A Case of Tubulointerstitial Nephritis in a Young Male

Vinay Srinivasa*, G. Kan, V. Manickam, V. Srivastava

Townsville Renal Unit, Townsville Hospital QLD, Australia

*Corresponding author: Vinay Srinivasa, Townsville Renal Unit, Townsville Hospital QLD, Australia. Email: vinay_srinivasa@health.qld.gov.au


Received Date: 09 May, 2018; Accepted Date: 16 May, 2018; Published Date: 23 May, 2018

Abstract

Tubulointerstitial Nephritis and Uveitis (TINU) syndrome is a rare disease reported in the literature that is unrecognised. 250 cases have been described in the literature since it was first reported by Dobrin et.al in 1975. Diagnosis requires histological confirmation by renal biopsy. We report a case of an 18-year-old male diagnosed with TINU syndrome who was treated with steroids and who has responded to treatment.

Introduction

Tubulointerstitial Nephritis and Anterior Uveitis syndrome (TINU) is a rare disorder. It was first described in 1975 by Dobrin, and since then more than 250 cases have been recorded [1-3]. Although reported in adolescents and females, it may present in all ages and in both sexes with an incidence rate of 0.1 to 2% worldwide [1,2]. It has been suggested that it is underdiagnosed due to lack of recognition [4].

Case History

We present a case of an 18-year-old male who was admitted to hospital from the ophthalmology clinic, with bilateral blurred vision consistent with bilateral uveitis. He had no past medical history and was not taking regular medications. He was a nonsmoker and did not drink alcohol. He was admitted under the neurology team and proceeded to have neurological investigations such as MRI brain and lumbar puncture. MRI brain and lumbar puncture were both negative. Additionally, HLA B27 testing was negative. Sarcoidosis was suspected but serum ACE level was normal, and imaging of his chest was not suggestive. Urinary beta 2 microglobulin was high at a value of 5802 µg/L and serum creatinine was slightly raised at 110 micromol/l. Urine protein excretion rate from urinary protein studies was approximately 1.9 grams per day. Based on these findings a renal biopsy was ordered. Results of renal biopsy showed active chronic tubulointerstitial nephritis and a diagnosis of TINU was made. Our patient was commenced on a course of oral steroids and following this his creatinine improved to 75 micromol/l, proteinuria resolved and urinary beta 2 microglobulin was decreased to a value of 1563 ug/L. Furthermore, his bilateral uveitis has disappeared after treatment with topical steroids.

Discussion

This case report presents several teaching and learning points about the diagnosis and management of TINU syndrome. It has been reported that TINU syndrome is under recognized with physicians not familiar with this disorder. TINU is said to occur more frequently in females (3:1) with the median age of onset being 15 years [2].

Definitive TINU is diagnosed, when acute tubulointerstitial nephritis is firmly established and the patient has bilateral anterior uveitis of sudden onset [1,2]. Acute tubulointerstitial nephritis is diagnosed by histological examination by kidney biopsy or by fulfilling the following criteria [5,6]; abnormal renal function (increased serum creatinine or decreased creatinine clearance) [1]; abnormal urinalysis: increased β2 microglobulin, low-grade proteinuria, presence of urinary eosinophils, pyuria or haematuria without infection, urinary white cell casts, or normoglycemic glycosuria; and [2] a systemic illness lasting ≥ 2 weeks, characterised by a combination of the following symptoms and laboratory findings: (a) signs and symptoms: fever, weight loss, anorexia, malaise, fatigue, rash, abdominal or flank pain, arthralgia, or myalgia; and (b) laboratory findings such as the evidence of anaemia, abnormal liver function, eosinophilia, or a Western erythrocyte sedimentation rate > 40 mm/h. When two of the 3 criteria are fulfilled, and typical uveitis is present, a diagnosis is probable for TINU [1]. There is often a time interval between the diagnosis of uveitis and that of interstitial nephritis making diagnosis difficult [1,2]. Moreover in 35% of patients with
TINU, ocular findings precede or develop concurrently with acute interstitial nephritis [2].

In our patient, the ocular findings of anterior uveitis and raised urinary beta 2 microglobulin were diagnosed simultaneously. The main differential diagnosis is sarcoidosis. Granulomatous changes are seen in uveitis secondary to sarcoid, this is not seen in uveitis as a result of TINU. Moreover, renal involvement in sarcoid is rare [2]. The pathogenesis of TINU is still unclear with autoimmune diseases such as Rheumatoid arthritis, sjogrens syndrome and infection such as EBV proposed as causative factors [1,2,4]. In our patient autoimmune serology and infection screen was negative. Treatment of TINU is with steroids. Uveitis is treated with topical steroids. Oral corticosteroids are often prescribed in patients with renal impairment with some studies reporting a mixed response. There have been few prospective randomized trials comparing steroid therapy with placebo and duration of treatment is uncertain [4]. 3-6 months of corticosteroids has been employed in several cases. In patients presenting with uveitis, renal functions must be evaluated as chronic renal damage may occur, early detection and steroid treatment may prevent renal complications [2,4].

Conclusions

TINU syndrome is an underdiagnosed disorder and should be considered in the differential for any patient presenting with anterior uveitis with associated renal impairment and high urinary beta 2 microglobulin. Urinary protein studies should be employed in the workup of a patient with bilateral uveitis as this may increase detection of TINU. Renal involvement in TINU is responsive to oral corticosteroids.

References