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PRESENTING FEATURES AND COMPLICATIONS OF ACUTE GLOMERULONEPHRITIS

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SUMMARY

A prospective study done on 42 patients (27 male (64.2%) and 15 - Female (35.7%) with Acute post Streptococcal glomerulonephritis (APSGN) during three years in pediatric hospital in Mosul Examination and investigations were done on all patients, including general urine examination (G.U.E) Complete Blood picture (C.B.P), antistreptolysine O-titer (A.S.O), serum creatinine, blood urea, Throat swab for culture and sensitivity, Immunological test and chest roentgenogram, presenting Features and Complications were studied.

The common presenting features were gross hematuria which forms 73.8% and common presenting sign were puffiness of the face which form 88.0% and the main complication was hypertension which form 40.4% cases. All patients discharged well from the Hospital.

INTRODUCION

Acute post infectious glomerulonephritis is the most common cause of acute glomeulonephritis in childhood, it is most often a complication of an infection due to group A - Beta hemolytic streptococcal, the most common types are 12, 49 in addition to type 2, 4,25 less commonly.[1,4]

The site of infection is frequently pharynx in which cases streptococcus is most likely to be type 12, also 1 and 4, the second most common site of infection is the skin due most frequently to type 49, also type 2, 25, 57, 60 less frequently.

Acute glomerulonephritis may follow also other bacterial infections, staphylococcal (shunt infection), Bacterial endocardities, pneumococcal infection, klebsiella species, meningococcus species, salmonella typhi.[3,4]

In addition to other infectious agents are mycoplasma pnemonia, coxsackie virus, Echovirus, Epstien Barr virus. Hepatites B virus, influenza virus, mumps virus, robeola virus, cytomegalovirus, spirochetal infection including treponema pallidum and leptospirosis.[5]

It can follow also a parasitic infestations including plasmodium malariae and falciparum, toxoplasmosis, schistosomiasis, filariasis, trypanosomiasis in addition to rickettsial infection as scrub typhus and fungal infection as coccidioides immitis.[5]

In epidemics of nephritogenic pharyngitis, nephritis may develop in up to 20% of children but the usual incidence of nephritis following streptococcal infection is less than 1%.[3]

The interval between infection and onset of nephritis varies between 2-3 days to more month, it averages 10 - 14 days.[7]

APSGN affect all ages[7] but the highest incidence is in the early school years and it is unusual in children less than 3 years old.[8] Gross histology shows enlargement of involved kidney with punctate cortical hemorrhage.[2]

Pathogenesis

Renal biopsies performed early in the course of post streptococcal glomeulonephritis reveal diffuse endocapillary proliferative glomerulonephritis with infilteration of the glomeruli with polymorphnuclear leukocytes and monocytes, glomeruli capillary walls are delicate and free of necrosis, occassional discrete protienaceous deposits projecting from the outer aspect of the capillary wall toward a urinary space (hump) may be recognised by light microscopy crescents may involve a few glomeruli.[9]

These humps generally held to be composed of immune complexes immunoglobulins and complement were readily demonstrated in these, but specific streptococcal element was difficult to identify recently however it has been shown that nephritogenic strains of streptococci form a highly cationic extracellular protien (the nephritis strain associted protien) which has an affinity for the glomerular basement in the kidney by the addition of specific antibody and complement to the deposited cationic eventially leads to fibrosis with the loss of function of variable number of glomeruli.

Clinical Feature

The onset of nephritis is typically heralded by puffiness of the face and dark coloured urine, initially the oedema is most prominant around the eyes when the child awakes in the morning but later is spread to involve the lower extermities and abdominal cavity as well, the amount of oedema is only moderate and may not be appreciated if the physician is unfamiliar with child's normal appearance, the urine is scanty in amount, cloudy and brown rather than red in colour, parents describe it's appearance as being like tea or cola.

There may be some mild suprapubic discomfort and dysuria vague abdominal pain and pain across the back are common. High temperature is uncommon but thus occur in some cases a typical mode of onset may be with either severe circulatory congestion or cardic failure when the patient present with shortness of breath, moist rales in the chest or pleural effusion with the finding of cardiomegally and hepatomegaly the finding of hypertension, oedema and gross hematuria in association with other signs of cardic failure make the diagnosis of acute nephritis almost certain or the onset can be with blurred vision, severe headaches, altered mental status or convulsion due to hypertensive encephalopathy.

On physical examination children with classic type of acute nephritis appear pale and lethargic with poor appetite there may be evidence of prior streptococcal infection such as residual exudate on the tonsils, enlarged cervical nodes post scarlatiniform peeling or healing pyoderma.

Ascitis is often difficult to be detected but better appreciated by abnormal fullness of abdominal contour when the child is supine, mild to moderate hypertension are usually ranging (from 120 - 180 mmHg) systolic and diastolic pressure of (80 - 120 mmHg). Ophthalmoscopic examination is not remarkable and the reminder of the physical exmination is usually normal.[5]

A wrong diagnosis of hemorrhagic cystitis is frequently made in clinically mild cases of nephritis in which there is onset with hematuria but without hypertension or oedema still mider cases may be detected by the finding of microscopic hematuria and few casts in rotein urin analysis, the most useful pointers to a correct diagnosis of nephritis in these patients are:[7]

1. Clinical, bacteriological or serologic evidence of previous streptococcal disease in the patient, his siblings or house hold contacts.
2. A lowered concentration of C3 in the serum.
3. Red blood cell casts are found in the urine.

Laboratory Findings

- **Urine analysis:** The urine is often diminished in volume and with colour ranging from smokey grey to reddish brown, protienuria is usually proportional to the degree of hematuria and in the range of a trace to +2 but protien excretion may be greater and exceed $2\ gm/m^2$ of body surface area/day. Microscopic examination of the urine usually reveals numerous red and white blood cells and there is also red blood cell casts which are evidence of glomular bleeding.
- **Hematological abnormalities:** shows mild normochromic normocytic anemia, the other blood components are usually normal although there may be some elevation of white blood cells count and ESR.
- **Tests for renal function**: significant number of children will have elevation of blood urea nitrogen (BUN), and serum creatinine will be normal but the glomerular filteration rate is usually depressed and few children will have some azotemia with the development of metabolic acidosis and hyperkalemia.
- **Bacteriological and serological examination :** group A - Beta hemolytic streptococci may be cultured from nasopharynx or from the skin lesion, if direct cultural evidence of post streptococcal infection is lacking serological test to demonstrate an immune respose to streptococcal antigen should pursued, elevation of antibody titers to streptolysin - o (A.S.O) will be seen within 10 - 14 days following streptococcal infection in most patients and will remain elevated for several months. Unfortunately, the response of A.S.O titer following skin infections with streptococcal is poor, other tests of antibody titer notably antihyalurinidase and antideoxy - ribonuclease B will generally be elevated after streptococcal infection regardless of the site.
- Immunological test an important and very consistent finding in APSGN is lowering of third complement C3.
- serum C3 level will be below normal at onset of symptoms in 80 - 90 % of cases, while depression of C3 level is common in APSGN, C4 is less consistently lowered. (10)
- **Radiographic studies:** chest roentgenogram may show the heart is generally of normal size and contour although mild cadiomegally may be seen, in contrast to the relatively normal cardic silhouette evidence of pulmonary fluid overload may be marked by unilateral or bilateral pleural effusion are not un common.

X-ray examination of the abdomen will usually show haziness suggestive of ascitis.

Radiological and ultrasound evaluation of the kidney will not reveal findings specific for APSGN.

Differential Diagnosis

The clinical course and long term prognosis of APSGN may be quite different from other forms of post infections nephritis, in addition other renal disease may present in a manner quite similar to APSGN.

For this reason it is important to distinguish between APSGN and others conditions which may mimic it, these conditions are:[3]

1. Any form of chronic glomerulonephritis acute exacerbation.
2. Henoch-Schoenlein (Anaphylactoid) purpura with renal involvment.
3. Idiopathic hematuria

a. Benign =
b. Recurre =
c. IgA nephropathy.

4. Hereditary nephritis (Alports syndrome).
5. Rarely systemic lupus erythematosus.

Management of the Patient with Acute Nephritis

The treatment is supportive, it seems reasonable to reccomend bed rest until the sign of glomerular inflammation and circulatory congestion (primarily hypertension) subside but prolonged forced period of inactivity are of no demonstrable benifit in the healing process.

Fluid retention, circulatory congestion and oedema may be treated with sodium and fluid restrictions or loops diuretics (furosemide). Diuresis alone will often ameliorate mild to moderate hypertension, if severe hypertension is present vasodilator drugs such as nitro prusside, hydralazine or diazoxide may be useful for a cute hypertensive emergency a combination of hydralazine and reserpine given intramuscularly has long been popular in pediatric practice.

Recent reports indicate that intravenous diazoxide dramatically lowers the blood pressure because of direct effect on arteriolar smooth muscle within few minutes of injection Methyl dopa administered parenterally has an onset of action approximately two hours but it may paradoxically cause a cute rise of blood pressure.

Encephalopathy and pulmonary congestion will generally improve with lowering of blood pressure and the relief of circulatory overload.

Treatment with ion exchange resin and / or dialysis may be required for cases with severe oliguria, fluid overload and hyperkalemia.

Mild protein restriction is desirable for azotemic patients, 7 - 10 days course of antimicrobials (e.g. penicillin or erythromycin should be given if streptococcal infection is documented[1,3]

Prognosis

In cases of mild or moderate severity symptoms disappear spontaneously in few days, urinary abnormalities decrease progressively during the following weeks or months and seldom present for more than 12 months.

Complete recovery occurs in more than 95% of children with APSGN,1% or less will show crescentic glomerulonephritis and go into early uraemia and only 1 - 2 % usually older children will show uraemia later.[6]

Follow Up

The frequency of need for follow up, examination for patients with ASPGN will depend to great extent on the individual patient and the severity of the disease, we generally like to see patients 4 - 6 weeks interval for the first 6 months after onset of nephritis. Measurement of blood pressure and examination of urine for red cells and protien will be the best measure to evaluate recovery. After 6 months if hematuria and protien uria have resolved, a yearly urine analysis and blood pressure determination should be sufficient.[1]

PATIENTS AND METHODE

A prospective studied done on 42 patients, with acute post streptaccal glomerulonephritis (APSGN) they were of age group ranging between 3-13 years, history was taken and physical examinations were carried on each of them.

All of them were investigated and the investigations done were, GUE, CBP, BUN, serum creatinine, ASO titer, throat swab for culture and sensitivity Immunological test and Chest x-ray. The presenting feature and complication were analysed on the patients.

RESULTS

Male were more commonly affected than female (1.8:1). The main age group affected was between 3 - 7 years, it forms about 70% of the patients.

History of recent tonsilitis was found in 32 patients (76.1%) 3 - 21 days before admission, 6 patients (14.2%) gave history of skin infection in form of impetigo contagiosa, infected wound and minor abscess on the foot 2 - 4 weeks before admission.

The commonest complaints for those patients at admission was puffiness of the face found in 22 patients 52.3%, 15 patients (35.7%) present with generlized oedema, 3 patients 7.1% with dyspnoea, 1 with abdomenal pain 2.3% and 1 with hematuria 2.3%.

Symptoms, Signs, the laboratory data and the complications were summarized in tables (1,2,3,4,5,6,7,8).

Table -1: The presenting symptoms in 42 patients with APSGN.

Symptoms	No. of patients	%
Gross hematuria	**31**	**73.8**
Oliguria	**29**	**69.0**
Fever	**19**	**45.2**
Dyspnoea	**17**	**40.4**
Abdominal pain	**6**	**14.2**
Dysuria	**2**	**4.7**

Table-2: The presenting sign in 42 patients with APSGN.

Sign	No. of patients	%
Puffiness of the face	**37**	**88.0**
Hypertension (mild, moderate, severe)	**34**	**80.9**
Leg oedema	**30**	**71.4**
Pallor	**25**	**59.5**
Generlized oedema	**28**	**66.6**
Hepatomegaly	**2**	**4.7**
Ascities	**7**	**16.6**

Table-3: Haematological findings in 42 patients with APSGN.

Haematological data	No. of patients	%
Haemoglobine (7 - 11 gm/dl)	**30**	**71.4**
(12 - 13 gm/dl) White blood cells	**12**	**28.5**
(4.5 - 15.5) x10^9/L	**39**	**92.8**
(16 - 28) x10^9/L Plateles	**3**	**7.1**
ASO titer		
(0 - 200) T.U.	**13**	**30.9**
(400 - 600) T.U.	**29**	**69.0**
Platlet normal	**40**	**100**

Table - 4: Finding of general urine analysis done on 42 patients with APSGN.

Urine analysis	No. of patients	%
R.B.Cs full field	**15**	**35.7**
30 - 40 /HPF	**4**	**9.5**
20 - 30 /HPF	**11**	**28.1**
10 - 20/HPF	**12**	**28.5**
Pus cells 30 - 40/HPF	**3**	**7.1**
20 - 30/HPF	**7**	**16.6**
10 - 20/HPF	**12**	**28.5**
0 - 10/HPF	**20**	**47.6**
Protien No protein	**2**	**4.7**
mild – moderate (trace, +, ++)	**31**	**73.8**
Heavy protienuria(+++, ++++)	**9**	**21.4**
Granular Cast	**42**	**100**

Table-5: Evaluation of renal functions in 42 patients with APSGN.

Renal function tests	No. of patients	%
Blood urea (10 - 20 mg/dl	**10**	**23.8**
>20 mg/dl	**32**	**76.1**
Serum creatinine (0.2 - 0.7) mg/dl	**36**	**85.7**
>0.8 mg/dl	**6**	**14.2**

Table -6: Results of throat swab for culture and sensitivity in 42 patients with APSGN.

Throat swab result	No. of patients	%
Streptococcus viridans	**10**	**23.8**
No pathogen isolated	**30**	**71.4**
Gram +ve diplococcus isolated	**2**	**4.7**

On 30 patients chest X-ray done, 19 of them were normal 63.3.%, 11 of them were abnormal that is 36.6% of cases :
9 show evidence of pulmonary congestion.

2 show pulmonary oedema and cardiomegaly.

Immunological test Lowering serum C3-Level in 35 of patients (83.3%), 7 of patient (16.7%) was normal. Serum C4-level normal in All Cases.

Complications

Among 42 patients 17 patients developed moderate to severe hypertension which form 40.4%. 9 patients have evidence of pulmonary congestion clinically and radiologically which form 21.4 %. 2 patients developed acute pulmonary oedema and heart failure probably reflect fluid overload (1), and these complications are listed in table 7.

Table 7: Complications noted in 42 patients with APSGN.

Complications	No. of patients	%
Acute pulmonary oedema and heart failure	**2**	**4.7**
Evidence of pulmonary congestion clinically and radiologically	**9**	**21.4**
Moderate to severe hypertension	**17**	**40.4**
Acute renal failure	**0**	**0**

DISCUSSION

APSGN is the commonest cause of acute glomerulonephritis in childhood (5) post streptococcal glomerulonephritis is more prevalent during the school age years with an average age of onset of 6 - 7 years, clinical manifestation are rare before three years, the disease also occurs during adolescent and adulthood.[5]

This study proved that APSGN is more common in male than female as most series report, this is difficult to explain because the suceptibility to streptococcal infection is apparently not sex related and rheumatic fever does not show any prediliction for male. The true incidence is unknown because the majority of cases are probably sub clinical, it was found that 50% of cases with APSGN, during epidemic were asymptomatic.[3]

The most common nephritogenic strain is type 12 which is responsible for streptococcal pharyngitis and type 49 responsible for streptococcal skin infection and APSGN after skin infection is less common than after pharyngitis as in our study (2). The lated period between streptococcal infection of pharynx is 3 days - 3 weeks average 10 days while is more prolonged following the skin infection with average about 2 weeks.[10]

In this study the majority of the patients with post streptococcal nephritis present during winter and early spring and following streptococcal pharynagitis (76.1%), while (14.2%) following skin infection, while in the warmer part of the world acute nephritis related to streptococcal pyoderma is more common in the summer months when insect bite and abrasions on bare feet are frequent, surgical wounds, skin lesion of chicken pox, scabies and atopic dermatitis occasionally serve as the portal of entery of streptococcal.[5]

All the patients in this study gave no history of previous similar attack although in other studies 5 patients out of 123 patients show 2 attacks (7) but recurrence is extremely rare.[8]

In this study the main presenting symptom was gross hematuria form 73.8%, the main presenting sign was puffeness of the face form 88.0% and the main complication was moderate - severe hypertension which found in 17 patients (40.4%). The percentage of hypertension in this study is as follows:

Table 8: Percentage and degree of hypertension detected in this study.

Degree of hypertension	No. of patients	%
Mild hypertension BP 130/90 mmHg	**17**	**40.4**
Moderate hypertension BP > 140 - 150 / 100 mmHg	**15**	**35.7**
Severe hypertension 2 BP > 120 mmHg Diastolic	**2**	**4.7**

* Hypertension in children is defined as being 2 standard deviation above the mean for age and sex for above 95th percentile for age,[3,4] Those children with mild hypertension respond only to bed rest, fluid and salt restriction. Those with moderate and severe hypertension were treated by diuretics (frusemide), hydralazine I.V. In addition to bed rest fluid and salt restrictions in all our patients the blood pressure reaches normal level within 3 - 7 days, according to other studies it seems important to treat all children who have diastolic blood pressure persistently in excess of 90 mmHg in order to reduce the incidence of morbidity in those patients.[4] The laboratory results in this study reveals anemia which was mild normochromic normocytic found in 71.4% of cases which is mainly dilutional parallel to the degree of fluid retention, there is also some evidence to suggest that alteration in red cell production or break down may contribute to the decrease of hematocrit.[3]

Leukocytosis is found in 7.1% of the cases 76.1 % of patients show elevation of blood urea nitrogen and 14.2% show elevation of serum creatinine A.S.O titer was raised in 69% of cases, Serum C3-level was lowerd in 83.3% of cases renal biopsy and magnetic resonance Imaging was not done because there was no need to do it in this study.

CONCLUSIONS

All the 42 patients discharged well from the hospital after improvement of their clinical symptoms and increasing their urine output and renal function evaluation before discharge was normal, accordingly the prognosis was good, but the long term of prognosis depend on the follow up of these cases for the development of hypertension and deterioration of renal function.

Differences in the severity of initial illnesses possibly are related to the strain of streptococcus implicated, may affect the ultimate outcome. It can probably be said that greater than 90% of children with APSGN should be expected to recover without significant alteration in the renal function.[3]

In comparison of the long term of follow up study of the patients with APSGN apparently the prognosis associated with epidemics is excellent, children with sporadic APSGN with no underling renal disease or persistent nephrotic syndrome or severe impairment of renal function initially also have an excellent outcome.[4]

RECOMMENDATION

APSGN disease of all ages but it is most common in early chilhood, sub clinical cases specially among family member should be evaluated to estimate the true incidence of the disease.

It is reasonable to try to irradiate the B-hemolytic streptococci for a small community (i.e. a family) in which a case of glomerulonephritis has occurred. Organism is highly sensitive and easily removed of long acting penicillin. There is no way of preventing acute glomerulonephritis developing into persistent glomerulonephritis accordingly treatment is directed solely at saving the patient's life and shortening the duration of acute attack.

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