Mannitole bağlı akut böbrek yetmezliği: olgu sunumu

Mannitol-induced acute renal failure: case report

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Özet

Mannitol, akut oligürikrenal yetmezliğin önlenmesi ve tedavisi yanında akut beyin ödemi, kardiyovasküler cerrahi ve akut glokomda osmotikdiüretik olarak kullanılmaktadır. En önemli yan etkisi elektrolit imbalansı ve renal yetersizliktir. Literatürde mannitole bağlı akut böbrek yetmezliği ile ilgili olgu ve çalışmalar mevcuttur. Bu yazıda akut glokom nedeniyle mannitol tedavisi uygulanan 77 yaşında olguda gelişen akut böbrek yetmezliği sunulmaktadır.
Anahtar Kelimeler: Mannitol, akut böbrek yetmezliği
Kısa Türkçe başlık: Mannitole bağlı akut böbrek yetmezliği

Abstract

Mannitol is used for the prevention and treatment of acute oliguric renal failure and as osmotic diuretic in acute brain edema, cardiovascular surgery and acute glaucoma. Its most important side effect is electrolytic imbalance and renal failure. Articles and studies related with mannitol-induced acute renal failure were reported in literature. This study presents the acute renal failure that developed in a seventy-seven-year-old male patient after the mannitol treatment due to acute glaucoma.
Keywords: *Mannitol, acute renal failure*
Kısa İngilizce başlık: *Mannitol-induced acute renal failure*

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Introduction

Mannitol increases the osmotic pressure in intravenous applications and shows a diuretic effect. It is generally used for the prevention and treatment of acute oliguric renal failure and as osmotic diuretic in acute brain oedema, cardiovascular surgery and acute glaucoma. Its most important side effect is electrolytic imbalance and renal failure (1).This report presents the acute renal failure that developed in a case receiving the mannitol treatment due to acute glaucoma.

Case Report

The seventy-seven-year-old male patient applied with the complaints of fatigue, pain, and burning on urination. The physical examination showed that the tongue was dry, turgor tonus was decreased on the skin, TA: 110/70 mm/Hg, temperature: 37.2 C, and no feature was detected during examinations of the other systems. His history included a cerebrovascular accident, Type 2 diabetes mellitus (DM), hypertension and glaucoma diagnoses. 1 gram of metformin 2x1 and 3 mg of glimepiride 1x1 were used due to DM; and 16 mg of candesartan 1x1 and 10 mg of amlodipine 1x1 were used due to hypertension. According to the examinations conducted after the admission in the hospital, the determinations were as follows; urea: 88 mg/dl, cre: 2 mg/dl, Na: 144 mg/dl, K: 3.9 mg/dl. An ample amount of leukocyte was detected as a result of the complete urine examination. The patient was hospitalised in the internal diseases clinic, due to dehydratation, prerenal acute renal failure and urinary infection. Metformin and glimepiride treatment was interrupted, insulin aspart and insulin detemir treatment was started. Due to the dehydratation and prerenal acute renal failure, a paranteral support treatment was applied with 1000 cc 0.9% Isotonic NaCl and 1000 cc 5% Dextrose (16 U was tamponed with crystallised insulin). In urinary system ultrasonography, it was determined that the size, contours, parenchyma thickness and echogenicity of both kidneys are normal; no proteinuira was detected during the urinary examination. One day later, the determinations were as follows; urea: 48 mg/dl, creatinine: 1.4 mg/dl, Na: 136 mmol/L, K: 4.7 mmol/L, WBC: 12400/mm^3, neutrophile: 79.4%, eosinophile: 1.3%, CRP: 75.3. Serratialiguefaciens was detected in urinary culture that was taken due to the urinary system infection; 500 mg 2x1 bottle of cefepime was started in accordance with the culture antibiogram result (creatinine clearance:50 ml/min/1.73m2). There was no reproduction on the control urinary culture, which was taken 48 hours later. Following the determination of hyperemia on scleras, the patient was consulted to eye diseases department and evaluated as acute Glaucoma; 300 cc (1 gr/kg) of 20% mannitol solution was intravenously given every half hour. Since there was an increase on urinary, creatinine values of the patient, who did not use nephrotoxic drug, after the mannitol treatment, as well as the eosinophile (5.1%) increase and hyponatremia, the mannitol was interrupted, urea, creatinine and electrolyte were followed daily and the parentaral liquid treatment was regulated with a 0.9% of NaCl, in accordance with the urinary follow-up. Cefepime treatment was sustained. On the 3rd day of the follow-up, the urea and creatinine value respectively increased to 118-4.3 mg/dl and urinate of approximately 6000 cc occurred; no metabolic acidosis was detected on the blood gas. On the 12th day of the follow-up, while the eosinophile receded to 2.8%, urea and creatinine values respectively receded to 48-1.1 mg/dl. Table 1 illustrates renal functions and electrolytic values.

Table 1: Laboratory Parameters of the Case.

Day	Urea (N:6.6-48.5mg/dl)	Creatinine (N:0.7-1.3mg/dl)	Sodium (N:136-145mmol/L)	Potassium (N:3.5-5.1mmol/L)
Day 0*	48	1.4	136	4.7
Day 1	54	1.6	128	4.2
Day 2	94	3.2	129	5.1
Day 3	118	4.3	132	4.8
Day 4	114	3.4	139	4.6
Day 5	84	2	138	4.7
Day 7	63	1.5	137	5
Day 10	60	1.2	136	4.4
Day 12	48	1.1	139	5

AccordingtotheobjectivecausalityassessmentbytheNaranjoprobabilityscale (Naranjoscore of 5, Table 2), mannitol-inducedacuterenalfailurewasprobable.

Table2: Naranjoadversedrugreactionprobabilityscalecalculatedforourcasesummedup 5 points, indicating a probableassociationbetweenacuterenalfailureandmannitoltreatment.

Question	Response	Points
Are there previous conclusion reports on this reaction?	Yes	+1
Did the adverse event appear after the suspect drug was administered?	Yes	+2
Did the AR improve when the drug was discontinued or a specific antagonist was administered?	Yes	+1
Did the AR reappear when drug was readministered?	Don't know	0
Are there alternate causes [other than the drug] that could solely have caused the reaction?	Yes	-1
Did the reaction reappear when a placebo was given?	No	+1
Was the drug detected in the blood [or other fluids] in a concentration known to be toxic?	+1	0
Was the reaction more severe when the dose was increased, or less severe when the dose was decreased?	Don't know	0
Did the patient have a similar reaction to the same or similar drugs in any previous exposure?	Don't know	0
Was the adverse event confirmed by objective evidence?	Yes	+1
Total		5

Discussion

Mannitol is an osmotic diuretic agent, which is used for the prevention and treatment of acute oliguric renal failure as well as acute brain oedema, cardiovascular surgery and acute glaucoma. When mannitol is given intravenously, it forms the osmotic diuresis by increasing the osmolarity of the extracellular fluid (1). It increases the transition of water from tissues, including the brain, cerebrospinal fluid and eyes, to the plasm. By this way, it causes a decrease on the cerebral oedema, intraocular or intracranial pressure, cerebrospinal fluid volume and pressure. Mannitol increases the sodium excretion. Renal functions, urinary flow, serum sodium and potassium levels should be followed during the mannitol treatment. If there is a decrease in the urinary quantity, clinical condition of the patient should be reassessed and the mannitol treatment should be interrupted, when necessary. Fluid and electrolyte disorders are the most important side effects observed during the mannitol treatment (2-5).

Articles and studies related with mannitol-induced acute renal failure were reported in literature. Dehydratation, tubuloglomeruler feedback, osmotic damage, increase of vasoactive substances (renin, angiotensin-I and angiotensin-II) and vasoconstriction play a role in mannitol-induced acute renal failure (1, 2, 6, 7). There are also studies showing renal damages caused by the use of mannitol in anti-oedema treatment after cerebral trauma (8).

Mannitol-induced acute renal failure is a complex process. Comorbid factors (diabetes mellitus, hypertension, geriatric age, urinary tract infection), nephrotoxic drug use, volume depression can be confusing. In our case, the acute renal failure is thought to have developed due to the fact that he is at the geriatric age, has diabetes and started antibiotherapy as a result of the urinary system infection. However, mannitol-induced acute renal failure was considered to have developed in our case due to the fact that there is no kidney pathology in the urinary ultrasonography, no proteinuira in urinary examination, renal functions were corrupted after the mannitol treatment, eosinophile increased, and renal functions became normal even though the mannitol treatment was interrupted and other treatments were sustained. The fact that the patient had eosinophile made us consider the possibility of acute interstitial nephritis; but as well as the clinical and laboratory recovery without the application of steroid, the sudden recovery of the table right after the interruption of the Mannitol treatment and having no need for another treatment excluded this diagnosis.

CausalityassessmentwasperformedbytheNaranjoalgorithm. The adverse drug reaction is assigned to a probability category from the total score as follows: definite if the overall score is 9 or greater, probable for a score of 5-8, possible for 1-4 and doubtful if the score is 0. AccordingtotheobjectivecausalityassessmentbytheNaranjoprobabilityscale (Naranjoscore of 5, Table 2), mannitol-inducedacuterenalfailurewasprobable (9). Mannitol is a clinically and commonly used agent. In non-overdose usages, renal functions and electrolyte should be carefully followed, due to the acute renal failure that might develop as a result of the mannitol treatment.

Kaynaklar

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