teri qoplamining quruqligi, qo'l-oyoq uchlarining sovuqligi va ilgari kuzatilmagan psixomotor qo'zg'alishlar aniqlandi. Arterial qon bosimi 50/40 mm.sim.ust., pulsi 90 ta/daqiqa. Teri giperpigmentatsiyalari aniqlanmadi. Laborator tekshiruvlarda ph 7,44 (N —7,35-7,45), giponatriemiya 104 mmol/l (N — 130-150mmol/l), giperkalemiya 7,2 mmol/l (N — 3.5-5.4 mmol/l), gipoxloremiya 76 mmol/l (N — 98-107 mmol/l), glyukoza 5,5 mmol/l (N — 3,5-6,1 mmol/l), ertalabki kortizol 2,45 mkg/dl (6,2–19,4 mkg/dl), ertalabki AKTG 15,1 pmol/l (N — 1.6-13.9 pmol/l), TTG (100 mkg levotiroksin fonida) 1,87 mME/L (N — 0,3-4,0 mME/L), erkin T4 1,69 ng/dl (N — 0,93-2,0 ng/dl) aniqlandi. MRT tekshiruvi: gipofiz bezida patologik o'zgarishlar aniqlanmadi. Nevrolog xulosasi: dismetabolik ensefalopatiya. Psixiatr ko'rigi: emotsional beqarorlik, psixomotor qo'zg'alish, suidsidal fikrlar, vaziyatga nomutanosib affektiv reaksiyalar va o'tib ketuvchi shaxsga nisbatan dezorientatsiya asosida, boshqa kasallikka bog'liq organik psixotik buzilish (F06.8) tashxisi qo'yildi. Klinik tashxis: Autoimmun poliglandulyar sindrom II. Birlamchi surunkali buyrak usti bezi yetishmovchiligi. Autoimmun tireoidit. Medikamentoz kompensirlangan eutireoz. Bemorga gidrokortizon atsetat 50 mgdan har 6 soatda mushak orasiga dastlabki 3 kun davomida hamda infuzion terapiya o'tkazilishi fonida umumiy va ruhiy holati yaxshilandi, gemodinamik ko'rsatkichlari (qon bosimi 120/70 mm.sim.ust., pulsi 86 ta/daqiqa) hamda elektrolit muvozanati (Kaliy 4,0 mmol/l, Natriy 132 mmol/l, Xloridlar 81 mmol/l, Glyukoza 6,4 mmol/l) barqarorlashdi.

Xulosa: Shmidt sindromida o'rin bosar gormonal terapiyaga to'liq rioya qilmaslik adrenal kriz, og'ir elektrolitlar disbalansi hamda neyropsixiatrik buzilishlar rivojlanishiga olib keladi.

SCHMIDT SYNDROME MANIFESTING WITH PSYCHONEUROLOGICAL DISORDERS: CLINICAL CASE

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Abstract:

Primary chronic adrenal insufficiency is a severe endocrine disorder characterized by deficiency of glucocorticoids and mineralocorticoids, which leads to significant metabolic, cardiovascular, and neuropsychiatric disturbances. We present a clinical case of a 35-year-old female patient with a long-standing history of primary adrenal insufficiency who was admitted with symptoms of acute decompensation due to interruption of hormone replacement therapy. The patient developed an adrenal crisis manifested by severe hypotension, recurrent vomiting, marked electrolyte imbalance (hyponatremia, hyperkalemia), and significantly reduced serum cortisol levels. Alongside somatic deterioration, pronounced neuropsychiatric manifestations, including psychomotor agitation and dysmetabolic encephalopathy, were observed.

Comprehensive laboratory and instrumental evaluation confirmed the diagnosis of adrenal crisis without structural pituitary abnormalities. Intensive therapy with glucocorticoids, mineralocorticoids, and infusion correction of electrolyte disturbances resulted in stabilization of hemodynamic parameters, normalization of biochemical markers, and regression of neuropsychiatric symptoms. This case highlights the critical role of continuous hormone replacement therapy in preventing life-threatening adrenal crises and emphasizes the close interaction between endocrine dysfunction and neuropsychiatric manifestations.

Key words:
Primary adrenal insufficiency, Adrenal crisis, cortisol deficiency, hyponatremia, hyperkalemia,

psychomotor agitation, dysmetabolic encephalopathy, hormone replacement therapy, endocrine emergency, neuropsychiatric disorders.