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NEPHROGENIC ASCITES CURED BY PERSISTENT ULTRAFILTRATION : CASE REPORT

PERSİSTAN ULTRAFİLTRASYON İLE TEDAVİ EDİLEN NEFROJENİK ASİT: OLGU SUNUMU

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SUMMARY

Treatment of nephrogenic ascites is a challenge. We report a patient with nephrogenic ascites successfully treated by persistent ultrafiltration.

Fifty-six year old male on hemodialysis presented with complaints of fatigue, loss of appetite and abdominal distension. The work up for etiology revealed the diagnosis of nephrogenic ascites. On diagnosis, salt restricted diet was prescribed and hemodialysis schedule was changed to 4 times a week, 5-6 hours per each vvith controlled ultrafiltration. Throughout 4 weeks of hospitalization, 10 liters of fluid was removed by means of paracentesis (2 liters) and ultrafiltration (8 liters) and his body weight decreased to 58 kg from 68 kg. Meanwhile his ascites regressed and his appetite and fatigue improved. Diameters of cardiac chambers, pulmonary artery pressure were decreased and cardiac valvular regurgitations were regressed. Although hemodialysis is to treat nephrogenic ascites, data reporting successful results are accumulating. As here, nephrogenic ascites can be cured by strict salt restriction, effective dialysis and persistent ultrafiltration in contrary to general belief. To increase the duration and frequency of hemodialysis and/or ultrafiltration other than to increase ultrafiltration rather, strict salt restriction and patience are the key issues in treatment.

ÖZET

Nefrojenik asit tedavisi güç olan bir klinik tablodur. Bu yazıda kararlı ultrafiltrasyonla tedavi edilmiş bir olgu sunulmuştur. 56 yaşında hemodiyaliz hastası halsizlik, iştahsızlık ve karında şişlik yakınması ile başvurdu. Yapılan incelemelerle olgu nefrojenik asit olarak kabul edildi. Sıkı tuz kısıtlaması ile beraber, diyaliz programı haftada 4 gün, 5-6 saat olarak değiştirildi. Kontrollü ultrafiltrasyon uygulandı. 4 haftalık hastanede yatış sırasında parasentez(2 litre) ve ultrafiltrasyon (8 litre) ile toplam 10 litre sıvı çekildi. Bu sırada hastanın vücut ağırlığı 68 kg'dan 58 kg'a indi, asiti azaldı, iştahsızlık ve halsizlik yakınması geriledi. Kalp boşluklarının çapları, pulmoner arter basıncı azaldı, kapak yetmezlikleri azaldı. Hemodiyalizin nefrojenik asit tedavisinde etkin olmadığı bildirilmesine karşın, son zamanlarda başarılı sonuçlar bildiren yayınlar artmaktadır. Hastamızda olduğu gibi, genel kanının aksine, nefrojenik asit sıkı tuz kısıtlaması, etkin diyaliz ve kararıı ultarfiltrasyon ile tedavi edilebilir. Ultrafiltrasyon hızını arttırmaktan ziyade hemodiyaliz/ultrafiltrasyon sayı ve/veya süresini arttırmak ve sıkı tuz kısıtlaması başarıda anahtar noktalardır.

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INTRODUCTION

Nephrogenic ascites is a condition characterized by the presence of massive ascites in a patient with end stage renal failure in the absence of any other expianation (1).

Its incidence is unknovvn. İt has high mortality rates. peritoneovenous shunt are reported in the literatüre. However renal transplantation is said to be the most effective treatment modality. Fluid removal by hemodialysis is reported to be ineffective for the ascites resolution in previous studies (2,3). On the other hand, there are data reporting that it can be cured by persistent ultrafiltration (1,4). Herein, we report a patient with nephrogenic ascites treated by persitent ultrafiltration.

CASE REPORT

Fifty-six year old male on hemodialysis presented with complaints of fatigue, loss of appetite and abdominal distension in February 2000. The etiology of renal failure was obscure. He has undergone coronary artery bypass grafting surgery and ventricular aneursymectomy in 1989 and 1999 respectively. On diagnosis of end stage renal failure during the preoperative evaluation of the latter operation, he has begun on hemodialysis in September 2000. During his work up for renal transplantation, he was diagnosed as having prostate adenocarcinoma and undergone transurethral resection followved by hormone theraphy and radiotherapy. He was on maintenance hemodialysis (high-flux, 3,5 hours, thrice per week) in the same center. Meanvvhile, he observed to developed abdominal distension and ascites. The vvork up for etiology did not reveal any diagnosis, and the patient has been considered to have nephrogenic ascites.

On admission to our center, his vital signs were as follovvs: blood pressure: 120/70 mmHg, pulse: 80/min rhythmic, respiratory count: 20/min, body temperature: 36,5 °C. Jugular venous distension, murmurs at mitral (3/6 pansystolic radiating to left axilla), aortic and tricuspid area (2-3/6, systolic), abdominal distension, and ascites were noted in physical examination. Organomegaly could not be assessed because of ascites. Ankle edema was not present. He was 68 kg in vveight and his abdominal circumference was 98 cm (figüre 1). Initial and follow up, BUN, creatinine, hematocrit serum albumin and cardiothoracic indexes in chest radiograms were presented in table 1. Initial chest x-ray disclosed cardiomegaly but no pleural effusion. İn echocardiography, especially on the right side, ali cardiac chambers were found to be dilated. Severe mitral, tricuspid and aortic regurgitation were noted. Hepatic veins were distended. Pulmonary artery pressure was 70-75 mmHg. Additionally, left ventricular diastolic and systolic functions were found to be disturbed. Ejection fraction was 40-50%. Ascitic fluid albumin and vvhite blood celi count were 2,7 g/dL and 155/mm^3 respectively. Serum-ascites albumin gradient was 0,5 g/dL. Cytological, bacteriologic and mycobacteriologic examinations of ascitic fluid did not reveal any diagnostic data. Portal dupplex sonography was not compatible with portal Successful resolution by means of peritoneal diaiysis and hypertension. Compiling ali the data, he was considered as nephrogenic ascites.

On diagnosis, salt restricted diet was prescribed and hemodialysis schedule was changed to 4 times per week, 5-6 hours per each with controlled ultrafiltration. Throughout 4 vveeks of hospitalization, 10 liters of fluid was removed by means of paracentesis (2 liters) and ultrafiltration (8 liters) and his body vveight decreased to 58 kg from 68 kg. Meanvvhile his ascites regressed (Figüre 2) and his appetite and fatigue improved as vvell as clinical (Table 1) and echocardiographic (Table 2) parameters throughout 4 months period.

Table 1. Course of clinical parameters

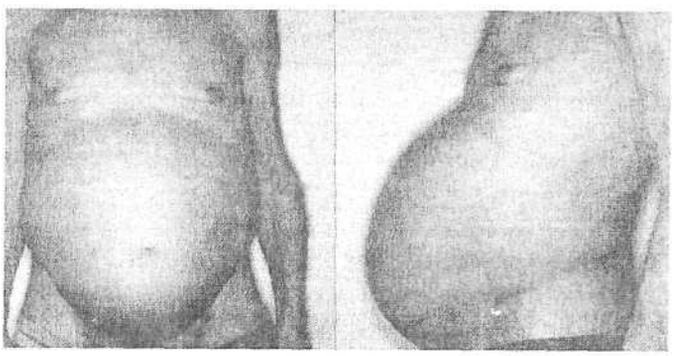
	Body Weight	Abdominal Circum ference (cm)	Hct (%)	Serum albumin (g/dL)	BUN (mg/dL)	Serum Creatinine (mg/dL)	CTI (%)
Baseline	68	98	24	3,2	39	7,7	0,47
1[a] month	58	90	29	3,6	61	9,3	0,45
fmonth	57	88	32	3,8	88	12	0,45
3[rd] month	56	86s	33	4	186	13	0,42
4* month	56	84	33	4,2	188	13	0,42

Hct: hematocrit, BUN: Blood urea nitrogen, CTI: cardiothoracic index.

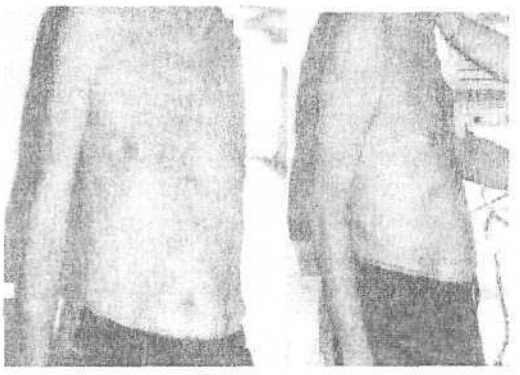
Table 2. The course of echocardiographic findings

	LVEDd (mm)	LA (mm)	EF	MR	TR	AR	PAP (mm Hg)
Baseline	60	54	45	4	4°	2°	70-75
1[st] month	60	52	45	4	4°	2°	60-65
2[nd] month	54	43	48	2	1°	1 °	25-30
4[th] month	50	39	50	2	1°	Minimal	20-25

LVEDd: Left ventricle end-diastolic diameter, LA: Left atrium diameter, MR: Mitral regurgitation, TR: Tricuspid regurgitation, AR: Aortic regurgitation, PAP: Pulmonary artery pressure, EF: Ejection fraction.



Figüre 1. Nephrogenic ascites in the patient before treatment



Figüre 2. The picture of the patient after resolution ot the ascites.

DISCUSSION

Nephrogenic ascites is an entity that manifests itself as refractory ascites in patients with end stage renal failure, where portal hypertension, infections and malignant processes per se have been excluded. Most of these patients are undergoing hemodialysis (1). Neither the exact cause nor the pathogenesis of the ascites formation is ciearly understood. Hovvever leaky peritoneum, disturbed lymphatic drainage of the peritoneal fluid, chronic volume overload with hepatic congestion are suggested in the pathogenesis. Heart failure and hypoalbuminemia may be contributing factors (5). Interdialytic weight gain of these patients are often excessive (6). Patients frequently present with hypertension, moderate to massive ascites, minimal ankle edema, cachexia and history of dialysis associated hypotension (2). High protein content, low serum-ascites albumin gradient and low leukocyte count are the general properties of the ascitic fluid (1).

Treatment of nephrogenic ascites is controversial. Gluck et al (3) has reported that continuous ambulatory peritoneal dialysis, peritonovenous shunt and renal transplantation appear to be effective in controlling ascites formation. Cintin et al (5) reported that strict fluid control, intensive hemodialysis, high protein diet, intravenous albumin infusion-, intraperitoneal steroid injection and paracentesis as well as implantation of peritoneal pump have ali been ineffective in the treatment. On the other hand, Han SG et al (1) reported that some hemodialysis patients with nephrogenic ascites were successfully treated by daily hemodialysis within 3 weeks time. Similarly Töz et al (4) reported that dilated cardiomyopathy and ascites in a 16 year old hemodialysis patient was completely resolved by persistent ultrafiltration in two months time. Conflicting results are probably due to ignorance of importance of salt restriction and ultrafiltration rate. To increase the duration and/or number of hemodialysis or ultrafiltration sessions may be more appropriate than to increase ultrafiltration rate. Inadherence to salt restriction and excessive weight gain in those patients generally results in excessive ultrafiltration efforts. As the amount of fluid dravvn increases, number of hypotension episodes increases ovving to inadequate fiil of the intravascular compartment, in tum necessitating fluid infusion or termination of dialysis. Poor cardiac status further augments hypotension episodes occurring during ultrafiitration. Additionally increased intravascular hyperosmolaiity, again due to delayed filling of intravascular compartment, causes increased thirst reflex and further volume overload. As a result increased number of hypotension episodes and intravascular hyperosmolaiity, both being due to excessive ultrafiltration rate, creates a vicious cycle and augments voiume overload. Therefore strict salt restriction and prevention of excessive weight gain are at least as important as persistent ultrafiltration in the treatment of nephrogenic ascites.

In our patient, the etiology of ascites was unclear. Hovvever the presence of ischemic cardiac disease, incompliance to the salt restriction and excessive interdialytic vveight gain seems to be most important factors. İn the present case, strict salt restriction, increased duration and frequency of hemodialysis and persistent ultrafiltration resulted in 10 kg decrease in dry weight and have resolution of the symptoms. At the end of 4^{th} month, the patient's ascites has completely resolved and his nutritional status has improved. Additionaily cardiac status of the patient improved as the volume overload was discarded. Diameters of cardiac chambers were decreased, mitral, tricuspid and aortic regurgitaions regressed and ejection fraction improved. Our findings were compatible with that of Cirit et al. They reported improvements in ali patients with mitral and tricuspid regurgitations, hovvever they observed improvements in only 1 out of 3 patients vvith aortic insufficiency (7).

As a conclusion, nephrogenic ascites can be cured by strict salt restriction, effective dialysis and persistent ultrafiltration in contrary to general belief. To increase the duration and frequency of hemodialysis and/or ultrafiltration rather than to increase ultrafiltration rate, strict salt restriction and patience are the key issues in treatment of nephrogenic ascites.

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