Simultaneous Presentation of Gastric Carcinoma and Crescentic Glomerulonephritis

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Dear Sir,

Although the association of membranous glomerulonephritis with adenocarcinoma is well described [1], less well recognised is an association between crescentic glomerulonephritis and carcinoma. Only two series [2, 3], and a single case report [4] document this rare association. We report an additional case that presented with both disease states simultaneously.

A 65-year-old farmer was admitted with advanced uraemia [creatinine 2.33 mmol/l (26.3 mg/dl), urea 60 mmol/l (339 mg/dl)] and malaena. Nephrosis was absent and clinical examination was unremarkable except for a pericardial friction rub. Peritoneal dialysis was commenced. Further investigations revealed enlarged echogenic kidneys by ultrasonography, and a bleeding large sessile antral gastric polyp at endoscopy. Gastric biopsy demonstrated adenocarcinoma. Renal biopsy showed advanced crescentic glomerulonephritis with extensive sclerosis. No immune deposits were detected by either immunofluorescence or electron microscopy. Screening was negative for antibodies to neutrophil cytoplasm, cryoglobulins and paraproteins. The tumour was positive for carcino-embryonic antigen but this antigen was not demonstrated on glomerular staining. Malignant cells were found in dialysis effluent without evidence of macroscopic metastases radiologically. The patient declined palliative surgery and died 5 weeks after diagnosis.

The retrospective series of Whitworth et al. [2] found 4/60 patients with carcinoma who subsequently manifested crescentic glomerulonephritis, and Biava et al. [3] noted 7/81 in their series. Notably, 9 of these cases occurred after the tumour was found, at intervals up to 3 years. Many underwent varying chemotherapies and other treatment during this interval and this confounds attempts to causally link the two conditions. The nephrotic syndrome was uncommon and most did not demonstrate immune deposits, although this does not exclude an immunological mechanism such as T cell activation. There is some evidence that, like membranous lesions, the crescentic glomerulonephritis may be arrested if resection of the tumour is possible. This case demonstrated simultaneous presentations of the two conditions and provides further evidence of a cause and effect association. We suggest that malignancy should be considered in the differential diagnosis of crescentic glomerulonephritis, particularly in those over 40 years of age.

References

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