CONSEQUENCES OF DELAYED TREATMENT IN SHUNT NEPHRITIES
A Case Report

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SUMMARY :

A 12-year-old who had a ventriculoatrial shunt for hydrocephalus due to congenital aqueduct stenosis and later developed shunt nephritis is reported. His renal function detoriated progressively within eight months while he was lost to follow-up. After removing the shunt, and with appropriate aggressive treatment renal function improved and complement levels returned to normal. Early removal of the shunt is mandatory is such a condition but even in delayed cases removal is beneficial.

KEY WORDS :

Shunt nephritis, removal of the shunt, antibiotic therapy.

INTRODUCTION

The first two cases of shunt nephritis were reported by Black and coworkers in 1965 (1). Since then about 100 cases have appeared in the literature (1,4,6-8). Immune mediated glomerulonephritis usually occurs a year or more, following development of bacteraemia due to a low virulance organism usually Staphylococcus albus or Staphylococcus aureus (6).

We report a 12-year-old boy who developed renal failure due to untreated shunt nephritis and improved after removal of the shunt and with appropriate treatment.

CASE REPORT

A 12-year-old boy presented with a one-year history of oedema of the eyelids and extremities and vomiting for 2 weeks.

He had a ventriculo-atrial shunt inserted at 6 years of age for hydrocephalus due to congenital stenosis of the aqueduct. The shunt system was never revised and on physical examination he was pale-looking, febrile (37.5°C) and oedematous. His blood pressure was 140/90 mm. Hg. He had hepatosplenomegaly, urine analysis revealed +4 proteinuria with a 1010 specific gravity with erythrocytes, leucocytes and granular casts in the sediment. Other laboratory findings were as follows : Hb 8.5 gr/dl, WBC 6700/mm³, ESR 150 mm/st, BUN 43 mg/dl, Cr 2.4 mg/dl, total protein 5.4 gr/dl, albumin 1.6 gr/dl, plasma electrolytes were normal, RF was (-), CRP, IC and mixed cryoglobulins were (+). C3 24 mg/dl, C4 8 mg/dl mg/dl. Blood culture grew proteus, IVP showed bilateral enlarged kidneys and widened parenchymas. A renal biopsy showed membranoproliferative glomerulonephritis with focal sclerosing.

Removal of the shunt and antibiotic treatment were planned but the patient was lost to follow-up for 8 months. Later he was hobpitalized with oedama and dyspnaea. Renal function showed deterioration with BUN between 124 and 171 mg/dl, Cr between 6 and 9 mg/dl. Antibacterial therapy was started and his ventriculoatrial shunt was removed. Following the operation a peritoneal dialysis was performed, daily fresh frozen plasma infusions were given for immune complex solubilization, and antibiotic treatment was continued. His renal functions improved within a month C3 and C4 reached the normal levels 130 mgé/dl and 26 mg/dl respectively. On his last check-up blood pressure was 140/100 mm Hg. 10.7 gr/dl, WBC 7300 /mm³, BUN 25 mg/dl, Cr 1 mg/dl, total protein 6 gr/dl, albumin 3.9 gr/dl. Urinary protein loss decreased from 3 gr/m²/day to 1 gr/m²/day CrC1 reached 103 ml/dk/173 m² from 15 ml/dk/1.73 m².

DISCUSSION

The association of glomerulonephritis and infected ventriculoatrial shunt is a well known clinicopathological entity (1-4,6-9). The typical clinical picture is an association of chronic septicaemia characterised by low grade fever, hepatosplenomegaly and anemia and renal involvement characterised by proteinuria associated with haematuria (1,5,7,8). Patients may

present with a full blown nephrotic syndrome. Renal insufficiency is usually absent at onset but develops subsequently in untreated cases (2,4). The levels of CH50, C1q, C3, C4 are depressed and a mixed polyclonal cryoglobulinaemia, and positive rheumatoid factor are often, present, cirulating immune complexes are found, membranoproliferative glomerulonephritis is characteristic of shunt nephritis (4,7,8).

With the clinical, laboratory and histopathological findings of the patient, a diagnosis of shunt nephritis was made. The recognised treatment for shunt nephritis such as appropriate antibiotic therapy and removal of the infected shunt were unfortunately delayed. Even with this delay the patient showed rapid improvement after treatment. Persistent high blood pressure and proteinuria might be considered as due to focal glomerulosclerosis.

The earliest possible removal of the shunt is necessary to prevent irreversible renal damage. Even in delayed cases, to stop further antigenic stimulation, removal of the shunt is beneficial.

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REFERENCES

1. Black JA, Challacombe DN, Ochenden BG : Nephrotic syndorme associated with bacteremia after shunt operations for hydrocephalus. Lancet ii: 921-924, 1965.
2. Dobrin RS, Day NK, Omie PG, et al : The role of complement immunoglobulin and bacterial antigen in coagulase negative Staphylococcal shunt nephritis. Am J Med 59: 660-673, 1975.
3. Harkiss GD, Brown DL, Evans DB : Longutidinal study of circulating immune complexes in a patient with Staphylococcus albus-induced shunt nephritis. Clin Exp Immunol 37: 228-238, 1979.
4. McKenzie SA, Hayden K : Two cases of "shunt nephritis." Pediatrics 54: 806-808, 1974.
5. Moncrieff MW, Glasgow EF, Arthur LJH, et al: Glomerulonephritis associated with Staphyloccus albus in a Spitz Holter valve. Arch Dis Child 48: 69-72,1973.
6. Striker L, Striker GE : Glomerulonephritis due to other infections, in Massry SG, Glassock RJ (eds) : Text Book of Nephrology. Baltimore : Williams and Wilkins, 1989, pp 621-622.
7. Tınaztepe K, Saatci Ü, Göksu N, Göğüş S : Ventrikulo-atrial şanta eşlik eden membranoproliferatif glomerulonefritis. Patoloji Bülteni 4(4): 291-296, 1977.
8. Toth T, Redl J, Beregi E : Shunt nephritis with cresent formation. Int J Pediatr Nephrol 8(4): 231-234, 1987.
9. Wald SL, Mchawrin RL : Shunt associated glomerulonephritis. Neurosurgery 3 : 146-150, 1978.