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Case Report

Persistent hypocomplementemia in a 9 year old boy following acute post-streptococcal glomerulonephritis

Abstract

Acute post-streptococcal glomerulonephritis (APSGN) is still common in our region and it is characterized by acute onset of edema, gross hematuria, hypertension, acute kidney injury and temporary hypocomplementemia with a preceding group A beta-hemolytic streptococcal infection. Here is a report of Persistent hypocomplementemia even after 5.5 years in a 9year old boy. There are a few reports with prolonged hypocomplementemia but no similar cases with this much follow up has been reported in the literature.

Introduction

Acute post-streptococcal glomerulonephritis (APSGN) is a common form of acute glomerulonephritis in children [1]. It is presented with edema, hematuria, acute renal failure and hypertension and hypocomplementemia 7-10days following a group a β-hemolytic streptococcal pharyngitis or 2-4 weeks following an impetigo [2-4]. Subclinical forms are common in close contacts [5-7]. APSGN usually is a self-limiting disease and only requires conservative therapy for complications of acute kidney injury and also management of hypertension. Severe cases may occur with a rapidly progressive course (rapidly progressive glomerulonephritis RPGN) [8], which may need kidney biopsy and pulse therapy with corticosteroids. Alternate pathway activation and depression of C3 is the usual mechanism but in some cases there may be activation of classic pathway [4]. C3 Complement returns to normal level within 8 weeks. In our case despite a usual feature APSGN and spontaneous resolution of nephritis C3 is still low even after 5.5 years.

Case Report

A 9 year old boy referred for persistent low C3 following an episode of acute glomerulonephritis for kidney biopsy. He had been admitted 4 months ago to another center with typical features of acute, Glomerulonephritis (edema, gross cola color hematuria and oliguria) on 29 Nov 2011.He had history of sore throat and treatment with few days of oral amoxicillin about 10 days prior to the development of periorbital edema.

His physical examination according to summary sheet: mild periorbital edema, BP130/90 mmHg, body weight 40kg and no peripheral edema. His lab data during admission was as follows: Urinalysis many RBC, 1+protein and few RBC cast, BUN 25mg/dl,Cr0.9mg/dl,24hr urine protein 160mg and creatinine 750mg, ASO titer 800 Todd unit, C_3 0.3gm/l (0.9-1.8), C_4 0.16gm/l(0.1-0.4),CH_{50} 86(100-300),ANA and Anti-dsDNA level were normal and also HBsAg ,HCV Ab and HIV Ab was negative. CBC was in favor of thalassemia minor. He had received only a few days of oral furosemide for his high blood pressure and discontinued when BP was normal and his gross hematuria lasted only a week. Four months later he was referred with completely normal physical examination and normal laboratory data including a normal UA except for a low $\mathbf{C_3}$ 0.66gm/l for kidney biopsy. Regarding normal physical examination and renal function, kidney biopsy was not performed and he was advised for a regular follow up. His lab data in the past 5.5 years is demonstrated in table 1.

His physical examination on his last visit 8/03/2017: his body weight was 59 kg, Bp: 120/80 and his lab data is demonstrated on the last row of the above table.

In this child with a preceding upper respiratory infection, elevated ASO titer, depressed C_3 level, typical nephritis and spontaneous resolution in a short period of time, the first differential diagnosis is acute post streptococcal glomerulonephritis [1,3,4]. The unusual finding in this case is persistent hypocomplementemia which is expected to normalize within 8 weeks. Persistent hypocomplementemia beyond the 8 weeks from the initiation of the disease is an indication for kidney biopsy, keeping in mind other important alternative differential diagnoses such as membranoprolifeative glomerulonephritis and lupus nephritis.

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Table 1: summary of laboratory data in a 9year old boy with APSGN.

Date	Hb gm/dl	ESR mm/hr	BUN mg/dl	Cr mg/dl	ASO Todd unit	C_3 gm/l	C_4 gm/l	NO RBC in UA
29/11/2011	10.5	25	25	0.9	400	0.3	0.16	RBC
5/01/2012	10.8	15	15	0.8	400	-	-	25-30
27/03/2012	11	-	20	0.8	200	0.6	0.3	0-1
21/06/2012	-	-	-	-	-	0.65	0.25	-
23/08/2013	10.9	21	11	0.8	100	0.77	0.24	0-1
25/11/2014	10.2	11	21	0.9	-	0.7	0.18	
26/04/2015	11.3	3	12	0.8	800	0.58	0.14	0-1
18/08/2015	10.5	2	11	0.8	400	0.62	0.15	2-3
27/08/2016	13.2	3	12	0.9	1600	0.58	0.1	0-1
7/03/2017	13.1	3	11	0.8	400	0.76	0.16	2-4
4/07/2017	12.8	1	15	0.9	400	0.58	0.12	0-1

In this case, since the clinical examination, renal function tests and also urinalysis was entirely normal, I decided to follow him without performing the kidney biopsy. Keeping in mind that if membranoproliferative glomerulonephritis is considered as a differential diagnosis, with the mentioned clinical and laboratory findings he would not need any treatment.

The unusual finding in this case was persistent depression of C3 complement, in a regular search in PubMed three similar cases with prolonged hypocomplementemia was found but one of them with longer follow up was a case with mild form of dense deposit disease in association with streptococcal infection [9-11]. The other abnormal finding in our case is high Antistreptolysin O titer that after a decline again has raised to very high levels without recurrence of nephritis. This may be defined with infection with other serotypes of streptococcal infection.

The other shortage in this case is the lack of knowledge about his C3 before the onset of his nephritis but in cases with congenital C3 deficiency, different types of recurrent infections are expected [12-15], which was not observed in our case.

In conclusion the presented patient is unique in the literature for persistence of hypocomplementemia following a typical clinical course of APSGN without any clinical and other laboratory abnormality.

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