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Case report

A Rare Cause of Headache in a Patient with Poststreptococcal Acute Glomerulonephritis

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Abstract

We report a 34 year-old male patient with poststreptococcal acute glomerulonephritis who developed headache secondary to spontaneous intracerebral haemorrhage. He was treated medically and recovered completely. This case shows that spontaneous intracerebral haemorrhage can occur in patients with poststreptococcal acute glomerulonephritis. Whether this association is due to a decreased coagulation attributable to the post-infectious disorder or to an involvement of the brain vessel wall must be better clarified with further studies.

Keywords: headache, intracerebral haemorrhage, poststreptococcal acute glomerulonephritis

Introduction

The classic presentation of poststreptococcal acute glomerulonephritis (PSAGN) is a reversible nephritic syndrome with oliguric acute renal failure. Hematuria (characterized by red urine), headache, and generalized symptoms such as anorexia, nausea, vomiting, and malaise constitutes well known clinical feature of poststreptococcal glomerulonephritis [1]. Headache, which naturally occurs in the disease process, may therefore constitute a negligible symptomatology. We report a patient with PSAGN who was detected to have intracerebral haemorrhage during the evaluation for intractable headache. A description of his clinical course provides important information on the clinical features of PSAGN complicated by intracerebral haemorrhage.

Case report

A 34 year-old man was admitted to our hospital with edema at eyelids, hands and lower legs and oliguria which started five days ago. He was treated for 5 days with intravenous cefazolin for pharyngitis, two weeks before presenting symptoms. At physical examination, his blood pressure was 150/80 mmHg and hearth rate was 84 b.p.m., his body temperature was 36.5 ºC. Blood work results were as follows: Hgb 16.5 g/dL, WBC 9.500/mm³, platelet count 315.000/mm³, erythrocyte sedimentation rate 4 mm/hr, C-reactive protein (CRP) level 0.375 mg/dL (normal: <0.8 mg/dL). His daily urinary output was 400 ml and urinalysis showed a specific gravity of 1023, 2+ positive for protein and microscopic hematuria with numerous red blood cells and red blood cell casts. Serum biochemistry work-up revealed a serum creatinine of 1.3 mg/dL [ref. 0.7-1.3], blood urea nitrogen of 30 mg/dL [ref. 7-25] serum C3 level of 27.1 mg/dL [ref. 79-152], the serum antistreptolysin O (ASO) titer of 226 IU/mL [ref. 0-200]. Cryoglobulins, anti-nuclear and anti-neutrophil cytoplasmic antibodies were not detected. Serological tests for hepatitis B, C and HIV viruses were negative. Renal ultrasound showed normal-sized kidneys, non-distended urinary bladder and no evidence of hydronephrosis. The diagnosis of acute nephritic syndrome due to PSAGN was based on these laboratory and clinical findings. The patient was hospitalized. Two-dimensional and Doppler echocardiography demonstrated normal hearth function.

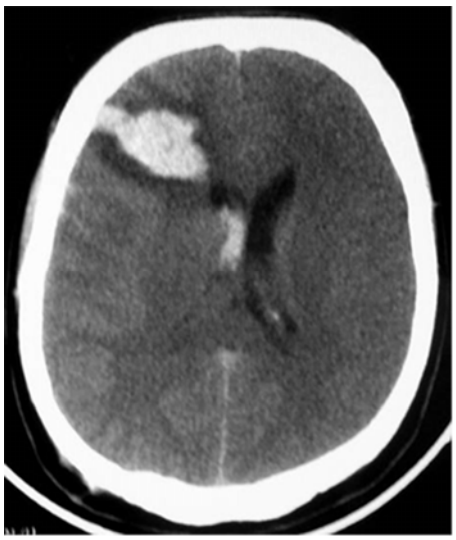

Fig. 1. Cranial computed tomography image demonstrate an intraparenchymal hemorrhagic lesion at frontal cerebral region which opened into the ventricular system

On the third day of admission, a headache located at the right parietal zone of the cranium was started. His neurological examination was completely normal. The headache was ascribed to the hypertension, and captopril 4 x 25 mg and paracetamol 3 x 500 mg daily was introduced. During the clinical course his symptoms including edema at

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eyelids, hands and lower legs improved and daily urinary output got normalized but headache did not improved. On the 7th day of admission cranial computed tomography (CT) showed an intraparenchymal hemorrhagic lesion at frontal cerebral region which opened into the ventricular system (Figure 1). Simultaneously examined routine coagulation tests revealed a prothrombin time of 13.8 sec [ref. 11.5-15.5, INR: 1.1] and activated partial thromboplastin time of 27.6 sec [ref. 26.5-40.0].

The patient received mannitol therapy for the consecutive three days. The brain magnetic resonance imaging (MRI) did not reveal any vascular or structural pathology underlying the intracerebral haemorrhage. Within 14 days, his symptoms including headache improved and he was discharged.

Discussion

PSAGN is an immune-mediated disease associated with throat or skin infections with certain nephritogenic strains of group A streptococci and usually diagnosed on clinical and serologic grounds without the need for renal biopsy [2]. Most patients have milder disease, and subclinical cases are common [1]. The long term prognosis is generally favorable, but some patients may have life-threatening acute complications due to impairment of renal functions or secondary to systemic hypertension. Hypertension is found in most of patients at initial presentation and in some cases may result with hypertensive encephalopathy [2]. However, intracerebral haemorrhage during the clinical course is unusual.

In the pathogenesis of intracerebral haemorrhage, it is known that acute raised blood pressure can definitely precipitate this life threatening state, particularly in previously normotensive individuals whose cerebral blood flow autoregulation is within the normal range [3]. In the present case, the patient did not have a history of chronic hypertension but his arterial blood pressure was at stage 1 hypertension level on admission. Undoubtedly, hypertension is neither a sufficient nor a necessary cause of intracerebral haemorrhage [3].

Co-existent glomerulonephritis and intracerebral haemorrhages have rarely been reported up to day. In a series by Sugiyama *et al*. in which the rapidly progressive glomerulonephritis (RPGN) cases were studied, two patients with RPGN associated with systemic lupus erythematosus were reported to die secondary to cerebral haemorrhages [4]. Intracranial haemorrhage as spontaneous subarachnoid haemorrhage has been reported in a 6-year-old boy with PSAGN [5]. In that case, the outcome was reported to be good as in our case. Gilboa *et al*. reported a child with PSAGN who developed spontaneous pulmonary haemorrhage. The patient in their case was successfully treated with methylprednisolone [6].

The present case shows that spontaneous intracerebral haemorrhage can occur in patients with poststreptococcal acute glomerulonephritis. Whether this association is due to a decreased coagulation attributable to the post-infectious disorder or to an involvement of the brain vessel wall must be better clarified with further studies.

Conflict of interest statement. None declared.

References

1. Hahn RG, Knox LM, Forman TA. Evaluation of poststreptococcal illness. *Am Fam Physician* 2005; 71: 1949-1954.
2. Sulyok E. Acute proliferative glomerulonephritis. In: Avner E, Harmon WE, Niaudet P, eds. *Pediatric Nephrology*. *Lippincot Williams & Wilkins*, Phiadelphia, USA: 2004; 601-613.
3. Warlow CP, Dennis MS, van Gijn J, Hankey GJ, Sandercock PAG, Bamford JM, Wardlaw JM. *Stroke a Practical Guide to Management*. *Blackwell Science Ltd*, London, UK: 2001; 339-375.
4. Sugiyama H, Makino H, Wada J, *et al*. A clinicopathological study of patients with immune complex type and pauci immune type rapidly progressive glomerulonephritis. *Nippon Jinzo Gakkai Shi* 1993; 35: 777-782.
5. DeBeukelaer MM, Young GF. Subarachnoid hemorrhage complicating acute poststreptococcal glomerulonephritis. *Arch Neurol* 1978; 35: 473-474.
6. Gilboa N, McIntire S, Hopp L, Ellis D. Acute noncresentric poststreptococcal glomerulonephritis presenting with pulmonary hemorrhage. *Pediatr Nephrol* 1993; 7: 147-150.