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INDIAN ACADEMY OF PEDIATRICS
INFECTIOUS DISEASES CHAPTER

FAQs *in* PEDIATRIC INFECTIOUS DISEASES



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2 Group A Streptococcus

Santanu Bhakta

1. What is the epidemiology of Group A Streptococcus (GAS) infection?

Overall, the epidemiology of GAS infection is complex. Group A streptococci are gram-positive coccoid-shaped bacteria that tend to grow in chains. Humans are the natural reservoir of GAS. It is highly communicable and can produce disease in normal individual of all ages. While disease in neonates is uncommon, probably due to transmitted maternal antibody, this organism commonly produces two main types of diseases.

Pharyngeal infection

Pharyngeal infection is very common in children between 5 to 15 years of age. They can spread via salivary droplets and nasal discharge and close proximity helps in transmission. Incubation period for pharyngitis is 2–5 days. If untreated, GAS has the potential to be an important upper respiratory tract pathogen causing outbreaks, especially in day care settings like schools, military barracks, crèche, etc. Chronic pharyngeal carriers of GAS rarely transmit to others.

Skin infection

Streptococcal skin infection (impetigo, pyoderma) occurs throughout the year in warmer climates. GAS cannot penetrate intact skin and thus, colonization of healthy skin by GAS precedes development of impetigo. Then it invades the breached skin like traumatic wounds, insect bites, burns, etc. to produce impetigo. Although, impetigo-serotypes may colonize throat, spread via skin to skin is usual and not via respiratory tract. Fingernails and perianal regions harbor GAS and important in spreading impetigo as we see multiple cases in same family. Both pharyngitis and impetigo occur in crowded homes and poor hygienic conditions.

Invasive GAS infections

The incidence of severe invasive GAS infections including bacterimia, streptococcal toxic shock syndrome, necrotizing fasciitis has increased in recent years and attacks very young and older persons. Risk factors

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include varicella, HIV infection, intravenous drug use, chronic pulmonary and cardiac disease. Severe invasive disease rarely follows GAS pharyngitis and is believed to be mostly through skin and mucous membrane though the portal of entry is unknown in 50% cases. Two types commonly associated with pharyngitis, rarely cause skin infections and those commonly associated with skin infections, rarely cause pharyngitis. A few of the pharyngeal strains (M type 12) have been associated with glomerulonephritis, but more of the skin strains (M types 49, 55, 57 and 60) have been considered nephritogenic. A few of the pharyngeal serotypes, but none of the skin strains, have been associated with acute rheumatic fever. M types 1, 3, 12, and 28 have been the most common isolates from patients with shock and multiorgan failure.

2. What are the common manifestations of Group A Streptococcus infection?

The most common infections caused by GAS, involves the respiratory tract and skin and soft tissues. In upper respiratory tract infection (URTI), it is an important cause for acute pharyngitis and pneumonia is rare. In skin and soft tissue infection, it is responsible for impetigo. Other manifestations are as follows:

Scarlet fever

Scarlet fever, caused by erythrogenic toxin-producing GAS, manifested by fever with characteristic rash appearing 24–48 hours after fever along with pharyngitis. Age distribution, mode of transmission and other epidemiological features are same as GAS pharyngitis.

Impetigo

Impetigo (or pyoderma) is of two clinical types; bullous, and more common, nonbullous. It is a superficial infection of skin occurring mostly in face and extremities and regional lymphadenitis is common. Bullous impetigo is less common and occurs mostly in neonates and young infants. Face, buttocks, trunk and perineum are mostly affected.

Erysipelas

Erysipelas is a relatively rare acute GAS infection of the deeper layer of skin and underlying connective tissue. Onset is abrupt and affected skin is swollen, red and very tender with systemic manifestation of fever.

Perianal dermatitis

Perianal dermatitis is a distinct clinical entity with well-demarcated perianal erythema, anal pruritus, painful defecation and blood-streaked stools. They are very tender but systemic manifestations are unusual.

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Vaginitis

Vaginitis is a common in prepubertal girls manifested as serous discharge, marked erythema and irritation of vulvar area with dysuria.

Severe invasive GAS infection

Severe invasive GAS infection is defined by isolation of GAS from a normally sterile body site. It includes three overlapping clinical syndromes. The first is streptococcal toxic shock syndrome. The second is GAS necrotizing fasciitis characterized by extensive local necrosis of skin and soft tissues. The third is the group of focal and systemic infections that do not meet the criteria for toxic shock syndrome or necrotizing fasciitis and includes bacteremia with no identified focus. It may be in the form of meningitis, pneumonia, peritonitis, puerperal sepsis, osteomyelitis, suppurative arthritis, myositis, and surgical wound infections.

3. What is streptococcal toxic shock syndrome?

It is a type of severe invasive disease produced by GAS and differentiated from other types of invasive GAS infections by the presence of shock and multiorgan system failure early in the course of the infection. Following are the criteria:

Case definition of streptococcal toxic shock syndrome (streptococcal TSS)

I. Isolation of group A Streptococcus

A. From a sterile site (blood, CSF, Peritoneal fluid, tissue biopsy specimen)
B. From a nonsterile body site (throat, sputum, vagina, open surgical wound, superficial skin lesion)

II. Clinical signs of severity

A. Hypotension
B. Clinical and laboratory abnormalities (requires two or more of the following) signs:
   i. Renal impairment
   ii. Coagulopathy
   iii. Liver abnormalities
   iv. Acute respiratory distress syndrome
   v. Extensive tissue necrosis, i.e., necrotizing fasciitis
   vi. Erythematous rash

Definite Case = An illness fulfilling criteria IA and IIA and IIB
Probable Case = An illness fulfilling criteria IB and IIA and IIB

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Pain is the most common initial symptom of streptococcal TSS, abrupt in onset and severe.

Fever is the most common early sign, although hypothermia may be present in patients with shock. Eighty percent of patients have clinical signs of soft tissue infection, such as localized swelling and erythema. Blood cultures are positive in 60% of cases. Renal impairment is very common and important component of streptococcal TSS. Persons of all ages are affected; most do not have predisposing underlying diseases. This is in sharp contrast to previous reports of GAS bacteremia, in which patients were either under 10 or over 60 years of age, and most had underlying conditions such as cancer, renal failure, leukemia, or severe burns, etc. The pathogenic mechanisms responsible for severe, invasive GAS infections, like streptococcal toxic shock syndrome is yet to be defined completely, but an association with streptococcal pyrogenic exotoxins A, B, and C has been suggested, which act as superantigens, and stimulate an intense activation and proliferation of T lymphocytes and macrophages resulting in the production of large quantities of cytokines. These cytokines are capable of producing shock and tissue injury, and are believed to be responsible for many of the clinical manifestations of severe, invasive GAS infections.

4. What are the suppurative and nonsuppurative complications of Streptococcal infection? Give a brief description of each.

Suppurative complication from GAS infection may occur due to spread of the infection to the adjacent structures because of inadequate treatment and can give rise to cervical lymphadenitis, peritonsilar abcess, retrophryngeal abscess, mastoiditis, sinusitis and otitis media.

The non-suppurative complications are acute rheumatic fever, acute poststreptococcal glomerulonephritis, poststreptococcal reactive arthritis and pediatric autoimmune neuropsychiatric disorders associated with *Streptococcus pyogenes* (PANDAS).

Acute rheumatic fever

Acute rheumatic fever is a nonsuppurative complication of group A beta hemolytic streptococcal (GABHS) sore throat. It usually occurs 2–4 weeks after streptococcal pharyngitis. It affects joints, skin, subcutaneous tissue, brain and heart. Except heart, all other effects are reversible, needing only symptomatic relief during the episodes. Cardiac complications are significant in absence of secondary prophylaxis and culminate into chronic and life-threatening valvular heart disease. Prevalence of acute rheumatic fever and rheumatic heart disease (RHD) in Indian population varies from 0.5/1000 to 11/1000 in various studies. Not all of the serotypes of GAS can cause rheumatic fever. Acute rheumatic fever can occur only after an infection of the upper respiratory tract. Patients with acute rheumatic fever almost always have serologic evidence of a recent GAS infection. Antimicrobial therapy that eliminates GAS from the pharynx also prevents initial episodes of acute rheumatic fever, and long-term, continuous prophylaxis that prevents GAS

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pharyngitis also prevents recurrences of acute rheumatic fever. Carditis and resultant chronic rheumatic heart disease are the most serious manifestations of acute rheumatic fever and account for essentially all of the associated morbidity and mortality.

Acute poststreptococcal glomerulonephritis (APSGN)

Acute glomerulonephritis can occur after a GAS infection of either the upper respiratory tract or the skin. This is a classic example of the acute nephritic syndrome characterized by the sudden onset of gross hematuria, edema, hypertension, and renal insufficiency. Acute poststreptococcal glomerulonephritis is one of the most common glomerular causes of gross hematuria in children and is a major cause of morbidity in GAS infection. Although epidemics of nephritis have been described in association with both throat (serotype 12) and skin (serotype 49) infections, this disease is most commonly sporadic. Early systemic antibiotic therapy for streptococcal throat and skin infections does not eliminate the risk of acute poststreptococcal glomerulonephritis. APSGN is most common in children aged 5–12 years and uncommon before the age of 3 years. The typical patient develops symptoms 1–2 week after an antecedent streptococcal pharyngitis or 3–6 weeks after a streptococcal pyoderma. Complete recovery occurs in more than >95% of children with APSGN. Recurrences are extremely rare.

Poststreptococcal reactive arthritis

It is a syndrome characterized by the onset of acute arthritis following an episode of GAS pharyngitis in a patient whose illness does not otherwise fulfill the Jones criteria for the diagnosis of acute rheumatic fever. Though it usually involves the large joints, in contrast to the arthritis of acute rheumatic fever, it may involve small peripheral joints as well as the axial skeleton and is typically non-migratory. In contrast to the arthritis of acute rheumatic fever, poststreptococcal reactive arthritis does not respond dramatically to therapy with aspirin or other nonsteroidal anti-inflammatory agents. A small proportion of patients with poststreptococcal reactive arthritis may go on to develop valvular heart disease. Therefore, these patients should be carefully observed for several months for the subsequent development of carditis.

Pediatric autoimmune neuropsychiatric disorders associated with streptococcus pyogenes (PANDAS)

PANDAS have been used to describe a group of neuropsychiatric disorders (particularly obsessive-compulsive disorders, tic disorders, and Tourette syndrome) for which a possible relationship with GAS infections has been suggested.

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5. What is the diagnostic test for Group A streptococcal pharyngitis?

Diagnostic studies for GAS pharyngitis are not indicated for children less than 3 years old because acute rheumatic fever is rare and the classic presentation of streptococcal pharyngitis are uncommon in this age group. Selected children less than 3 years old who have other risk factors, such as an older sibling with GAS infection, may be considered for testing.

Swabbing the throat and testing for GAS pharyngitis by rapid antigen detection test (RADT) and/or culture should be performed because the clinical features alone do not reliably discriminate between GAS and viral pharyngitis except when overt viral features like rhinorrhea, cough, oral ulcers, and/or hoarseness are present. In children and adolescents, negative RADT tests should be backed up by a throat culture. Positive RADTs do not necessitate a back-up culture because they are highly specific (>95%).

Anti-streptococcal antibody titers are not recommended in the routine diagnosis of acute pharyngitis as they reflect past but not current events.

Diagnostic testing or empiric treatment of asymptomatic household contacts of patients with acute streptococcal pharyngitis is not routinely recommended.

6. What is the treatment of streptococcal pharyngitis?

GAS is the most common bacterial cause of acute pharyngitis, responsible for 20–30% in children. Accurate diagnosis of streptococcal pharyngitis followed by appropriate antimicrobial therapy is important for the prevention of acute rheumatic fever; for the prevention of suppurative complications (e.g. peritonsillar abscess, cervical lymphadenitis, mastoiditis, and possibly, other invasive infections); to improve clinical symptoms and signs; for the rapid decrease in contagiousness; for reduction in transmission of GAS to family members, classmates, and other close contacts of the patient; to allow for the rapid resumption of usual activities.

Patients with acute GAS pharyngitis should be treated with an appropriate antibiotic at an appropriate dose for duration likely to eradicate the organism from the pharynx (usually 10 days). Based on their narrow spectrum of activity, infrequency of adverse reactions, and modest cost, penicillin or amoxycillin is the recommended drug of choice for those non-allergic to these agents.

Treatment of GAS pharyngitis in penicillin-allergic individuals should include a first generation cephalosporin (for those not anaphylactically sensitive) for 10 days, clindamycin or clarithromycin for 10 days, or azithromycin for 5 days (Table 1).

Clinicians caring for patients with recurrent episodes of pharyngitis associated with laboratory evidence of GAS pharyngitis consider that they may be experiencing more than one episode of bona fide streptococcal pharyngitis at close intervals, but they should also be alert to the possibility

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Table 1: Antibiotic regimens recommended for group A streptococcal pharyngitis

Drug, route	*Dose or Dosage*	*Duration or Quantity*
For individuals without penicillin allergy		
Penicillin V, oral	Children: 250 mg twice daily or 3 times daily; adolescents and adults: 250 mg 4 times daily or 500 mg twice daily	10 d
Amoxycillin, oral	50 mg/kg once daily (max = 1000 mg); alternate: 25 mg/kg (max = 500 mg) twice daily	10 d
Benzathine penicillin G, intramuscular	< 27 kg: 600,000 U: ≥ kg: 1,200,000 U	1 dose
For individuals with penicillin allergy		
Cephalexin, oral	20 mg/kg/dose twice daily (max = 500 mg/dose)	10 d
Cefadroxil, oral	30 mg/kg once daily (max = 1 g)	10 d
Clindamycin, oral	7 mg/kg/dose 3 times daily (max = 300 mg/dose)	10 d
Azithromycin, oral	12 mg/kg once daily (max = 500 mg)	5 d
Clarithromycin, oral	7.5 mg/kg/dose twice daily (max = 250 mg/dose)	10 d

Source: Shulman T, et al. Clinical Practice Guideline for the Diagnosis and Management of Group A Streptococcal Pharyngitis: 2012 Update by the Infectious Diseases Society of America by Stanford

that the patient may actually be a chronic pharyngeal GAS carrier who is experiencing repeated viral infections. If a physician suspects that "ping-pong" spread of infections is the explanation for multiple recurrent episodes of infections within a family, it may be helpful to obtain throat swabs from all family contacts simultaneously, and to treat those for whom culture or RADT results are positive. There is no credible evidence that family pets are reservoirs for GAS pharyngitis or that they contribute to familial spread.

7. How to manage acute rheumatic fever?

Management of acute rheumatic fever consists of diagnosis and treatment.

Diagnosis

Because no clinical or laboratory finding is pathognomonic for acute rheumatic fever, Jones criteria (Table 2) is used to aid in diagnosis and to limit over-diagnosis. The criteria, revised in 1992 by the American Heart Association (AHA) are intended only for the diagnosis of the initial attack of acute rheumatic fever and not for recurrences. Diagnosis is based on recognition of major and minor criteria supported by evidence of preceding streptococcal infection.

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Table 2: Diagnosis of rheumatic fever

Clinical and laboratory criteria	*Supportive evidence of preceding streptococcal infection (essential except for diagnosis of Chorea)*
Major criteria (more specific) • Carditis • Polyarthritis • Chorea • Subcutaneous nodule • Erythema marginatum	Antistreptolysin O ASO titer: >333 unit for children and 250 for adults. Anti-deoxyribonuclease B (normal values AntiDNase B titer 1:60 unit in preschool, 1:480 units in school children and 1:340 in adults)
Minor criteria (less specific) • Fever • Polyarthralgia • ESR. CRR polvmorphouuclear leukocytosis (follow standard laboratory values) • * ECG prolonged PR interval	**History of (within previous 45 days)** • streptococcal sore throat • scarlet fever • positive throat culture • positive rapid streptococcal antigen detection test

* Normal upper range—PR Interval: 3–12 years: 0.16 sec;12–14 year: 0.18 sec; >17y:0.20 sec

Source: Consensus Guidelines on Pediatric Acute Rheumatic Fever and Rheumatic Heart Disease; Working group on Pediatric acute Rheumatic fever and Cardiology chapter of IAP *These Guidelines were formulated at National Consultative Meeting on 20th May, 2007, IMA Hall, New Delhi, under IAP Vision 2007.*

First episode: Two major or one major and two minor criteria plus supportive evidence of previous Streptococcal throat infection.

Recurrence in a patient without established heart disease: Two major or one major and two minor criteria plus supportive evidence of previous streptococcal throat infection.

Recurrence in a patient with established heart disease: Two minor criteria and supportive evidence of previous streptococcal throat infection.

Rheumatic chorea and insidious onset rheumatic carditis: No requirement of other major manifestations or supportive evidence of streptococcal sore throat infection.

Treatment

According to clinical status, treatment for pain relief should be given (codeine or paracetamol till diagnosis is confirmed and aspirin after the diagnosis is confirmed). Hospitalization is needed for moderate to severe carditis, severe arthritis or chorea. Rest is individualized according to symptoms. For arthritis, rest for two weeks is adequate. Carditis without congestive heart failure (CHF) needs 4–6 weeks of rest. In cases of CHF, rest must be continued till the CHF is controlled. Appropriate diet is a must for a growing child with cardiac involvement. Total duration of anti-inflammatory therapy after the diagnosis of acute rheumatic fever is established, must be 12 weeks. Aspirin

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and steroids are primarily used to control inflammation. Naproxen and methylprednisolone can be used alternatively. Mild chorea is treated with quite environment, and sedatives like oral phenobarbitone or diazepam. If there is no response, then one may use haloperidol (0.25–0.5 mg/kg/d), sodium valproate (15 mg/kg/day), or carbamazepine (7–20 mg/kg/d) may be used.

Management of cardiac complication should be done according to the case (CHF, atrial fibrillation, valvular heart disease, etc.).

8. What are the guidelines for prophylaxis of acute rheumatic fever?

Secondary prevention of rheumatic fever is defined as the continuous administration of specific antibiotics to patients with a previous attack of rheumatic fever, or documented rheumatic heart disease (RHD). The purpose is to prevent colonization or infection of the upper respiratory tract with group A beta-hemolytic streptococci and the development of recurrent attacks of rheumatic fever. Secondary prophylaxis should be started only after establishing the diagnosis of acute rheumatic fever. Isolated ASO titer is not a criteria to start secondary prophylaxis. After surgery or intervention, secondary prophylaxis should be continued (Table 3).

Table 3: Drugs recommended for secondary prophylaxis

Drugs	*Dose*	*Sore-throat treatment (duration)*	*Secondary prophylaxis (interval)**
Benzathine Penicillin G (deep 1M inj)	1.2 million unit (>27 kg) after sensitivity test (AST) 0.6 million unit (<27 kg) (after sensitivity test) Contraindication: Penicillin allergy	single dose** single dose**	21 d 15 d
Penicillin V (oral)	Children: 250 mg qid Adult: 500 mg qid Contraindication: Penicillin allergy	10 d 10 d	twice a day twice a day
Azithromycin (oral)	12.5 mg/kg/day once daily	5 d	not recommended
Cephalexin (oral)	15–20 mg/kg dose bid	10 d	not recommended
Erythromycin (oral)	20 mg/kg/dose max 500 mg Contraindication: Liver disorder	not recommended	twice a day

** see text for duration of secondary prophylaxis and references*

*** only one dose is sufficient for GABHS pharyngitis*

Source: Consensus Guidelines on Pediatric Acute Rheumatic Fever and Rheumatic Heart Disease; Working group on Pediatric acute Rheumatic fever and Cardiology chapter of IAP
These Guidelines were formulated at National Consultative Meeting on 20th May, 2007, IMA Hall, New Delhi, under IAP Vision 2007

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Duration of secondary prophylaxis

i. No carditis: 5 years/18 years of age, whichever is longer.
ii. Mild to moderate carditis and healed carditis: 10 years/25 years of age, whichever is longer.
iii. Severe disease or post intervention patients: Lifelong. One may opt for secondary prophylaxis up to the age of 40 years.

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