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Association between Chronic HCV Hepatitis, Membranoproliferative Glomerulopathy and Cutaneous Sarcoidosis

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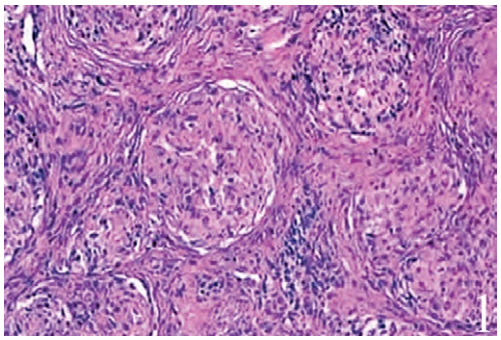

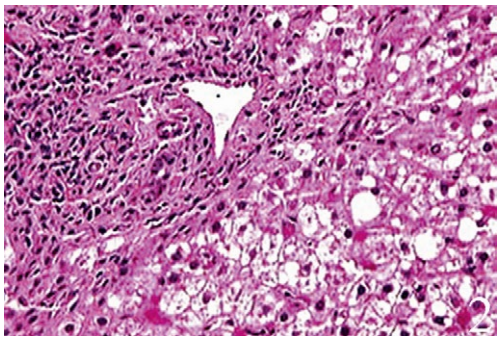

A 66-year old woman with chronic HCV hepatitis was being evaluated for antiviral therapy. On admission we observed a reddish indurated cutaneous lesion of about 1.5/2cm affecting the nose. Laboratory tests revealed proteinuria (5g/ 24 hours), mild hepatocytolysis, RNA- HCV level of 900,000UI/ml and positive serum cryoglobulins. Biopsy from the cutaneous lesion revealed circumscribed non-caseating granulomas composed of epithelioid cells, with Langhans giant cells and without necrosis, located in the superficial dermis (Fig.1, H&E x20). The CT scan of the thorax revealed multiple mediastinal adenopathies, but no pulmonary lesions; cytology after broncho-alveolar lavage evidenced lymphocytic alveolitis. The serum level of angiotensin converting enzyme was 48.9 U/ml (normal 15-20 U/ml). The diagnosis established was of stage I sarcoidosis with cutaneous manifestation. A liver biopsy, revealed mild steatosis and a METAVIR score of A2 F2 (Fig.2, H&Ex20). No granulomas were found in the biopsy.

We performed a kidney biopsy to investigate the nephrotic syndrome. Optic microscopy showed thickening of the capillary walls. Ultrastructural studies by electron microscopy showed a thickened basement membrane, with dense epi-membrane deposits, the "dome and spike" aspect. The diagnosis was of membranoproliferative glomerulonephritis (MPGN).

Kidney disease due to chronic HCV infection occurs mostly in association with type II cryoglobulinemia. Kidney biopsy frequently shows type I MPGN. In our patient, a type II MPGN was diagnosed [1].

Sarcoidosis could appear after interferon and ribavirin treatment for HCV; only rarely has been HCV-induced sarcoidosis reported [2-4]. Our patient did not require systemic treatment for sarcoidosis. Local corticosteroid application was recommended.

We started pulse therapy with metyl-prednisolone for the nephrotic syndrome, which was followed by a remarkable decrease of proteinuria [5]. After six cycles, we initiated the Peg-interferon and ribavirin treatment. Sustained virologic response was achieved. The patient is presently under follow-up, which includes monitoring of proteinuria, serum liver enzymes and pulmonary ventilatory function.

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Conflicts of interest: None to declare.

REFERENCES

1. Morales JM, Kamar N, Rostaing L. Hepatitis C and renal disease: epidemiology, diagnosis, pathogenesis and therapy. Contrib Nephrol 2012; 176: 10-23. doi: 10.1159/000333772
2. Faurie P, Broussolle C, Zoulim F, Trepo C, Sève P. Sarcoidosis and hepatitis C: clinical description of 11 cases. Eur J Gastroenterol Hepatol 2010; 22: 967-972. doi: 10.1097/MEG.0b013e32833834bc
3. Fallahi P, Ferri C, Ferrari SM, Corrado A, Sansonno D, Antonelli A. Cytokines and HCV-related disorders. Clin Dev Immunol 2012; 2012: 468107. doi: 10.1155/2012/468107
4. Brjalin V, Salupere R, Tefanova V, Prikk K, Lapidus N, Jõeste E. Sarcoidosis and chronic hepatitis C: a case report World J Gastroenterol 2012; 18: 5816-5820. doi: 10.3748/wjg.v18.i40.5816
5. KDIGO clinical practice guidelines for glomerulonephritis. Chapter 9: Infection related glomerulonephritis. Kidney Int Suppl 2012; 2: 200-208. doi: 10.1038/kisup.2012.22