glomerulonephritis. The child was started on amoxicillin/clavulanate failure secondary to hypertension were also in favor of acute hypertension post-streptococcal complications were suspected. High upper respiratory tract infection and features of edema, hematuria, investigations were otherwise normal. With the history of antecedent showed pulmonary congestion with cardiomegaly. Other investigations showed: haemoglobin 9.5 g/dl, WBC 23,870/mm3 shaped haemorrhages were present in both eyes on fundus examination. Flame on auscultation S1S2 soft, S3 gallop present. On Respiratory examination at admission weight recorded was 57 kg and height of the child was 167 cm. On General examination, the child was conscious and oriented, pale and anasarca present. Systemic cardiac examination revealed apex beat at left 5 intercostal space at the midclavicular line. On auscultation S1S2 soft, S3 gallop present. On Respiratory examination, bilateral basal crepitations were present. Flame shaped haemorrhages were present in both eyes on fundus examination. Other systemic examination was normal. Supportive treatment was given and started further evaluation. Laboratory investigations showed: haemoglobin 9.5 g/dl, WBC 23,870/mm3 (Neutrophils-88%). C reactive Protein – 48 mg/dl. Haematuria was confirmed by the urinalysis – 418 RBCs/hpf, with dysmorphic RBCs and child's general condition improved with resolution of hematuria and child's general condition improved with resolution of hematuria (RBC-/hpf) and edema decreased as evidenced by the reduction in weight (53 kg after one week). Features of Congestive cardiac failure resolved. The serum antistreptolysin O titre was negative after treatment was initiated Prednisolone and with Aspirin at the dosage of 75 mg/kg/day was and secondary prophylaxis with intramuscular benzathine benzylpenicillin (at the dose of 12,00,000 IU) started. Fever subsided, hypertension under control with B.P – 110/70 mm Hg and child's general condition improved with resolution of hematuria and also different bacteriology features. Their concurrent development in the same patient is known and has been described in the literature, coincidental occurrence of these two diseases preceded by a documented streptococcal pharyngitis is still an opportunity of scholastic significance for clinicians and students. PSGN and ARF have different epidemiology, immunology, and also different bacteriology features. Their concurrent development in the same patient is known and has been described in the literature, but these are rare presentations. The serotypes of group A Streptococci are divided into those with rheumatogenic and nephritogenic potential on the basis of the complication sequelae induced by them.

DISCUSSION:
As discussed earlier, PSGN and ARF are both postinfectious non-suppurative sequelae caused by Group A beta hemolytic Streptococcus infections. The presentation of clinical and laboratory features of these two diseases preceded by a documented streptococcal pharyngitis is still an opportunity of scholastic significance for clinicians and students. PSGN and ARF have different epidemiology, immunology, and also different bacteriology features. Their concurrent development in the same patient is known and has been described in the literature, but these are rare presentations. The serotypes of group A Streptococci are divided into those with rheumatogenic and nephritogenic potential on the basis of the complication sequelae induced by them.

The pathogenesis of ARF is well known. ARF is known to be caused by the mechanism of molecular mimicry. Some streptococcal antigens, like M protein and hyaluronate capsule, have identical antigen epitopes similar to human tissue proteins in the myocardium, brain, and joints. This way, they cause inflammation by inducing cross-reacting autoantibodies against human tissue proteins. The immunopathogenesis of PSGN is not yet completely understood. The most extensively studied mechanisms suggest circulating immune complex deposition, in situ immune complex formation, and molecular mimicry between streptococcal and human glomerular proteins, with consequent autoimmune response results in PSGN.
Though both ARF and PSGN are secondary to immunological injury, their coincidental occurrence is not usually seen, and there is no explanation yet for this interesting occurrence. It might be explained that some of the streptococcal strains had both nephritogenic and rheumatogenic features.

In 2003 study and literature review by Kula et al., stated that most of the previously reported cases of this condition were adults. They studied the characteristics of the pattern of occurrence in the previously reported cases. Of the nine cases, they studied five were male, and four were female, aged 3.5-16 years (mean 11.27 ± 1.42 years). All patients were treated with diuretics and salicylate and given secondary prophylaxis with intramuscular benzathine penicillin. In 4 of the reported cases of co-existent APGN and ARF acute rheumatic fever was the first feature which was followed by glomerulonephritis. Acute glomerulonephritis was the first feature in the remaining of the cases. However, some of these cases had a longer intermittent period between both features and even symptoms for both APSGN and acute rheumatic fever can present at the same time.

In a study done by Vardi et al., it was found that mitral insufficiency is frequently associated with patients in PSGN. Much remains to be learned about the complex interrelationships between streptococcus and host that result in the occurrence of rheumatic fever, glomerulonephritis, or, on occasion, both. Progress will undoubtedly be slow in those with this coincidental occurrence. Consequently, physicians should be careful about this unusual condition so as to initiate adequate prophylaxis in these patients. Early recognition of both the disease process is important to prevent future morbidity.

REFERENCES: