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LYMPHOMA AND TYPE I MEMBRANO-PROLIFERATIVE GLOMERULONEPHRITIS—A CASE REPORT

The association of non-Hodgkin's lymphoma (NHL) and glomerulonephritis (GN) is well known. The clinical features of the renal disease usually correspond to a nephrotic syndrome with histologically minimal glomerular changes, whereas membrano-proliferative glomerulonephritis (MPGN) is quite rare. We present the case of a man with renal infiltration by low-grade NHL, MPGN and nephrotic syndrome. Interestingly, the nephrotic syndrome subsided spontaneously.

Case report. A 68-year-old Syrian man consulted in his country because of cervical and axillary lymphadenopathies. A node biopsy yielded low-grade NHL. He received no therapy. The serum creatinine level was 150 μmol/l. Several months later he developed renal failure and was admitted to hospital. Physical examination showed cervical and axillary lymphadenopathies, splenomegaly and edema in the lower extremities. The blood pressure was 160/95. Hemoglobin was 123 g/l, s-albumin 28 g/l, s-creatinine 415 μmol/l, s-urea 431.8 mmol/l, s-potassium 6.1 mmol/l, C3 was 377 mg/l (normal 900–2000) and C4 843 mg/l (normal 200–500). There was a monoclonal IgG kappa peak in the serum electrophoresis. The urinary excretion of protein was 10.8 g/day. Other parameters concerning blood counts, biochemistry, immunoglobulins and coagulation were normal. Tests for rheumatoid factor, cryoglobulins, antinuclear antibodies, HIV, hepatitis, ASO and toxoplasma were negative. An ultrasound examination showed splenomegaly while the kidneys appeared normal. A renal biopsy was performed. All glomeruli were diffusely enlarged, with an increase in the number of endothelial and mesangial cells (Fig. 1). Some glomeruli showed a marked increase in the mesangial matrix and a 'double contour' pattern in the capillary loops (Fig. 2). There was a monomorphic interstitial infiltration by small round lymphocytes with scanty mitotic activity. Immunofluorescence showed focal, scattered granular reactivity for IgM, C1 and kappa and lambda light-chains along the capillary walls. Reactivity for IgA, IgG and C3 was negative. A lymph node biopsy revealed a low-grade B-cell NHL (according to the Working Formulation). The patient was treated with Nifedipin and Furosemide. The urinary excretion of protein descended to 0.9 g/day. The complement levels remained low. After six courses of therapy with cyclophosphamide, vincristine and prednisone (CVP) no lymphadenopathies were found at physical examination, although the bone marrow remained infiltrated; the serum creatinine level was 168 μmol/l. The patient then returned to his native country and was lost for further follow-up.

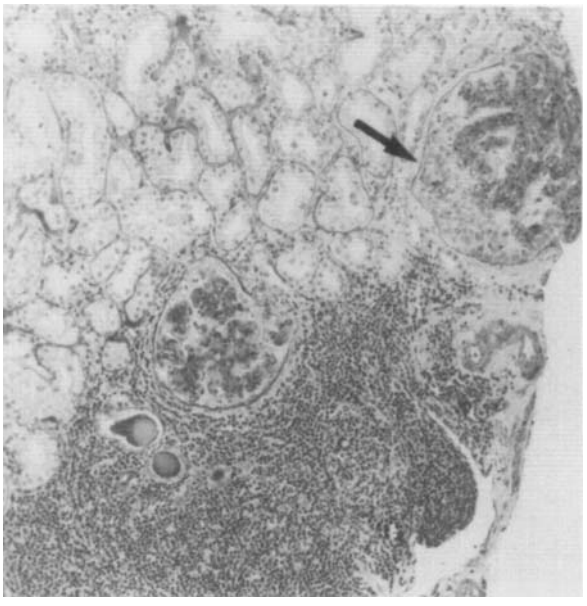

Fig. 1. Diffuse proliferative glomerulonephritis. One glomerulus is surrounded by an epithelial crescent (arrow). Diffuse interstitial infiltration by low-grade NHL is present. PAS, 40×.

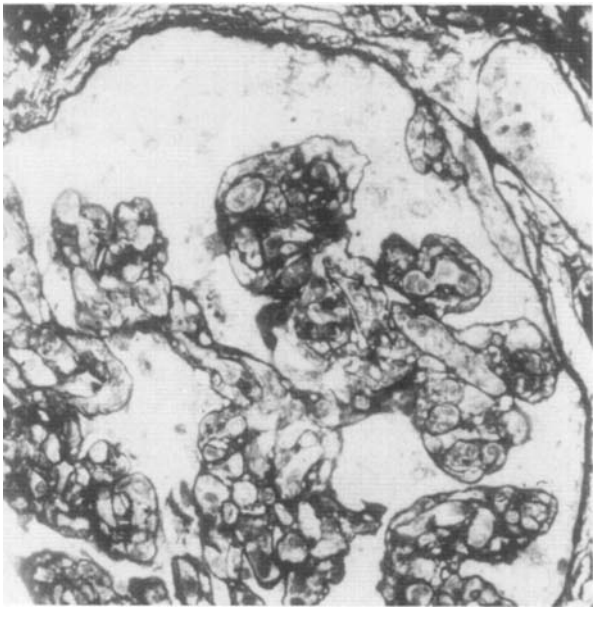

Fig. 2. Glomerulus showing marked increase in the mesangial matrix and 'double contour' in several capillary walls. PASM, 200×.

Discussion. In this case NHL with renal infiltration and type I MPGN coexisted. Renal infiltration by NHL is rather frequent. In an autopsy series of 696 patients with NHL, 33% had renal infiltration but renal failure had been detected in only 14% (1). This means that renal infiltration is usually clinically silent. Although a few cases of renal failure due to lymphomatous infiltration have been reported (2), the occurrence of clinical and

laboratory signs of nephropathy in a patient with NHL should give suspicion of other causes than renal metastases, for example, genuine GN. Minimal change glomerulopathy accounts for nearly 20% of these cases (3, 4). MPGN (5, 6) and crescentic GN have also been described. The pathogenesis of paraneoplastic GN remains uncertain, although autoimmune mechanisms may be involved, i.e. formation of immunocomplexes by antigens from tumor or normal tissues. Another proposed mechanism in lymphoproliferative disorders is T-lymphocyte dysfunction (7, 8). In well-documented cases the nephrotic syndrome has appeared at the same time as NHL, and some authors have reported remission of the syndrome in connection with treatment of NHL (9). In our patient it seems logical to correlate the clinical and laboratory nephropathy signs to the NHL, since they appeared only three months after the diagnosis of the malignancy and since no other causes of acute nephropathy were found. However, it is difficult to explain why the nephrotoic proteinuria disappeared before the administration of chemotherapy. Remission of the nephrotic syndrome after treatment of NHL has been reported (8), but we have not found reports of spontaneous remission. Spontaneous remission of idiopathic MPGN, although rare and transitory, has, however, been described (10). Powderly et al. (8) reported spontaneous remission of mesangioproliferative GN in a patient who had been treated 15 months previously for Hodgkin's disease; as the renal condition cleared without further treatment of the malignancy, the authors argued against an association. On the other hand, spontaneous remissions of NHL do occur, but this was not the case in our patient.

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PULMONARY HYPERTENSION—A RARE PRESENTATION OF CHORIOCARCINOMA

Gestational trophoblastic diseases (GTD) cover a spectrum of diseases with totally benign vesicular mole (hydatidiform mole) at one end of the spectrum and aggressive choriocarcinoma at the other end. The incidence of vesicular mole is 0.2 to 2 per 1 000 pregnancies (1). These patients are screened for potential malignant sequelae, and therefore few choricarcinomas nowadays present with advanced disease. However, trophoblastic tumours might occur after abortion and after normal pregnancies, and those occurring after normal pregnancy represent invariably choriocarcinoma. Rarely the tumour can present with pulmonary embolism and pulmonary artery hypertension. Recognition of this clinical entity is important due to the excellent outlook with appropriate treatment. We describe a case which was successfully treated with combination chemotherapy including etoposide, methotrexate (with calcium leucoverin rescue) actinomycin-D, cyclophosphamide and vincristine.

Case history. A 33-year-old female presented with 3 month's history of progressive exertional dyspnoea and later on dyspnoea at rest as well. She had had repeated episodes of right-sided chest pain during the previous month. She had a history of two abortions during the preceding 3 years and had had amenorrhoea for 2 months prior to presentation. On clinical examination she was cyanotic, had tachypnoea at rest, tachycardia (140/min) with a blood pressure of 100/60 mm Hg, left parasternal heave, a loud pulmonary second sound and raised jugular venous pressure. Pelvic examination revealed a soft cervix and a normal-sized uterus with bilateral ovarian cysts. Chest radiography showed a prominant pulmonary artery segment and ill-defined opacities in both lung fields. Electrocardiogram showed right heart dominance and echocardiogram revealed moderate pulmonary hypertension. Pulmonary wedge aspiration showed groups of undifferentiated malignant cells. The serum beta HCG concentration at presentation was 144 474 IU/l. Ultrasound of abdomen showed a normal uterus with clear uterine cavity and cysts in both ovaries. The patient was started on our high-risk chemotherapy protocol, alternating etoposide, methotrexate and actinomycin-D every week with vincristine and cyclophosphamide (EMA-CO). The beta HCG concentration decreased to normal within 12 weeks and chemotherapy was discontinued after 17 weeks. Obvious clinical improvement was noticed from the third week of therapy which was maintained throughout the course. At four months of treatment, the patient was asymptomatic except for dyspnoea on severe exertion; the signs of pulmonary artery hypertension had also disappeared.

Discussion. Bagshawe & Brooks (2) were the first to suggest that pulmonary artery hypertension (PAH) due to choriocarcinoma is potentially reversible. They also reported 30 years ago that choriocarcinoma could be found at necropsy in the pulmonary tree in patients who died of PAH. Still, not many know that choriocarcinoma can present with pulmonary embolism and PAH. The chest radiograph may be normal. V/Q scanning can help to confirm pulmonary emboli. Pulmonary angiography could be fatal due to dislodgement of the tumour. It is important to diagnose this clinical syndrome since it is curable if appropriately treated (3). Primary growth in the uterus or any other site may