## Letters to the Editor

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## Acute Glomerulonephritis in Human Brucellosis

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Dear Sir,

A 31-year-old man, worker of dairy products, developed nephritic syndrome after 1 month of illness characterized by fever, sweating, weakness, malaise, anorexia, headache, without localized findings. The patient presented abrupt onset of hematuria and proteinuria, accompanied by evidence of azotemia and circulatory congestion, hypertension, and edema. There was no clinical evidence of streptococcal infections, and the ASO titer was below 200 IU. The  $C_3$  titer was below 16 mg/100 ml. The renal biopsy specimen was observed by light, immunofluorescent, and electron microscopy, and it showed the pathognomonic lesions of acute glomerolunephritis (fig. 1).

Two months later, the patient presented again fever, weakness, malaise, sweating and gross hematuria. He showed IgG and IgM Brucella agglutinins at dilutions as high as 1:1,280, and he rapidly recovered on tetracycline treatment. The investigation of Brucella agglutinins on sera samples of former admission to the hospital showed a titer as high as 1:640.

The incidence of different kinds of glomerulonephritis in infections caused by microorganisms [1] is well known, and it is also well known that Brucella nephritis is characterized by a clinical nephritic syndrome with tubulointerstitial damage and segmental glomerular lesions [2].

We have reported for the first time, as far as we are aware, histological findings of acute glomerulonephritis in brucellosis. We wonder whether brucellosis might induce glomerular lesions by immunological mechanisms

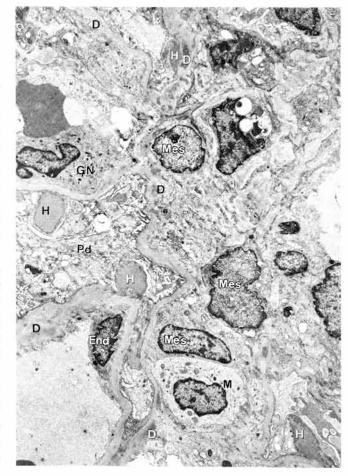


Fig. 1. Pathognomonic lesions of acute glomerulonephritis. Mes = MESANGIAL CELL; END = endothelial cell; Pd = podocyte; H = humps; d = electron-dense deposits; GN = granulocyte; M = monocyte. Glutaraldehyde fixation, Epon embedding, uranyl acetate and and lead citrate staining.

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[3] or if we have merely observed the coexistence of brucellosis and acute glomerulonephritis due to another disease. 3 Vogt, A.: New aspects of the pathogenesis of immune complex glomerulonephritis: formation of subepithelial deposits. Clin. Nephrol. 21:15-20 (1984).

## References

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