Shunt Nephritis

Şant Nefriti

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Abstract: Embolization and surgical therapy were planned for a patient with a vein of Galen aneurysm. First, ventriculo-atrial (VA) shunt placement was performed due to the prevailing hydrocephalus, which was secondary to the aneurysm. After the second embolization, the patient was discharged. Eight days later he was referred to our emergency service with high fever and hematuria. The case was diagnosed as shunt nephritis in the light of laboratory and clinical findings. Provision of a specific therapy helped symptoms and clinical signs attenuate. This case is worth reporting because such complication of shunts is rarely seen.

Key Words: Hydrocephalus, shunt complications, glomerulonephritis

Özet: Galen veni anevrizması saptanan bir olguya embolizasyon ve cerrahi tedavi planlandı. Öncelikle, anevrizmaya bağlı olarak gelişen hidrosefaliye yönelik olarak vetrikulo-atrial (VA) shunt takılması işlemi yapıldı. İkinci embolizasyondan sonra olgu taburcu edildi. Sekiz gün sonra olgu acil servisimize yüksek ateş ve kanlı işeme yakınmaları ile başvurdu. Olguya klinik tablo ve laboratuvar bulguları ışığında shunt nefriti tanısı konuldu. Özgül tedavi uygulanması ile hastanın yakınmaları ve bulgular geriledi. Shunt'ların bu komplikasyonu nadir görüldüğü için bu olgu sunulmuştur.

Anahtar Sözcükler: Hidrosefali, shunt komplikasyonları, glomerülonefrit

INTRODUCTION

Shunt nephritis, which is rarely seen as a complication of shunts, most often occurs in patients with VA type shunts (1,4,16). The agent is usually a coagulase-negative Staphylococcus epidermidis (13,14). Shunt nephritis is thought to be secondary to immune complex formation and deposition in the glomerular basement membrane of the kidney after chronic bacteremia (1,4).

Because the clinical presentation is usually non-specific, the diagnosis is usually made by laboratory findings (1,4,8,13). With early diagnosis and treatment of these patients, irreversible renal damage can be prevented (13).

In this paper, a patient presenting with a glomerulonephritis following aventriculoatrial shunt placement is presented and discussed.

CASE REPORT

A 47-year-old man with a previously diagnosed vein of Galen aneurysm was admitted to our clinic in July 1992. He was alert, cooperative but dysarthric. His right lower cranial nerves were involved. He had quadriparesis, bilaterally positive Babinski sign, and positive Achilles clonus at the right.

His vein of Galen aneurysm had been detected in another clinic in 1986 after the recent onset of epilepsy and was treated conservatively. One year

later, he was referred to the clinic on a second epileptic attack. Acute hydrocephalus was discovered and a VA shunt was performed, which was revised six months afterwards.

Embolization was performed twice and the patient was discharged. Eight days later he was admitted to our emergency service with stupor, high fever, and hematuria. The origin of the fever was not obvious after the initial examination. Hypoalbuminemia, mild azotemia, decreased serum C3 level, (++) proteinuria, occult and microscopic hematuria, and hypochromic microcytic anemia were found on laboratory examination (Table I). His urine culture was

Table I: The Laboratory Results of the Patient

Parameter	Result	Normal Values
Hemoglobin	8.1gr/dl	14-18 gr/dl
Hematocrit	25.2 %	42-52 %
Iron	56.0 ug/dl	60-145ug/dl
Iron-binding capacity	435 ug/dl	270-410ug/dl
BUN	43.0 mg/dl	7-21 mg/dl
Creatinine	2.1 mg/dl	0.9-1.6mg/dl
Total protein	5.7 g/dl	6.0-8.4 g/dl
Albumin	2.4 g/dl	3.5-5.0 g/dl
Blood C3 level	51.0 mg/dl	80-120 mg/dl

negative. Shunt nephritis was entertained as a possible diagnosis and 2 cc cerebrospinal fluid (CSF) was taken from the shunt reservoir for laboratory examination. Biochemical findings of the CSF were: glucose 43 mg/dl, protein 240 mg/dl, pandy (++), chloride 650 ng/dl, and 180 leukocyte/mm3 in the sediment. CSF cultures grew coagulase-negative Staphylococcus epidermidis. The ultrasonography of kidneys showed kidney enlargement and increase in renal echogenity, which were consistent with glomerulonephritis.

After external drainage was carried out through the proximal part of the shunt, he was given Vancomycin 500 mg t.i.d. parenterally and 20 mg/day intraventricularly. On the third day of the therapy the CSF became sterile and on the sixth day, a ventriculo-peritoneal (VP) shunt was performed. The patient's family refused to consent to renal biopsy.

The patient is being followed up by our clinic and nephrology clinic, and on final examination, that is six months after his initial admission, he was observed to be alert, and cooperative but with dysarthria and quadriparesis. Laboratory findings were improved.

DISCUSSION

Shunt nephritis was first described by Black et al in 1965 (2). The incidence of ventricular shunt infection has been estimated to occur in 2% and 40% of shunt patients. Less than 100 cases of shunt nephritis have been reported previously (1,4,7,8,10, 11,13,16,23).

The pathogenesis seems similar to that with subacute bacterial endocarditis with the chronic bacteremia. In these infections, IgG and IgM antigen-antibody immune complexes are formed and deposited in the renal glomeruli. The complement system is activated with subsequent depletion of circulating complement factors C3 and C4. Thickening of the glomerular basement membrane, accompanied by mesangial cell proliferation, leads to nephrotic syndrome (1,4,8,22).

Coagulase - negative Staphylococcus epidermidis have been reported in over 70% of patients with shunt nephritis. Other organisms such as Staphylococcus aureus, Staphylococcus albus, Propionibacterium acnes, Micrococcus, Listeria monocytogenes, and Cryptococcus neoformans have also been described in cases of infected shunts (3,6,9,10,11,14,16,17,18,20,21).

Symptoms of shunt nephritis are similar to other immune complex diseases of the renal glomeruli. Macroscopic or microscopic hematuria (90%), febrile episodes (88%), anemia (85%), hepatosplenomegaly (55%), non-thrombocytopenic purpura (20%) and hypertension (15%) are seen (1,4,8,22,23). Similar symptoms were observed in our patient.

Laboratory findings are similar to those of nephrotic syndrome: proteinuria, hypoalbuminemia, microscopic or occult hematuria, nephrolithiasis, iron deficiency or normochromic normocytic anemia, mild azotemia and low serum C3 concentrations may be present (1,13,14,20).

CSF cultures are thought to be unreliable as a diagnostic aid by some authors however "shunt tap" is a perfect tool to check for the presence of infection (12,15,16). Renal biopsy in shunt nephritis patient shows the characteristic endothelial proliferation with mesangial expansion and proliferation of

cellular elements, enlarged glomeruli and immuncomplex fixation of the glomerular basement membrane (1,2,13,19).

The three main goals in the treatment of shunt infections are clearing up the infection, maintaining a functioning device if still needed, and minimizing mortality and morbidity (15). Removal of the distal part of the shunt, external drainage of CSF, intraventricular antibiotic administration into the shunt reservoir and systemic antibiotic therapy are the first mode of treatment in patients with shunt nephritis (5,7,10,15). If CSF sterilization can be achieved by this mode, the shunt should be replaced (7,15,19,23).

If the patient with a central nervous system device has a fever of unknown etiology accompanied by nonspecific renal symptoms, shunt nephritis should be considered. Early diagnosis and treatment can prevent irreversible renal damage in these patients (8,13,15).

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