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KIDNEY CONDITION IN JIA

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Resume

Juvenile idiopathic arthritis (JIA) is systemic diseases that can involve various organs, including the kidneys. JIA can exhibit characteristic renal involvement, which requires proper treatment and diagnosis. In this review, we discuss renal involvement in classic JIA. Renal manifestations in JIA vary from asymptomatic to end-stage kidney disease. It is important to recognize that renal abnormalities can be a symptom of rheumatologic disease because they can provide important signals towards establishing a personalized treatment plan.

Keywords: Arthritis, juvenile; kidney diseases; children; glomerulonephritis; TIN

Introduction

The kidneys are important target organs involved with JIA. The field of pediatric rheumatology originated in the first half of the 20th century and has a relatively short history compared to other medical fields. It started principally with interest in juvenile chronic inflammatory arthritis, the most common childhood rheumatologic disease [1].

Currently, its scope is expanding to address rare disease groups that have recently been elucidated, including autoinflammatory syndrome. JIA mainly targeting the musculoskeletal system, blood vessels, and other tissues, and is still a significant cause of chronic illness in children worldwide; although it remains among one of the smallest pediatric subspecialities [2].

Pediatric rheumatologic diseases are frequently associated with renal disease as a part of systemic autoimmune disease, and in some diseases such as systemic lupus erythematosus and antineutrophil The spectrum of renal involvement in JIA: secondary amyloidosis, secondary tubulointerstitial nephritis. JIA is diseases, characterized by chronic noninfectious inflammation of the joints and encompasses a complex group of diseases. It is the most famous and frequent rheumatoid disease in children and is classified into several groups, according to clinical and laboratory characteristics. In the early history of JIA, gold nephropathy was an interesting kidney disease associated with the use of intramuscular gold salts, although there is currently no gold treatment. The pathological picture of gold nephropathy is drug induced membranous glomerulonephritis that usually resolves over time if gold treatment is stopped [3,4]. Cytoplasmic antibody-associated vasculitis, the kidney is the main target organ that can indicate the long-term prognosis.

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Renal manifestations in JIA vary from asymptomatic to end-stage kidney disease. It is important to recognize that renal abnormalities can be a symptom of rheumatologic disease because they can provide important signals towards establishing a personalized treatment plan. In addition, kidney abnormalities may be a presenting symptom of JIA; in this case, clinicians should attempt to identify the underlying disease. In this paper, we review kidney problems that can be accompanied by representative juvenile idiopathic arthritis (JIA). Sjogren's syndrome (SS), systemic sclerosis/scleroderma, and juvenile dermatomyositis (JDM), Lupus nephritis and antineutrophil cytoplasmic antibody-associated renal disease tend to expand over a wide range of topics, and thus were excluded from this review.

Although it is difficult to determine the cause of renal abnormalities in JIA, according to one prospective study in adults, proteinuria and decreased renal function were mainly due to drug side effects, while hematuria was associated with the disease itself [5]. The renal diseases associated with JIA have rarely been reported, yet include renal amyloidosis, glomerulonephritis, and drug induced tubulointerstitial nephritis (TIN). Amyloidosis is the most characteristic lesion associated with chronic systemic inflammation in JIA [6,7]

Renal amyloidosis

Amyloidosis is characterized by the deposition of amyloid fibrils in organs and there are a number of subtypes. Amyloid A (AA) amyloidosis is caused by the overproduction of the precursor of AA protein, which is produced in response to systemic inflammation, while amyloid light-chain amyloidosis is caused by the overproduction of monoclonal immunoglobulin light chains. Only AA amyloidosis (secondary amyloidosis) can occur in children with JIA [7,8].

In the past, amyloidosis was the main cause of death in JIA; however, recently, the prognosis has improved [8-10]. Renal amyloidosis occurs most commonly in systemic onset JIA (sJIA), followed by polyarticular JIA [8,9]. Renal amyloidosis insidiously progresses, causing massive proteinuria from an asymptomatic state, and consequently leading to endstage renal disease. Hematuria is rarely accompanied [8,11]. Regular urinalysis is required in patients with sJIA or polyarticular JIA as asymptomatic proteinuria is the most common initial symptom [7]. It can be confirmed by renal biopsy which demonstrates amyloid fibrils, although the correlation between the degree of amyloid deposition and clinical symptoms are The pathogenesis of renal involvement in JIA remains unclear. Immunologic abnormalities related to the occurrence of JIA,including hypergammaglobulinemia, abnormal B and T cell mitogen responsiveness, decreased T suppressor activity, and uncontrolled cytokine production, are presumed to lead to renal involvement [23,24, 26].

In JIA, the treatment for glomerulonephritis is generally conservative with angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers. not clear. Treatments that control the inflammatory cascade caused by the underlying diseases are critical. Several disease-modifying antirheumatic drugs and biologics have been used to control these diseases. Since renal amyloidosis occurs in a situation where JIA is not well controlled by standard drugs such as methotrexate, sulfasalazine, and hydroxychloroquine, it is usually treated by adding disease-modifying antirheumatic drugs or biologics or switching biologics after the diagnosis of amyloidosis [6,12].

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Interleukin-6 inhibitor, tocilizumab has become the standard treatment for sJIA, and it can play an important role in treating secondary amyloidosis by suppressing serum AA levels [7,13,14]. According to one large study conducted in 2008, of the 3,500 patients with JIA, 24 patients with biopsy-proven amyloidosis were detected. Ten patients died, but the cause of death was associated with JIA itself rather than with amyloidosis. Of the 4 survivors, three patients underwent kidney transplants, and 11 patients maintained normal renal function at last follow-up. Proteinuria improved completely in four patients who initially had proteinuria [6, 8]. Renal disease can be improved by early intensive treatment. Therefore, it is essential to monitor regularly the occurrence of amyloidosis in JIA patients.

Glomerulonephritis and drug-induced tubulointerstitial nephritis

Several studies on adult rheumatoid arthritis (RA) suggest that there is an association between RA and different types of glomerulonephritis, although studies on glomerulonephritis in JIA are extremely rare. Mesangial proliferative glomerulonephritis is the most commonly reported type of glomerulonephritis in adults with RA [6, 14, 15]. In JIA, membranous nephropathy, mesangial glomerulonephritis, focal segmental glomerulosclerosis, and crescentic glomerulonephritis have also reported [15-21]. Nephrotic syndrome in JIA is extremely rare and is usually caused by amyloidosis rather than glomerulonephritis [20, 25]. However, severe cases with rapidly progressive glomerulonephritis or massive proteinuria require intensive treatments with immunosuppressants [16-22, 27, 28, 29]. One should be aware that drugs used as treatments can also cause renal abnormalities. Drugs commonly used in JIA include nonsteroidal anti inflammatory drugs, proton pump inhibitor, methotrexate, sulfasalazine, leflunomide, etc. Since these drugs can often cause renal abnormalities such as acute tubular necrosis and tubulointerstitial nephritis (TIN). Clinicians should be careful to identify the cause of renal abnormalities in patients taking these drugs. **Conclusions.**

JIA in children can affect various organs,including the musculoskeletal, cutaneous, pulmonary, heart, gastrointestinal, central nervous system, and kidneys. It is necessary to understand the type of renal disease associated with each JIA forms to properly monitor and treat renal involvement in this patients. In certain cases where patients are initially expressing symptoms of renal disease alone glomerulonephritis or TIN, we should try to find the underlying diseases, and then renal disease can be a early diagnostic clue for underlying JIA.

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