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전신성 홍반성 낭창 환자에서 동반된 미세변화 신증후군 1예

서동범, 이경주, 송준호, 이승우, 김문재, 인하의대 내과학교실

전신성 홍반성 낭창은 자가면역 질환으로 병원성의 자가항체와 면역복합체와 연관되어 발생된다고 생각되어지고 있다. 전신성 홍반성 낭창은 자가항체의 과잉 생산되고, 여러 장기에 면역복합체가 축척 된다. 낭창성 신염은 항DNA항체가 사구체에 축척 되어 발생한다. 전신성 홍반성 낭창에서는 낭창성 신염이 동반되는데, 미세변화 신증후군이 동반된 예는 드물다. 저자들은 전신성 홍반성 낭창 환자에서 동반된 미세변화 신증후군 1 예를 보고하는 바이다.

증례: 41세 여자환자가 급속히 진행하는 체중증가와 전신부종을 주소로 내원하였다. 내원시 malar rash가 관찰되었고, 혈중 creatinine 2.2 mg/dL, albumin 1.6 g/dL, 24시간 소변 단백질 5.8g/day, 항핵항체 1:320 양성, 항 DNA 항체 1:320 양성, 백혈구 3400/mm3 이었다. 낭창성 신염 의증 하에 신생검 시행하였다. 신생검 상 광학 현미경상 정상이었고, 면역형광 현미경 소견 상 음성이었으며, 전자현미경상 족돌기의 융합소견이 관찰되었고, electron-dense deposit은 관찰되지 않았다. 미세변화 신증후군 진단 하에 경구 steroid 사용후 환자의 전신부종은 호전되었고, 혈중 creatinine도 1.1mg/dL로 감소하였다.

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Hemorrhagic Fever with Renal Syndrome with Secondary IgA Nephropathy-A Case Report-

Moon Ho Yang, [1]Tae Won Lee, [1]Chun Gyoo Ihm, [1]Myung Jae Kim
Department of Pathology, [1]Department of Internal Medicine, College of Medicine, Kyung Hee University, Seoul, Korea

Hemorrhagic fever with renal syndrome (HFRS) is one of the most important infectious disease in Korea and asian teritories, which is caused by Hantaan virus, transmitted by infected mice (Apordemus agrarius, Ratus ratus, laboratory room animals and others). Clinical findings are divided in 5 phases; febrile, hypotensive, oligouric, diuretic and convalescence phase. The pathologic characteristics show the importance of vascular dysfunction in the pathogenesis of this disease, characterized by selective hemorrhage in the right atrium, renal medulla, skin and the anterior pituitary glands in autopsy findings. Renal biopsy findings are variable according to the clinical phase and severity and were as follows: normal glomeruli, mesangial expansion, varying cellular proliferation, proliferative glomerulonephritis, acute tubular necrosis, medullary congetion, interstitial hemorrhage and inflammation. IF study reveals some granular mesangial deposition of IgA, IgG, IgM and C3 in human biopsy kidney and several experimental studies. IgA nephropathy is the most frequent nephropathy in Korea and divided in primary and secondary. The secondary IgA nephropathy is thought to be caused by hepatobiliary disease, mucosal disease, infection, malignancy, some hematologic disease and systemic autoimmune or heypersensitivity disease. We experienced IgA nephropathy in a 23-year-old HFRS patient. Urinalysis disclosed 1.3 gm/day of protein, 20-25 RBC/HPFs, many WBC/HPFs and some of the granular casts. Indirect IF study for Hantaan viral Ab. was 3+. A percutaneous renal biopsy was performed at 15th. hospital day of diuretic phase. Light microscopic findings show diffuse mild mesangial proliferative glomerulonephritis with a few mesangial fuchsinophilic deposits in trichrome stain, and evidence of ATN and patchy interstitial inflammation. IF study shows diffuse granular mesangial deposits of IgA and C3 and weak deposits of IgG and IgM. Electron microscopic study reveals occasional electron dense deposits, mostly in paramesangiums and some in axial area. In summary of these findings, we made a diagnosis of secondary IgA nephropathy associated with HFRS. But the superimposed primary IgA nephropathy can not be completely ruled out.