AN UNUSUAL CASE OF PROTEINURIA

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Many conditions cause proteinuria, and it has
been stated that any patient showing this sign
should not be lost sight of until a firm diagnosis
has been made. Occasionally the establishment
of such a diagnosis is difficult, but the case reported
here was finally carried to a successful conclusion.

Case Report

The patient, a married woman of 31, was admitted
in October, 1957, complaining of malaise and visual
disturbance. She had had a normal pregnancy seven
years previously. Two years before admission she
said that she had an attack of haematuria, associated
with puffiness of the eyes and feet. A diagnosis of
acute nephritis was made and she recovered with some
residual frequency. She started another pregnancy in
July, 1956, and was admitted to another hospital in
December with proteinuria and hypertension; a diag-
nosis of pre-eclampsia was made and labour was
induced five weeks prematurely. The baby was
healthy and survives, though due to the patient's
persistent ill-health she has never been able to look
after it. Discharged from hospital in January, 1957,
she was readmitted later in the same month for
persistent proteinuria and remained in hospital until
April. Since discharge she had suffered continual
malaise, losing 4 stone in weight and complaining of
oliguria and visual disturbances. Physical examina-
tion revealed no abnormality apart from a persistent
proteinuria. The fundi appeared normal and no
lesion could be found to account for the visual dis-
turbance. The patient was extremely disturbed, and
for this reason a psychiatric opinion was sought,
minimal attention being paid to the proteinuria. An
agitated state of depression was diagnosed, possibly
psychotic in origin, and treatment for this condition
undertaken with E.C.T.

Renal Investigations.—In view of the persistent pro-
teinuria a number of investigations were carried out.
The serum urea was 35 mg. % and the creatinine
clearance 75 ml./min., which was within normal limits.
A proteinuria of 11 to 15 g./day was present, but in
spite of this the serum albumin level was normal at
3.2 g. %. This was surprising, for at this level of
urinary protein loss, considerable serum protein
changes would be expected. In addition the serum
cholesterol was normal, and the urine deposit free
from formed elements. On paper electrophoresis of
serum and urine (Hardwicke, 1954), the urine pattern
appeared unusual and when the two specimens were
run in parallel on the same strip of paper (Fig. 1) a
significant difference in mobility between the two
"albumins" was found, while the β globulin seen in
the urine had no counterpart in the serum. Immunol-
ogical examination of the urine against an antibody
mixture raised with whole human serum (Gell, 1955)
gave a weak reaction for albumin but no reaction for
any globulin fraction. This finding was also unusual,
since globulins being present in all ordinary cases of
proteinuria. The mean molecular weight of the urine
proteins was therefore determined by colloid osmo-
metry (Rowe, 1957); this gave a value of 44,000, a
value considerably lower than in any case of
proteinuria previously examined, with the possible
exception of cases of myelomatosis.
FIG. 2.—Immunological comparison of the patient's serum and urine with whole egg-white. The egg-white and urine specimens show the similar zones of precipitation, representing identical antigens present in both. Serum shows no trace of either of these antigens.

At this point further specimens of urine were required and a catheter specimen was requested. The ward reported that the urine had been free of protein for the previous two days, and this was confirmed on the specimen obtained. However, a further 24-hour sample proved again to contain over 1 g.% of protein. Close inspection of this sample revealed a thin layer of clear gelatinous sediment which aroused suspicion. Specific precipitation tests were then carried out with rabbit anti-egg-white serum (Fig. 2), and this shows clearly the identity with egg-white of the urine proteins; this finding is further confirmed by comparative electrophoresis (Fig. 3). As a final check a further sample of urine was requested, great emphasis being laid, in the patient's presence, on the interest attached to this specimen. The eggs on the patient's locker were counted and the next day two eggs had disappeared while the 12 g. of protein was in the urine.

Simulated proteinuria is a rare condition in civilian practice and can be difficult to prove. In this case suspicion was aroused by the dissociation of this sign from any other evidence of renal disease. Incontrovertible proof was obtained by immunological techniques, and when challenged the patient admitted the deception, claiming that it was only done to call attention to the kidneys, which she still believed to be diseased. This case appears therefore to fall into the group of "deliberate disability" described by Hawkings, Jones, Sim, and Tibbetts (1956), who consider these problems are unconsciously motivated though consciously executed, and thus differ from true malingering.

I am indebted to the consultant staff of the Queen Elizabeth Hospital for referring the patient for examination, to my colleagues Drs. J. R. Soothill, D. S. Rowe, and D. R. Stanworth for advice and for the application of their specialized technical skills, and to the nursing staff for their co-operation in finally detecting the deception.

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