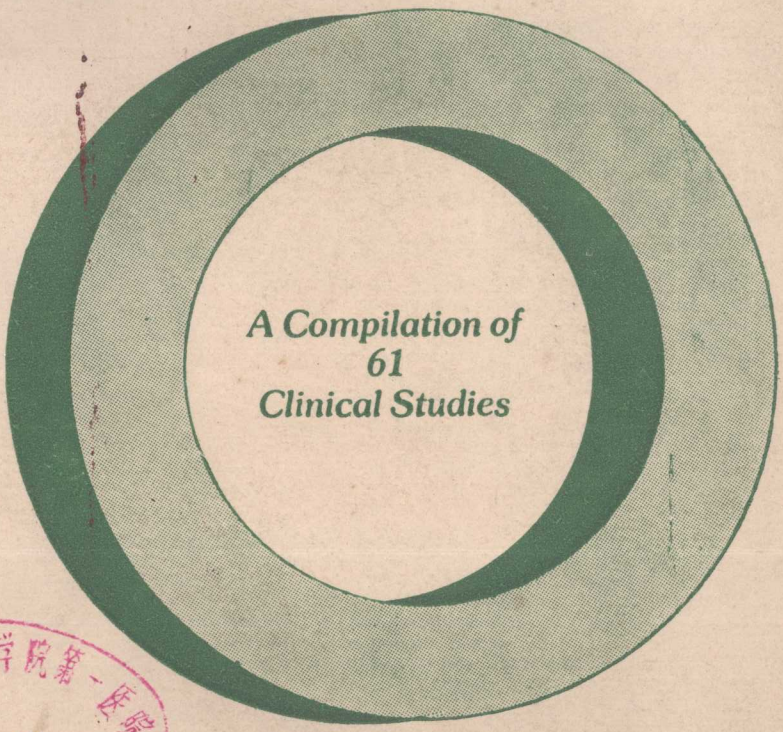
Kidney Disease Case Studies

Second Edition



ALVIN E. PARRISH, M.D.

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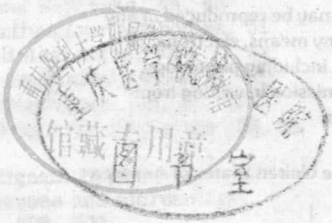
A Compilation of 61 Clinical Studies



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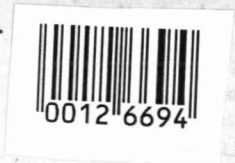

Library of Congress Card Number
79-90644

ISBN 0-87488-022-X

January, 1980

Printed in the United States of America

SIMULTANEOUSLY PUBLISHED IN:

Brazil	:	GUANABARA KOOGAN Rio de Janeiro, Brazil
Europe	:	HANS HUBER PUBLISHERS Bern, Switzerland
Japan	:	IGAKU-SHOIN Ltd. Tokyo, Japan
South and East Asia	:	TOPPAN COMPANY (S) Pte. Ltd. Singapore

Preface

In this second edition of *Kidney Disease Case Studies*, we have updated the information and added new cases to illustrate additional problems in clinical nephrology. We feel that the presentation of illustrative cases represents a different approach to the study of medicine, and even though cases are selected to represent specific diseases, they still may not fall neatly into clinical textbook descriptions. This method of presentation provides a more realistic approach to what the physician faces in clinical practice.

The cases are presented as initial work-ups were obtained, and information concerning biopsy material is given whenever it is either applicable or available. The answers to the questions presented with each case are often fairly straightforward and obvious to those who are familiar with the subject being discussed. We have provided answers, which to the best of our knowledge are correct, and pertinent literature is cited to justify the answers and to encourage further study of particular topics of interest.

notice

The editor(s) and/or author(s) and the publisher of this book have made every effort to ensure that all therapeutic modalities that are recommended are in accordance with accepted standards at the time of publication.

The drugs specified within this book may not have specific approval by the Food and Drug Administration in regard to the indications and dosages that are recommended by the editor(s) and/or author(s). The manufacturer's package insert is the best source of current prescribing information.

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KIDNEY DISEASE

CASE 1: HEMATURIA, SORE THROAT, UREMIA

HISTORY

An 18-year-old woman presents with a history of swelling around her eyes for the past 2 days and dark brownish, cloudy, coffee-colored urine of one day's duration. She had sore throats and tonsillitis repeatedly for many years and had recently been told that she should have a tonsillectomy. She had noticed that her rings had been tight for the past 24 hours. She gives an additional history of having a cold about 10 days earlier along with her usual sore throat. She denies any kidney disease although one uncle died at age 60 of "kidney trouble."

PHYSICAL EXAMINATION

She was a normally developed woman with facial puffiness and periorbital edema. BP 150/100, P 80, T 37°C. Physical examination was otherwise normal except for a grade 2 systolic murmur over the base of her heart, bilateral CVA tenderness, and 2+ ankle edema.

LABORATORY DATA

Hemogram: Hemoglobin - 14 Gm/dl; Hematocrit - 44%; WBC - 9800; Normal differential count; LE-negative

Urinalysis: SP. Gr. - 1.028; Protein - 4+; Sediment - many RBC, RBC casts. Few granular casts. Few WBC. Creatinine clearance - 20 ml/min

Blood Chemistry: BUN 164 mg/dl; Creatinine - 10.8 mg 1 dl; Cholesterol - 250 mg/dl

Serology: ASO-350 total units

Throat Culture: Beta-hemolytic streptococci

COURSE

On the day of admission her urine output was 376 ml/24h and it remained low until the 3rd hospital day when it increased to 3600 ml and remained above 1000 ml/day thereafter.

Her blood pressure fell to 110/70 on the 5th hospital day; her BUN and creatinine began to decrease at the same time and returned to normal after 30 days.

Her urine continued to show 1-2+ protein, RBC and granular casts until 3 months after the onset of her disease but was clear after this time.

A repeat creatinine clearance corrected to a surface area of 1.73 m^2 was 68 ml/min at 3 months and 102 ml/min at 1 year following her illness.

QUESTIONS

1. Immunofluorescent staining of the kidney biopsy should show:
   A. linear deposits of IgG and Beta 1-C
   B. granular deposits of IgG and Beta 1-C
   C. spike-like deposits of IgG and Beta 1-C
   D. subendothelial deposits of IgG and Beta 1-C

2. Deposition of IgG and Beta 1C are thought to be due to:
   A. deposition of soluble antigen-antibody complexes on the glomerular basement membrane
   B. the presence of antiglomerular antibody
   C. antiglomerular basement membrane deposits
   D. trapping of serum proteins by the glomerular basement membrane

3. Poststreptococcal glomerulonephritis has a prognosis as follows:
   A. 50% of patients progress to some form of chronic nephritis
   B. about 2% of patients have persistent abnormal urine
   C. all patients with poststreptococcal glomerulonephritis have persistent renal impairment
   D. 25% of patients recover

4. Which of the following are also immune complex nephropathies?
   A. Membranous glomerulonephritis
   B. Lupus nephritis

C. Membranoproliferative glomerulonephritis
D. Berger's syndrome
E. All of the above

5. Which of the following is the most characteristic finding in acute glomerulonephritis?
A. Hematuria with RBC casts
B. Hypertension
C. Edema
D. Hypercholesterolemia

6. Hypertension in acute glomerulonephritis is thought to be due to:
A. increase in vascular volume
B. renal ischemia
C. increased aldosterone excretion
D. increased renin production
E. all of the above

7. Acute glomerulonephritis following a nephritogenic streptococcal infection:
A. occurs in a majority of patients
B. rarely occurs
C. occurs in 10-20% of patients
D. may be asymptomatic

8. Which of the following diagnostic procedures would be the most appropriate in this case?
A. Creatinine clearance
B. Renal biopsy
C. Urinalysis
D. ASO titer

ANSWERS

1. (B) Acute proliferative glomerulonephritis is characteristically associated with electron dense deposits on the epithelial side of the glomerular basement membrane (Fig. 1.1). These are thought to be antigen-antibody complexes and correspond to the granular appearance of the fluorescence seen in immunofluorescent staining[3] (Fig. 1.2). The antigen, presumably streptococcal in origin, during the first 1-12 days of the disease may be distributed in the mesangium and in the endothelial side of the glomerular basement membrane.[4]

2. (A) (See Fig. 1.2). Fluorescein labeled anti-IgG and Beta 1-C staining of frozen sections of kidney biopsies taken from

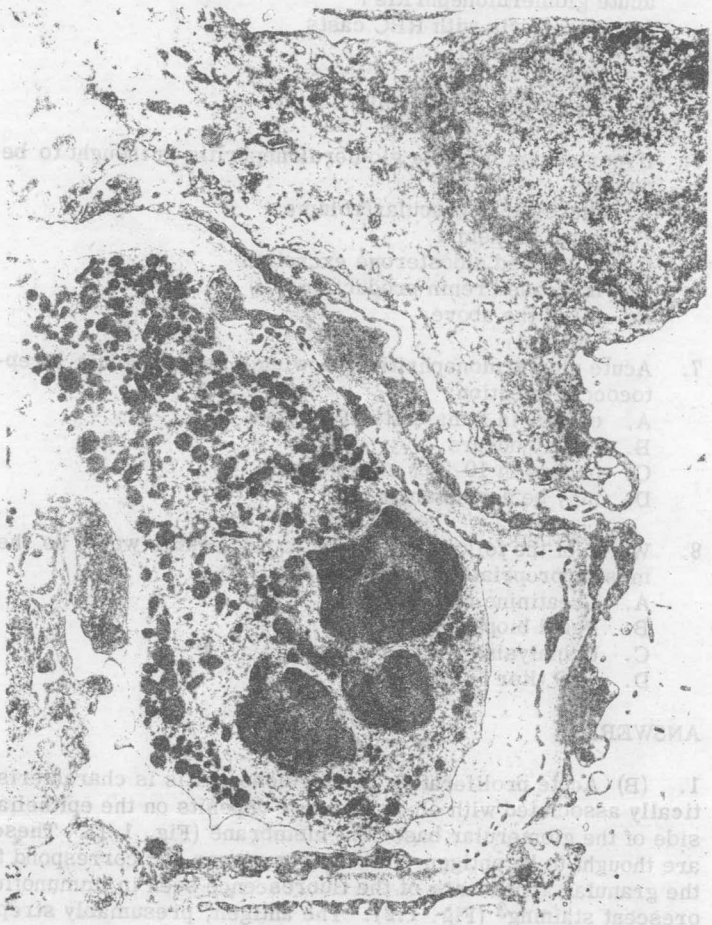

FIG. 1.1: Electron microscopy in acute poststreptococcal glomerulonephritis showing electron dense deposits on the epithelial side of the basement membrane.

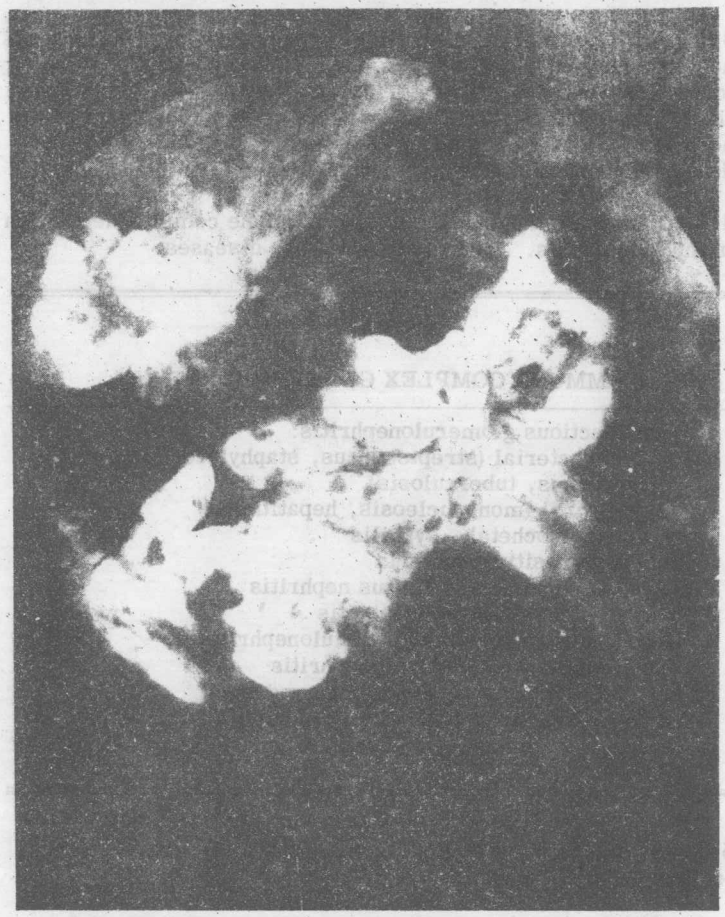

FIG. 1.2: Immunofluorescence staining showing IgG deposits in the glomeruli.

patients with acute poststreptococcal glomerulonephritis show the presence of both gamma globulin (IgG) and complement (Beta 1-C) located along the outside of glomerular basement membrane.

3. (B) or (A) There are conflicting reports concerning the prognosis. Baldwin[1] reports that more than half of his 60 patients, followed 2-15 years, had evidence of renal damage. In 760 patients followed 2-6 years, Potter[2] found a 1.8% incidence of abnormal urine, 1.4% incidence of hypertension, and an 8% incidence of transient urinary abnormalities.

4. (E) All are thought to be due to immune complex deposition in the glomerulus. Table 1.1 lists these diseases.

TABLE 1.1

IMMUNE COMPLEX GLOMERULOPATHY

1. Postinfectious glomerulonephritis:
   a) Postbacterial (streptococcus, staphylococcus, pneumococcus, tuberculosis)
   b) Postviral (mononucleosis, hepatitis)
   c) Postspirochetal - syphilis
   d) Postparasitic - malaria
2. Lupus erythematosis - lupus nephritis
3. Membranous glomerulonephritis
4. Membranoproliferative glomerulonephritis
5. Focal sclerosing glomerulonephritis
6. IgA, IgG nephropathy (Berger's syndrome)
7. Glomerulopathy associated with certain malignancies
8. Glomerulopathy associated with endocarditis and shunt infection

5. (A) Hematuria with RBC casts is the most frequent manifestation of the disease.

6. (E) All of these factors probably play a role in the hypertension of acute glomerulonephritis. Hypertension may not be present during the acute phase of the disease. Lewy et al.[5] reported an incidence of 46.5% of blood pressure higher than 140/90 in children. In our experience with adults, the incidence was 53%.

7. (D) The asymptomatic occurrence of glomerulonephritis has been reported by a number of authors. In a recent study of 248 children with streptococcal infection, 54 showed no symptoms of

renal disease, although urinary abnormalities or depressed serum complement levels were found, and renal biopsy showed changes consistent with glomerulonephritis.[6]

8. (C) The correct answer is a matter of opinion since all of these tests may be of help. We would choose a good urinalysis, looking for protein, red cells, and red cell casts, since it is simple and usually diagnostic. There have, however, been several reports of individuals with biopsy proven glomerulonephritis who do not have diagnostic changes in the urine.[7,8]

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CASE 2: HEMATURIA, OLIGURIA

HISTORY

A 53-year-old man gave a history of having a sore throat with fever, facial and ankle edema, 1 month before being seen. He was admitted to another hospital at that time and was thought to have acute glomerulonephritis. During that hospitalization his clinical status rapidly deteriorated, with a rising BUN, nausea, vomiting, bloody urine decreasing in amount. He was transferred to a dialysis center after one week. His BUN at that time was 210 mg/100 ml.

PHYSICAL EXAMINATION

He was an acutely ill man constantly retching. BP 190/100, P 100, T 36°C. His fundi showed arterial narrowing. A pericardial friction rub was heard over the lower sternum. His lungs were clear but he had moderate ascites and facial and ankle edema.

LABORATORY DATA

Hemogram: Hemoglobin - 7.8 Gm/100 ml; Hematocrit - 28%; WBC - 12,000 with 78% segmented forms; Platelets - normal

Urinalysis: Sp. Gr. - 1.015; Protein - 4+; Sugar - 0; pH - 6.0; Sediment - 18-30 RBC/HPF. Many RBC casts; Creatinine clearance - 2 ml/min

Blood Chemistry: BUN - 175 mg/100ml; Creatinine - 15.2; Cholesterol - 183 mg/100ml

Serology: VDRL - negative; ASO titer - 625 Todd units

Throat Culture: negative

COURSE

The patient worsened progressively. His urine output fell to 0 and remained so until his death 2 weeks later. Hemodialysis was started, but because of technical difficulties, was never satisfactory, and the patient developed pulmonary edema and died. A renal biopsy done during his 2nd week of illness is shown in Fig. 2.1.

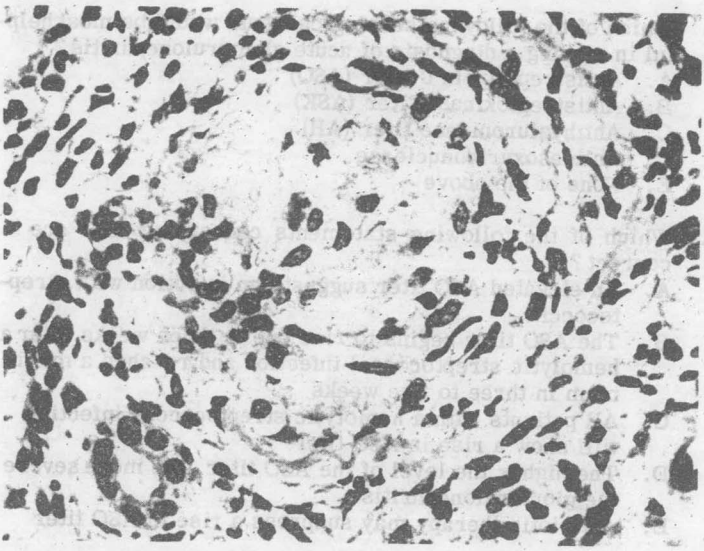

FIG. 2.1: Biopsy showing far advanced changes in the glomerulus.

QUESTIONS

1. Acute glomerulonephritis associated with prolonged oliguria is:
   A. associated with a high mortality rate
   B. a frequent complication of glomerulonephritis
   C. not associated with a poor prognosis
   D. indicative of tubular involvement

2. A renal biopsy would most likely show:
   A. numerous epithelial crescents
   B. nodular densities throughout the glomeruli
   C. glomerular sclerosis
   D. chronic glomerulonephritis

3. Which of the following therapeutic procedures should have been used?
   A. Hemodialysis
   B. Transfusion with red blood cells
   C. Kidney transplantation
   D. Antihypertensive therapy
   E. Digitalis
   F. Penicillin