Case report

Goodpasture’s syndrome associated with multiple sclerosis

Henderson RD, Saltissi D, Pender MP. Goodpasture’s syndrome associated with multiple sclerosis. 

Multiple sclerosis (MS) has been reported to occur in association with other autoimmune diseases. Here we report the case of a woman with primary progressive MS who developed and died of Goodpasture’s syndrome associated with anti-glomerular basement membrane antibodies. Of interest she had the human leukocyte antigen haplotype (DRB1*1501-DQA1*0102-DQB1*0602) most frequently associated with these two diseases; however, it is likely that other genes, particularly those involved in immunoregulation, also contributed to her susceptibility to these diseases. The association of MS and autoimmune renal disease may be more common than currently recognized.

Multiple sclerosis (MS) can occur in association with other autoimmune diseases (1–3) and has been reported in a patient with membranous nephropathy (4), but to our knowledge the concurrence of MS and Goodpasture’s syndrome has not previously been reported. Here we report the case of a woman with MS who developed and subsequently died of Goodpasture’s syndrome associated with anti-glomerular basement membrane antibodies.

Case report

In 1990 at the age of 44 years she began to develop progressively increasing unsteadiness of gait and clumsiness of the upper limbs. On examination in 1993 she had dysarthria, head titubation, right optic atrophy, a coarse postural tremor and severe ataxia of the upper limbs, spastic ataxic paraparesis, impaired joint position and vibration sense in the lower limbs, generalized hyperreflexia and extensor plantar responses.

Magnetic resonance imaging of the brain revealed multiple periventricular cerebral white matter lesions consistent with MS and there were oligoclonal immunoglobulin G bands present in the cerebrospinal fluid that were not present in the serum. On human leukocyte antigen (HLA) typing she had DRB1*1401/1501-DQA1*0101/0102-DQB1*0603/0602. A diagnosis of primary progressive MS was made.

Her symptoms gradually progressed and in early 1997 she could walk only 20 m with bilateral assistance.

In February 1997 she was admitted to the Royal Brisbane Hospital with fatigue, nausea and oliguria-anuria for 3 days. There was no past or family history of renal disease and in 1993 her blood urea and serum creatinine had been normal. There was no history of hypertension, and her only regular medication was fluoxetine. Examination of the cardiovascular and respiratory systems was normal and she was clinically euvoletic.

On admission the blood urea was 31 mmol/l (normal range [NR]: 3.5–9.0) and serum creatinine 0.82 mmol/l (NR: 0.05–0.12). Urine microscopy revealed numerous leukocytes and erythrocytes but no casts. Urine culture grew Escherichia coli in low numbers. An ultrasound scan showed normal kidney size and no evidence of obstruction. Immune screening revealed markedly elevated serum antibodies against glomerular basement membrane (greater than 1000 units) and mildly elevated serum antineutrophil cytoplasmic antibodies of the cytoplasmic type (cANCA) with a titre of 40.

Despite treatment with plasmapheresis, dialysis and intravenous methylprednisolone (initially 1 g daily for 3 days), over the subsequent 2 weeks she developed progressive respiratory failure requiring
mechanical ventilation. There was one episode of haemoptysis. Her chest X-ray showed bilateral parenchymal shadowing. She died of respiratory failure 46 days after admission.

At a limited autopsy there was bilateral diffuse renal glomerulosclerosis with fibrotic crescents obliterating all the renal glomeruli so that immunofluorescence studies of the kidneys could not be performed. On the basis of the clinical, laboratory and histological findings, it was concluded that her acute renal and pulmonary disease was due to Goodpasture's syndrome caused by anti-glomerular basement membrane disease.

Discussion

The occurrence of MS and anti-glomerular basement membrane disease in the same individual raises the possibility that she was genetically predisposed to autoimmune disease. Interestingly, she had the HLA Dw2 (DRB1*1501-DQA1*0102-DQB1*0602) haplotype that is most frequently associated with MS (5, 6) and anti-glomerular basement membrane disease (7). However, it is likely that other genes, particularly those involved with immunoregulation, also contributed to her susceptibility to these autoimmune diseases. The association of MS and autoimmune renal disease may be more common than currently recognized.

References