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Case Report Olgu Sunumu

NEUROGENIC BLADDER INDUCED BY CHRONIC ALCOHOL ABUSE: A CASE REPORT

Kronik Alkol Kullanımına Bağlı Nörojenik Mesane: Olgu Sunumu

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ABSTRACT

Long-term alcohol consumption may produce wide-ranging effects on the autonomic and peripheral nervous systems like almost all body tissues, but the pathogenesis is still under debate. We presented an extremely rare case of autonomic neurogenic bladder with bilateral hydroureteronephrosis in a male patient describing progressive abdominal distension over a year that is most significant in the last 4 months following alcohol withdrawal after a long duration of alcohol abuse. Chronic alcoholism should be considered in the differential diagnosis of a neurogenic bladder, which may be caused by autonomic dysfunction due to chronic alcohol abuse and may be precipitated by alcohol withdrawal.

Keywords: *Alcohol, neurogenic bladder, autonomic neuropathy*

ÖZ

Uzun dönem alkol tüketimi otonomik ve peripheral sinir sistemlerinde, vücudun hemen hemen tüm dokularında olduğu gibi geniş spektrumda etkiler yaratır, ancak patogenezi hala belirsizliğini korumaktadır. Burada, uzun süreli alkol tüketimini takiben alkolü bıraktıktan sonraki bir yıl içinde gittikçe artan ve son 4 ayda belirginleşen karın şişliği tarifleyen bir erkek hastada bilateral hidroüreteronefroz gelişmiş oldukça nadir bir nörojenik mesane olgusu sunulmuştur. Kronik alkol kullanımı, alkolün bırakılmasıyla belirginleşebilen otonomik disfonksiyona bağlı nörojenik mesaneye sebep olabilir ve ayırıcı tanısında göz önünde bulundurulmalıdır.

Anahtar Kelimeler: *Alkol, nörojenik mesane, otonom nöropati*

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INTRODUCTION

Polyneuropathy is the most common neurological complication in alcoholism with a frequency ranging from 12.5 to 29.6% (1). Although there is only limited information concerning autonomic neuropathy in alcoholics, it is also known to be a common finding in these subjects (one quarter to one third of the alcoholics) (2,3). Herein we presented an extremely rare case diagnosed to have autonomic neurogenic bladder resulted in bilateral hydroureteronephrosis in whom there was no etiologic factor other than long term alcohol consumption that may account for the neurogenic bladder.

CASE REPORT

A 40-year-old man was admitted with complaints of abdominal pain and distension for about one year, aggravated in the last 4 months. Abdominal pain was accompanied by nausea, vomiting, regurgitation and pyrosis, especially in the morning and on exertion. He also described dysuria, polyuria, nocturia, pollakiuria and polydipsia for 5 months. He had no weight loss. He had a history of 15 pack-year cigarette smoking, and alcohol consumption over 10 years (700 ml/day Turkish beverage "Raki" containing 280 g alcohol) until the last four months. He had no drug usage and family history was unremarkable.

His vital signs were normal and general condition was quite good. Physical examination revealed abdominal distension with flattened umbilicus but no hepatosplenomegaly or ascites, anterior hypospadias and reduced vibration sensation in all extremities. Laboratory investigation shoved impaired renal function (Table 1).

Urinalysis was positive for leukocyte and culture revealed positive bacterial growth (*E. coli*>100.000 cfu/ml). Plain radiographies of chest and abdomen were normal. Abdominal ultrasonography showed huge cystic structures filling the whole abdomen which seem to be related with kidneys. Magnetic resonance urography revealed bilateral huge hydronephrosis with extremely thinned renal parenchyma and bilateral moderate dilatation and tortuosity of ureters, as well as bladder distension (Figure 1). Abdominal computed tomography did not show any mass lesion that may cause urinary obstruction. Patient was catheterized with an 18F Foley catheter and about 7.5 L urine was drained in 4 hours and ciprofloxacin was ordered for urinary infection. On urodynamic test, patient did not sense micturition until the capacity of 720 ml; maximal intravesical pressure was 11 cmH_2O and uninhibited contraction was not observed. In subsequent pressure-flow study, no micturition and no increase in detrusor pressure were observed. On cystoscopy, in spite of normal calibration of external meatus and no urethral obstruction, the bladder distension was confirmed. These findings were consistent with flaccid type neurogenic bladder. Lumbosacral and cranial magnetic resonance imaging was unremarkable in respect to possible causes of neurogenic bladder. There were no other clinical findings concerning vitamin B12 or thiamin deficiency. Serum ethanol level was undetectable. Serum vitamin B12, folate and thiamin levels were 403 pg/ml (200-700 pg/ml), 4.08 ng/ml (3-16 ng/ml) and 28.3 ng/ml (20-50 ng/ml), respectively. Erythrocyte transketolase activity for thiamin metabolism was 140.7 U/L (123.8-206.2 U/L). Although there were no findings regarding transverse myelitis in neurological and radiological examinations, investigations for possible vasculitis, collagenous diseases or bacterial or viral infections including viral hepatitis, syphilis, brucellosis, and tuberculosis, were all negative. Antinuclear antibody was negative and thyroid function tests were normal. Electroneuromyography of extremities showed sensorial axonal polyneuropathy and cardiac autonomic tests were found normal. In the absence of other causes, long term alcohol abuse was assumed to account for the neurogenic bladder and peripheral polyneuropathy in this patient.

After catheterization complete relief from abdominal distension and related symptoms was achieved, hydronephrosis and bladder distension significantly decreased on ultrasonography and renal functions

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improved moderately (Table 1). Patient was discharged from hospital on 25th day of admission with self-performed clear intermittent catheterization (CIC) every four hours. During about two years of follow-up his renal functions remained stable and magnetic resonance urography on the second year revealed significant improvement in hydroureteronephrosis but no complete recovery (Figure 2).

Table 1. Laboratory findings of the patient with alcohol induced neurogenic bladder at presentation and discharge

Parameter (normal ranges)	At presentation	At discharge (on 25th day of admission)
Hemoglobin (11-18 g/dl)	13.4	13.8
Hematocrit (35-60%)	43.2	45.5
MCV (80-99.9 fl)	84.4	87.2
Leukocyte (4.5-10.5 x10^3/mm^3)	9.5	7.1
Platelet (150-450 x10^3/mm^3)	239	305
ESR mm/h	100	40
FPG (70-110 mg/dl)	96	72
Sodium (134-150 mmol/L)	137	134
Potassium (3.5-5.5 mmol/L)	5	5
Urea (10-40 mg/dl)	141	60
Creatinine (0.4-1.2 mg/dl)	3.24	2
Creatinin clearance (>90 ml/min)	34	64
Uric acid (2.3-7.5 mg/dl)	7.4	7.2
AST (5-40 IU/ml)	17	17
ALT (5-40 IU/ml)	16	15
Total bilirubin (0-1.2 mg/dl)	0.60	0.42
Direct bilirubin (0-0.2 mg/dl)	0.13	0.08
ALP (65-300 IU/L)	146	122
LDH (220-450 U/L)	313	306
GGT (7-49 U/L)	45	43
CPK (0-170 U/L)	100	126
Total protein (6.4-8.7 mg/dl)	7.8	7.2
Albumin (3.2-5.2 mg/dl)	4.1	4.0
Calcium (8.4-10.4 mg/dl)	9.0	8.6
Phosphorus (2.6-4.5 mg/dl)	4.1	4.4

MCV: mean corpuscular volume, ESR: erythrocyte sedimentation rate, FPG: fasting plasma glucose, AST: aspartate transaminase, ALT: alanine aminotransferase, ALP: alkaline phosphatase, LDH: lactic dehydrogenase, GGT: gamma-glutamyltransferase, CPK: creatine phosphokinase

DISCUSSION

Neurogenic bladder in chronic alcoholism is reported extremely rare, only a few reports, in the literature (4-7). It was due to thiamin deficiency in one case and alcoholic myopathy in another (4,5). Two other cases were due to autonomic neuropathy presented with abdominal distension because of urinary retention, one with severe bilateral hydroureter, and the other with acute presentation soon after alcohol withdrawal which let the suggestion that neurogenic bladder due to alcoholic neuropathy could be precipitated by alcohol withdrawal (6,7).

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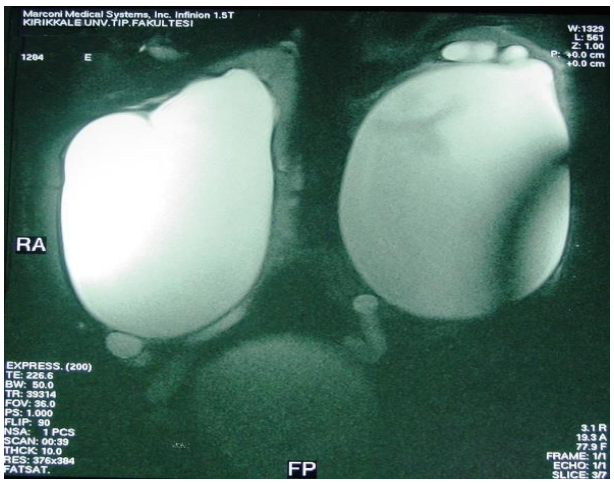

Figure 1. A coronal section of Magnetic Resonance Urography of the patient with chronic alcoholism obtained at presentation. Bilateral huge hydronephrosis with extremely thinned renal parenchyma and bilateral moderate dilatation and tortuosity of ureters as well as bladder distension

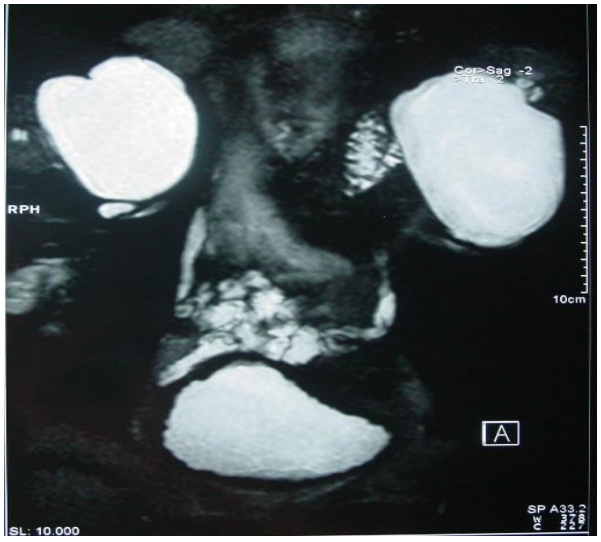

Figure 2. A coronal section of Magnetic Resonance Urography of the same patient obtained two years after the initiation of clean intermittent catheterization. Bilateral hydronephrosis significantly improved when compared to previous one.

The present case was also concluded to have autonomic neurogenic bladder due to chronic alcoholism as we could not find any other cause. Patient described progressive abdominal pain and distension that was precipitated by the withdrawal from alcohol after a long-term abuse which may support the suggestion that neurogenic bladder could be precipitated by the withdrawal of alcohol, but the mechanism needs to be clarified.

Thiamine deficiency frequently is associated to chronic alcoholism, therefore can obscure the clinical picture of alcoholic neuropathy. However, the observation of polyneuropathy in alcoholic subjects with normal thiamine status confirms the direct toxic effect of alcohol on peripheral nerves (8). Our patient was not undernourished, had no history of weight loss and we did not detect thiamine deficiency. Therefore, we concluded that the most probable cause of flaccid neurogenic bladder in this patient was the direct toxic effect of alcohol on autonomic nerves. The patient was also diagnosed electrophysiologically to have peripheral polyneuropathy which was also considered to be related with alcohol abuse.

Neurogenic bladder due to autonomic dysfunction may be caused by chronic alcohol abuse and may be precipitated by alcohol withdrawal. Therefore, chronic alcoholism should be considered in the differential diagnosis of a neurogenic bladder.

Conflict of Interest: All authors declare that there is no conflict of interest.

Researchers' Contribution Rate Statement: All authors made substantial contributions to the article. Idea-planning: HK, HB, FUT; analysis-interpretation: HK, HB, FUT; data acquisition: HK, BC; manuscript preparation HK, BC, HB and final manuscript approval HK.

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Informed Volunteer/Consent Form: Written informed consent was obtained from the patient for publication of this case report.

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