Impairment of renal function after intravenous immunoglobulin

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Introduction

In this report, we describe two cases of renal failure following intravenous immunoglobulin (IVIG) treatment for chronic inflammatory demyelinating polyneuropathy (CIDP). Renal biopsy showed identical findings in both cases.

Case 1

A 72-year-old woman with CIDP was treated with IVIG. During the second day of treatment, the patient noticed an increase in abdominal girth and a decrease in urine output, without other symptoms. Laboratory findings included serum creatinine of 4.6 mg/dl (407 μmol/l) and blood urea nitrogen of 65 mg/dl (23.2 mmol/l). Urinalysis showed 2+ protein and many hyaline casts. A renal biopsy was done to determine the cause of renal failure.

Case 2

A 72-year-old man was hospitalized due to new onset of bilateral leg weakness. Following investigations, a diagnosis of CIDP was made and the patient was treated with IVIG. Over the next 3 months, the patient had a progressive rise of his creatinine levels, from 1.1 to 2.8 mg/dl (97 to 248 μmol/l). Creatinine clearance was 20 ml/min. Urinalysis showed 2+ red blood cells. Urine protein was 217 mg/24 h. The patient underwent a renal biopsy to determine the cause of renal failure.

Renal biopsy

Both cases showed similar findings. The glomeruli did not show any significant changes. Tubules showed marked degenerative changes with swelling and prominent cytoplasmic vacuolization of the proximal tubular epithelial cells. The vacuolization was extensive and isometric (Figure 1A). There was no significant

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interstitial inflammation. Immunofluorescence micro-
scopy was negative for immune deposits in the
glomeruli. Electron microscopy showed glomeruli
with no significant changes while tubules showed
prominent vacuolization (Figure 1B).

Renal biopsy diagnosis. Acute tubular necrosis, with
extensive isometric tubular vacuolization.

Discussion

We show the renal biopsy findings of two cases that
developed acute renal failure with characteristic
tubular changes following IVIG treatment for CIDP.
The pathological findings included tubular epithelial
cell injury characterized by isometric intracytoplasmic
vacuolization and cellular swelling. The vacuolization
is due to substances like sucrose and maltose in the
IVIG preparations that result in osmotic nephrosis.
Acute tubular necrosis is often transient and
reversible following cessation of IVIG treatment.
Renal function should be monitored during IVIG
treatment.

Conflict of interest statement. None declared.