IgA Nephropathy

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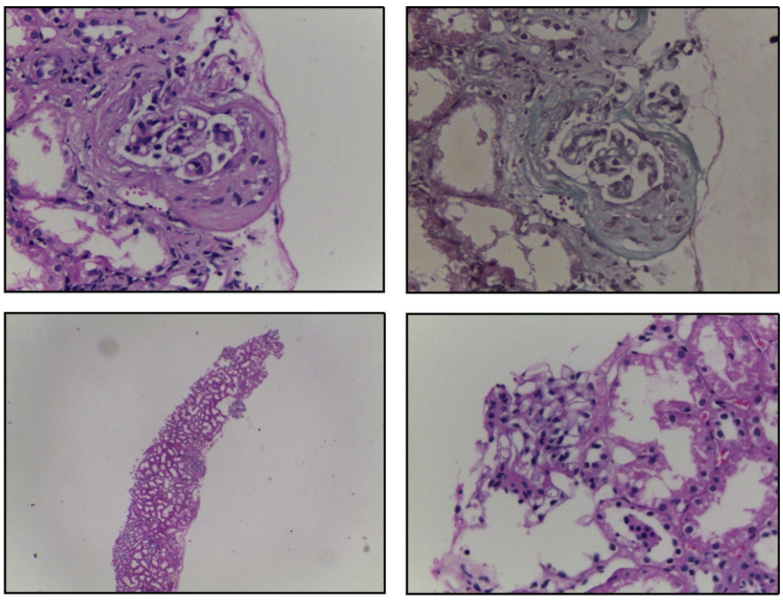

Figure 1.

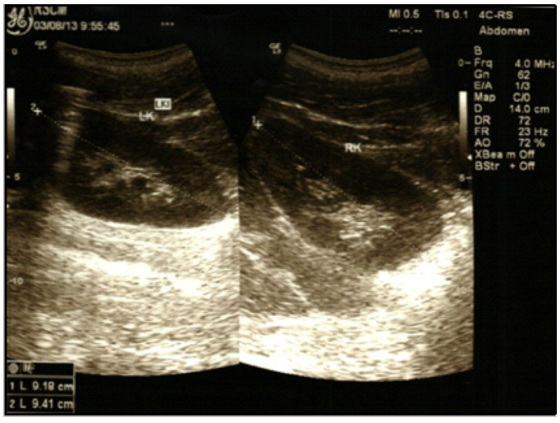

Figure 2.

IgA nephropathy was first described histologically by Berger and Hinglais in 1968. At that time, this kind of glomerulonephritis was known as the pathologic terminology of intercapillary deposits of IgA and IgG (in France: les depots intercapillaires d'IgA-IgG).[1] Currently, IgA nephropathy is common worldwide and it is diagnosed by clinical presentation, laboratory data especially urinalysis and urinary quantitative protein, and also by kidney biopsy.[2] Not many studies have published histopathological presentation of IgA nephropathy as well as clinical trials to clarify the treatment choice of patients with IgA nephropathy.

This is a case of a 33 year-old man with a history of general edema (face, eye, and lower extremities) two years ago. He had been diagnosed as suffering from nephrotic syndrome by the physician and treated without having a kidney biopsy with methylprednisolone. He received methylprednisolone with tappered doses, but after he stopped taking the drug, general edema reoccured, just as his first clinical presentation. Edema always appeared each time methylprednisolone treatment was discontinued. The physician prescribed him methylprednisolone again and referred him to Cipto Mangunkusumo Hospital to undergo a kidney biopsy.

From physical examination, there was edema on the lower extremities, but there no hypertension was found after several check-ups. From the laboratory examination, there is hypoalbuminemia (albumin 2,8), hipercholesterolemia (total cholesterol 259), antinuclear antibodies (ANA) was positive, and a high level of serum IgA (481). Serum ureum was 18 while serum creatinine 0,7. From the urinalysis, there are erythrocyte sediments, hyalin casts, blood, and protein was +3. Urinary quantitative protein was 3459,5. From the renal ultrasound, the size and form of the kidney was normal, with firm borders between cortex and medullae. From the biopsy, there is a deposit of IgA in the mesangial. The biopsy showed mesangial proliferative glomerulonephritis with fibrosis in the capsula of Bowman. From the reviews of renal pathologists, it was concluded that the biopsy indicated IgA nephropathy.

Diagnosis of primary IgA nephropaty was proven by the kidney biopsy. From the clinical presentation, the patient is classified as having moderate to high risk of IgA nephropathy with normal glomerular filtration rate (GFR). The treatment option is optimal supportive therapy with tappered dose of steroid, anti-proteinuric agent such as angiotensin-converting enzymes (ACE)-inhibitors or angiotensin II receptor blockers (ARBs), and controlled dietary intake (restricted protein intake).[2] For optimal treatment, the urinary quantitative protein was reduced to 314 and edema was resolved. The successful treatment of IgA nephropathy also depend on optimal evaluation of urinary quantitative protein, serum ureum/creatinine, and blood pressure. If there is an increase of urinary protein or the patient falls to RPGN (rapid progressive glomerulonephritis) or nephrotic syndrome, we must add an immunosupressive agent.[2] The choice of immunosupressant agent based on evidence is cyclophosphamide or mycophenolate mofetil.[3] The prognosis of the disease is good. IgA nephropathy progresses slowly and only half of the patients progress to ESRD (end-stage renal disease) within 25 years.[4]

ACKNOWLEDGMENTS

We thank Endang Sri R. Hardjolukito, MD, MSc, Head of Department of Anatomical Pathology Faculty of Medicine University of Indonesia, Cipto Mangunkusumo Hospital and Mrs. Uppy, the pathology slide photographer, for the histopathology photographs used in this article.

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