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CLINICAL INFORMATION

Syndrome of inappropriate antidiuretic hormone secretion related to Guillain–Barré syndrome after laparoscopic cholecystectomy

Mensure Yılmaz Çakırgöz[a], Esra Duran[b], Cem Topuz[a], Deniz Kara[a], Namigar Turgut[a], Ülkü Aygen Türkmen[a], Bülent Turanç[c], Mustafa Önder Dolap[d], Volkan Hancı[e,*]

[a] *Department of Anesthesiology and Reanimation, Okmeydani Training and Research Hospital, Istanbul, Turkey*
[b] *Şehit Kamil State Hospital, Gaziantep, Turkey*
[c] *Department of Anesthesiology and Reanimation, Çorlu Vatan Private Hospital, Tekirdağ, Turkey*
[d] *Department of General Surgery Çorlu Vatan Private Hospital, Tekirdağ, Turkey*
[e] *Department of Anesthesiology and Reanimation, School of Medicine, Dokuz Eylül University, Konak, Turkey*

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Abstract
Background and objectives: Guillain–Barré Syndrome is one of the most common causes of acute polyneuropathy in adults. Recently, the occurrence of Guillain–Barré Syndrome after major and minor surgical operations has been increasingly debated. In Guillain–Barré syndrome, syndrome of inappropriate antidiuretic hormone secretion and dysautonomy are generally observed after maximal motor deficit.
Case report: A 44-year-old male patient underwent a laparoscopic cholecystectomy for acute cholecystitis. After the development of a severe headache, nausea, diplopia, and attacks of hypertension in the early postoperative period, a computer tomography of the brain was normal. Laboratory tests revealed hyponatremia linked to syndrome of inappropriate antidiuretic hormone secretion, the patient's fluids were restricted, and furosemide and 3% NaCl treatment was initiated. On the second day postoperative, the patient developed numbness moving upward from the hands and feet, loss of strength, difficulty swallowing and respiratory distress. Guillain–Barré syndrome was suspected, and the patient was moved to intensive care. Cerebrospinal fluid examination showed 320 mg/dL protein, and acute motor-sensorial axonal neuropathy was identified by electromyelography. Guillain–Barré syndrome was diagnosed, and intravenous immune globulin treatment (0.4 g/kg/day, 5 days) was initiated. After 10 days in the intensive care unit, at which the respiratory, hemodynamic, neurologic and laboratory results returned to normal, the patient was transferred to the neurology service.
Conclusions: Our case report indicates that although syndrome of inappropriate antidiuretic hormone secretion and autonomic dysfunction are rarely the initial characteristics of

* Corresponding author.
E-mail: vhanci@gmail.com (V. Hancı).

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Guillain–Barré syndrome, the possibility of postoperative syndrome of inappropriate antidiuretic hormone secretion should be kept in mind. The presence of secondary hyponatremia in this type of clinical presentation may delay diagnosis.

Introduction

The prevalence of hyponatremia in the postoperative period is 4.4%.[1] Syndrome of inappropriate antidiuretic hormone secretion (SIADH) develops due to the insufficient inhibition of antidiuretic hormone secretion linked to plasma hypotonia. Hyponatremia after surgical intervention is a well-known cause.[2] In case studies in the literature, SIADH-linked acute hyponatremia develops in the first few days after surgical intervention, may cause death and is related to general surgical stress and/or pain response.[2–5] Only one case related to Guillain–Barré syndrome (GBS) has been cited.[6]

In GBS, SIADH and dysautonomy are generally observed after maximal motor deficit.[7–9] In this case report, the rare clinical progression of GBS is reported in a dysautonomous patient, causing SIADH-linked serious hyponatremia in the early postoperative period before definite weakness and areflexia after a laparoscopic cholecystectomy.

Case report

A 44-year-old male patient presented to the emergency service with a complaint of abdominal pain. On physical examination, sensitivity in the left subcostal region, rebound and muscle rigidity were diagnosed, and the 'Murphy' sign was positive. An abdominal ultrasound showed thickening of the bile duct wall, dilatation of the biliary tract and high values of Alanin aminotransferaz (ALT) (145 (0–45) IU/L) and Gama glutamil transferaz (GGT) (73 (0–50) IU/L). The patient was admitted to the surgical service with a diagnosis of acute cholecystitis. Preoperative examination showed that the patient had been monitored for ulcerative colitis (UC) for the previous ten years, used no medications and had no previous operative history. Except for the preoperative physical examination and high ALT and GGT values from the laboratory examination, the patient had no other symptoms. After 8 h of fasting, the 85 kg ASA II-E risk group patient was given 3 mg IM dormicum premedication and taken to the operating table. His electrocardiograph (ECG), non-invasive blood pressure, end-tidal CO_2 (et CO_2) and SpO_2 were monitored. Anesthesia was induced with intravenous (iv) propofol (2.5 mg/kg), fentanyl (1 μg/kg), and rocuronium (0.6 mg/kg), and the patient was subsequently intubated. Anesthesia was maintained with 1–2% sevoflurane and 60–40% N_2O–O_2. During the laparoscopic cholecystectomy, his hemodynamic values and oxygen saturation were stable, and supplementary muscle relaxants and iv anesthetic agents were not administered. During the nearly 50-min operation, 8 mL/kg/h Isolyte-S was given, and 200 cc urine was passed. Our patient was monitored in the recovery room and moved to the relevant service before postoperative complications arose.

At 12 h postoperative, the patient developed a severe headache, nausea, lethargy, diplopia, urine retention, and attacks of hypertension; however, computed tomography (CT) of the brain was normal. Laboratory tests provided the following results: Na: 120 mmol/L (135–148 mmol/L); K: 3.8 mmol/L (3.5–5.1 mmol/L); Cl: 98 mmol/L (101–109 mmol/L); uric acid: 1.9 mg/dL (3.5–7.2 mg/dL); Blood urea nitrogen (BUN): 20 mg/dL (17–43 mg/dL); creatinine: 0.5 mg/dL; and White blood cells (WBC): $24.0 \times 10^3/mm^3$. His plasma osmolarity was 240 mOsm/kg, urine osmolarity was 515 mOsm/kg and urinary sodium was 89 mmol/L. Adrenal functions (Adrenocorticotropes hormon (ACTH): 30.6 pg/mL [0–46]; cortisol: 20.02 μg/dL [6.7–22.6]; Antidiuretic hormon (ADH): 4.9 pmol/L; aldosterone: 20.0 pg/mL; and aldosterone/renin ratio: 2.38), thyroid function tests, vitamin B12 and folate values were normal. In light of these results, the patient was diagnosed with SIADH, fluid restrictions were applied, and fusemide and 3% NaCl treatment was initiated.

On the second day postoperative, the patient developed back pain, numbness starting at the hands and feet and moving upwards from both extremities, loss of strength, difficulty swallowing and respiratory difficulty. After evaluation by the neurology unit, a diagnosis of GBS was made, and the patient was moved to the intensive care unit. Physical examination showed limited neck extension and back pain during neck movement. Knee, hip, and ankle movements were restricted due to pain. The sensitivity of joint regions increased on touch and during movement; however, there was no swelling or temperature increase in the joints. Neurologic examination showed good orientation and cooperation. Dysarthria, dysphagia and dysphonia were present. Stiff neck and other signs of meningeal irritation were not present. In addition, bilateral ptosis, isochoric pupil IR +/+, normal eye movement along the midline globe and no nystagmus were present. Hearing was normal. There was bilateral central facial paralysis. The upper and lower extremities had long socks, short gloves-style sensory defects. After becoming comfortable with opioid analgesics, examination showed a score of 5/5 on the 'Modified Research Council (MRC)' scale for muscle strength; the upper extremity proximal and distal muscles were 3/5, and the lower extremity proximal and distal muscles were 2/5. Deep tendon reflexes were hypoactive above, and no value was found below. There was no pathologic reflex. Fundus examination was normal. His temperature (axillary) was 36.6 °C, heart rate was 78/min, and respiration was 24/min. Arterial blood gas (ABGs) were pH: 7.48; $PaCO_2$: 36.9 mm Hg; PaO_2: 115 mm Hg; SaO_2: 96%; and HCO_3: 22 mmol/L. His respiratory difficulty was not bad enough to require a respirator.

A detailed history obtained from the patient's family indicated that nearly three weeks before the operation,

the patient had suffered from an upper respiratory tract infection. On the patient's first day in intensive care, a lumbar puncture was performed to obtain cerebrospinal fluid for biochemical, culture and viral serology investigations. Cerebrospinal fluid (CSF) examination showed normal pressure, clear liquid, no cells, protein of 320 mg/dL and all other parameters normal. There was no bacterial production from the CSF culture. On the patient's second day, an EMG was performed. Acute motor-sensorial axonal neuropathy (AMSAN) was observed in the upper and lower extremities on the electromyograph. A diagnosis of GBS was made, and treatment was initiated with intravenous immunoglobulins (0.4 g/kg/day, 5 days). Symptomatic treatment was given for hypertension attacks. Opioid analgesics were given to treat pain. Pain continued until the 10th day but decreased over time. The patient was monitored in the intensive care unit for 10 days. When hyponatremia and swallowing improved, respiratory difficulties resolved, hemodynamics were stable, upper extremity motor deficits resolved, and lower extremity surficial pain and heat sensitivity returned to normal, the patient was transferred to the neurology service. On final physical examination, his upper extremity distal and proximal muscle strength was 4 (MRC) and 3 (MRC) for lower extremities. Deep tendon reflexes were normal.

Discussion

SIADH was first diagnosed in 1957 by Schwartz et al., Bartter and Schwartz re-examined the condition in 1967 and listed the cardinal symptoms of SIADH as (a) plasma hypoosmolarity with similar hyponatremia, (b) plasma osmolarity higher than urinary osmolarity, (c) excessive renal excretion of sodium, (d) clinical absence of factors causing edema or volume depletion, and (e) normal renal and adrenal function. Currently, SIADH is known to be caused by many diseases, malignant and otherwise.[1]

Postoperative SIADH is reported to occur after major surgeries, such as abdominal and open heart operations, is less common after minor surgeries, and may also occur after cataract extraction under local anesthetic.[2–5] Nausea associated with increased serum ADH may be related to postoperative SIADH, and it may be part of the stress response to surgery and pain.[2,3]

GBS is the one of the most common causes of acute polyneuropathy in adults. The incidence is thought to be 1–2/100,000. It can occur at any age, and there is a slight predominance in males.[10] The disease begins in the lower extremities, and over the course of hours or days, it ascends, characterized by weakness in the arm and facial muscles. The majority of patients have a history of upper respiratory tract or gastrointestinal system infections in the 1–4 weeks prior to symptoms.[11] While the pathogenesis of GBS is unknown, it is accepted as a hypersensitive humoral and cellular immune response, generally attacking peripheral nerve system components.[10]

Recently, the occurrence of GBS after major and minor surgical operations has been increasingly debated. The relevant literature is limited to case reports.[10,12] There is no information on increased risk after certain operations. The majority of cases have a history of bacterial or viral infection, surgery, pregnancy, inflammatory bowel diseases (IBDs), connective tissue diseases, such as lupus, or other malignancies. These data indicate that GBS may be triggered by an unidentified pathogen or event affecting the immune system.[10]

Our patient developed postoperative GBS clinical features during UC remission. IBD may progress related to the patient's clinical course or can occur as completely independent extraintestinal symptoms. UC patients have been identified with both central and peripheral nerve system indications.[14] There are three case reports of patients with a history of UC developing GBS in the literature. Two of the patients were in remission, and one was in a relapse period.[13–15] In these cases, it was thought that GBS may be a symptom of extraintestinal UC.[13] Our patient had a history of upper respiratory tract infection three weeks prior. Combined with the trigger of surgical stress and considering the low incidence of both diseases and without other apparent causes of acute peripheral polyneuropathy, a foundation of an autoimmune disease such as UC may be responsible for changed immune function, causing GBS.

SIADH may develop during or after maximal motor deficit in 30% of GBS patients and generally 65% of dysautonomy cases.[7,9,12,16] Hoffmann et al.[7] noted an isolated GBS patient with no signs of dysautonomy or initial symptoms of SIADH. Our patient initially had normal strength and reflexes with initial symptoms of SIADH and dysautonomy, making him the second case with these clinical characteristics in the literature.[12] Visceral afferent fibers may be affected together with autonomic dysfunction and parasympathetic and sympathetic fibers, leading to sympathetic and parasympathetic insufficiency and hyperactivity linked to neuropathy. These factors combined with vascular tension receptors affecting peripheral autonomic fibers causing abnormal ADH secretion from the neurohypophysis reduce the effects of vagal inhibition.[12] However, without dysautonomy, the relationship between GBS and SIADH has not been clearly explained.

The pathogenesis of GBS-related SIADH is uncertain. Among the hypotheses, pathogenesis may be linked to changed osmoreceptor responses due to new lower threshold values in the osmoregulatory system, vasopressin-increased tubular sensitivity or ADH secretion affecting cardiac volume and afferent peripheral autonomic neuropathy of osmolarity receptors.[7,12] Recently, publications have proposed that a multifunctional cytokine, interleukin 6 (IL-6), may play a central role in the immunopathogenesis of SIADH linked to GBS.[17]

On examination, the back pain and distal sensorial symptoms experienced by our patient were initial signs of GBS; however, they were overshadowed by acute medical management problems of hyponatremia and dysautonomy. The patient's mental changes, secondary symptoms to hyponatremia, made it difficult to evaluate daily symptoms and delayed the definite diagnosis of weakness. In spite of the complications, late-developing areflexia was identified early, and the initial conservation of reflexes and normal strength showed the possibility of important peripheral neuromuscular dysfunction to be low.

Our case report indicates that although SIADH and autonomic dysfunction are rarely the initial characteristics of GBS, the possibility of causing postoperative SIADH should

be kept in mind. The presence of secondary hyponatremia in this type of clinical presentation may delay diagnosis. It should not be forgotten that early GBS diagnosis and appropriate treatment principles can lead to important treatment results in cases such as this.

Conflicts of interest

The authors declare no conflicts of interest.

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