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On the Cover

Case Description:

What is the Diagnosis?

An 18-year-old female, diagnosed with seronegative polyarticular juvenile inflammatory arthritis at 4 years of age, presented with anasarca. She had been treated with methotrexate and nonsteroidal anti-inflammatory drugs (NSAIDs) for the past 9 years. On examination, she had an inflammatory, nondeforming symmetrical polyarthritis involving large joints, mainly the knees. Her serum creatinine was 2.1 mg/dl and 24-hour urine protein was 3.9 g. ANA was negative. C3 and C4 were normal.

Image Description:

Kidney biopsy showed the deposition of amorphous, acellular, eosinophilic material in the glomeruli (left) and on the basement membrane of some tubules and the walls of blood vessels. These deposits on Congo red staining gave an orange-red appearance under light microscopy, apple green birefringence under polarized light, and intense red fluorescence under fluorescence microscopy using a Texas red filter (middle). Immunofluorescent staining showed no deposition of immunoglobulins, complements, or light chains. The amyloid subtype was confirmed as AA amyloid by immunohistochemistry (right).

Teaching Points:

The fibrils in various types of amyloidosis have similar appearance under light and electron microscopy. They all generate birefringence under polarized light with Congo red dye. Immunohistochemistry can be used to determine amyloid subtype. Laser-capture microdissection followed by mass spectrometry is the new diagnostic tool for amyloid typing.

Amyloidosis occurring in childhood is extremely rare and is usually of AA type, complicating chronic inflammatory diseases. Proteinuria in children with juvenile inflammatory arthritis could be due to the use of NSAIDs, antirheumatoid drugs such as gold and penicillamine, and certain indigenous medicines. AA amyloidosis may develop in children who have long-standing, poorly controlled inflammatory arthritis. It is important to recognize the cause of proteinuria in these patients. AA amyloidosis occurs as a reaction to a chronic inflammatory condition. Infections and inflammation cause the liver to produce serum amyloid A (SAA) protein, an acute phase reactant protein, in high levels. When the inflammation persists, a portion of the SAA protein called the AA protein may be deposited in various tissues as insoluble AA amyloid fibrils. The kidney is the most affected organ in AA amyloidosis, leading to proteinuria, nephrotic syndrome, or kidney impairment. It is a progressive disease leading to kidney failure.

In addition to controlling the inflammation, treatment options for AA amyloidosis include colchicine and antiproliferative cytokines like IL1 β , TNF, and IL-6. Since there was no active inflammatory process at present, antiproliferative treatment options were not considered. She is currently on diuretics, salt restriction, and angiotensin-converting enzyme inhibitors. Renal transplantation may be considered later.

(Text and images provided by Anila Abraham Kurien, Department of Pathology, Renopath Center for Renal and Urological Pathology, Chennai, India; Jerry Joseph, Department of Nephrology, Stanley Medical College, Chennai, India; and Edwin Fernando, Department of Nephrology, Stanley Medical College, Chennai, India.)