Proteinuria with Analbuminuria

To the Editor:

We read with interest the Case Report by Sun et al. (1). The patient exhibited a type of proteinuria that appears not to have been described hitherto. However, we are not content with the report, because some important aspects of the case were not adequately documented.

When one is describing a novel type of proteinuria we feel it is essential that the following information should be given:
(a) the amount of protein excreted in the urine per 24 h.
(b) the day-to-day variation in urinary protein excretion.
(c) whether or not the electrophoretic pattern was consistent from day-to-day.

It is unclear how β2-microglobulin was identified in the urine, because the paper by Leurrel (2)—we are assuming that this is the paper referred to by the authors—we are unable to trace the reference as quoted—deals only with the electrophoretic mobility of different protein fractions in plasma, cerebrospinal fluid, and urine. We consider that identifying proteins solely on the basis of their electrophoretic mobility is insufficient when such an unusual case of proteinuria is evaluated. Immunological techniques such as immunofixation or immunelectrophoresis should be used to identify and confirm the presence or absence of different proteins.

The patient they describe suffers from diabetes mellitus, ischemic heart disease, hypertension, and chronic renal failure. We are not told whether he received insulin or any other treatment for these conditions, other than an unspecified amount of salicylate for headaches. We would like to know if his renal failure was believed to be secondary to analgesic abuse, diabetes, or perhaps due to intrinsic renal disease.

Unusual electrophoretic patterns of urine concentrates have been described in three situations: (a) in overload proteinuria (3), which may be due to Bence Jones proteinuria when in some cases the free light chains may polymerize to form dimers, trimers, etc. in hemoglobinuria, lysozymuria, and myoglobinuria, or when patients are receiving gelatin-hydrolysate plasma expanders, which are largely catabolized and excreted in the urine (4).

These infusions give rise to increased amounts of β2-microglobulin in the urine (5). (b) Patients with tumors of the urinary tract such as carcinoma of the bladder occasionally get "histuria" or post-renal proteinuria (6) when tumor proteins, which are not normally found in serum, are excreted in the urine. (c) Very occasionally, patients with Munchausen's syndrome have been known to add protein, such as egg white, to their urine, which will give rise to bizarre electrophoretic patterns (7). We would like to know that these causes of proteinuria were looked for and excluded as far as possible in this case.

The authors are incorrect in their statement that albumin is not reabsorbed by the renal tubules. It has been estimated that under normal circumstances filtration at the glomerulus and subsequent reabsorption and catabolism by the renal tubules account for 10% of total albumin catabolism (8).

References


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An author of the paper in question responds:

To the Editor:

The main purpose of our paper was to document a case of proteinuria with analbuminuria. The electrophoretograms of two urine specimens and one serum specimen should have served this purpose well. Other clinical and laboratory data were provided to explain the possible pathogenesis. For the benefit of the patient, tests were performed only when they were relevant to the diagnosis and/or prognosis of the disease. For conciseness, unnecessary information was not included.

The inquiries posed by Hutchison and O'Reilly seem to be irrelevant to what we wanted to achieve. Our conclusion that the urinary proteins in this case were tubular in origin was based on the following facts: (a) they are small molecules, as demonstrated by sodium dodecyl sulfate/polyacrylamide gel electrophoresis (SDS/PAGE); (b) they are not identical to any of the serum proteins, which should have been present in urine if glomerular permeability was increased; (c) only a small amount of urinary proteins (1) was present, as evidenced by the low staining intensity of protein bands in the SDS/PAGE pattern and by the detection of 1+ protein on urinalysis. As we mentioned in our paper, absolute quantification of urinary protein is not necessary for distinguishing between tubular and glomerular proteinuria. Therefore, we do not believe that studies of 24-h urinary protein, daily urinary protein excretion, and daily electrophoresis would help to explain the pathogenesis of analbuminuria in our patient.

β2-Microglobulin, as its name indicates, is designated by its electrophoretic mobility and low relative molecu-