라 모두 진행되었으나 lovastatin 투여군이 단순 신증후군 유발군 보다 경화도의 단계별 변화에서 현저한 감소를 보였다.

4) 신동맥 Microfil 관류 소견상 단순 신증후군보다 lovastatin 투여군에서 엽간 및 소엽간 세동맥의 분절과 염전 발생 부위가 적었고 사구체 모세혈관망의 결손도 적게 관찰되었다.

이상의 결과로 보아서 신증후군에서 고지혈증은 사구체 경화증의 하나의 중요한 원인 요소로 생각되며 효과적인 지질대사 개선제는 고지혈증에 의한 진행성 사구체 경화성 변화를 경감시킬 수 있는 것으로 사료된다.

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Studies on the Role of Interleukin-4 and Fc e R II in the Pathogenesis of Minimal Change Nephrotic Syndrome

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Minimal change nephrotic syndrome (MCNS) has been often associated with elevated IgE levels and referred to involve immune dysfunction. We investigated the role of interleukin-4 (IL-4) in the pathogenesis of MCNS through the regulation of membrane FceR II, low affinity receptor for IgE, expressed on B-cells. A significantly higher expression of membrane FceR II was found on fresh B-cells of MCNS (N=18) than on those of normals, as analyzed by fluorescence activated cell scanner by double antibody staining with anti Leu 16 (pan B marker)-FITC AND ANTI Leu 20 (FceR II)-phycoerythrin.

Sera from these patients also demonstrated increased IL-4 activity as compared with those of normal subjects. We observed that nephrotic T-cells has a greater ability (up to 9 fold) than normal cells to produce IL-4 (5~100 U/ml) to effectively induce FceR II expression (up to 6 fold). These results suggest that MCNS may be a T-cell disorder involving abnormal production of IL-4 and that the counter-regulation of FecR II by IL-4 lplay an important role in pathogenesis of MCNS.

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Brush Border Deficiency of renal Proximal Tubule in Idiopathic Fanconi Syndrome (Clinico-Pathological Correlation Presentation)

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Clinicopathological correlation of idioapthic Fanconi syndrome was assessed in 36/12 year old girl.

The case was characterized by hyperchloremic metabolic acidosis (Cl 114 mEq/L, pH 7.29, pCO_2 16 mmHg, HCO_3 8 mEq/L), renal glycosuria (FBS 74 mg%, urine glucose (lll) with 196 mg%), hypophosphatemic rickets (P 2.3 mg%, $FEPO_4$ 32.4%, flared, frayed and cupped epiphyses of radius and ulna), hypouricemia (uric acid 2.0 mg%), hypokalemia with K wasting (2.9 mEq/L, FE K 60.7%) and renal aminoaciduria.

Further work up on functional study of renal tubule revealed proximal tubular acidosis, evidenced by 21.2% of $FEHCO_3$, 54 mmHg of (U-P) pCO_2 and 5.0 of urine pll.

In autopy specimen, pronounced deficiency or lack of brush border of renal proximal tubule and effaced microvilli as well as segmental thickening of tubular membrane, hitherto unreported, were notably found. thickening of tubular membrane, hitherto unreported, were notable found.