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Synopsis: Current Trends in the Management of Acute Chest Syndrome in Children with Sickle Cell Disease

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Introduction

Acute chest syndrome (ACS) is a common and potentially life-threatening pulmonary complication of sickle cell disease (SCD) in children.[1] It is diagnosed by the occurrence of a new pulmonary infiltrate on chest imaging in conjunction with fever and/or respiratory symptoms, and it is a major cause of morbidity and mortality in paediatric SCD.[2,3] The term "acute chest syndrome" highlights the challenge of differentiating infectious pneumonia from other SCD-related pulmonary processes, including inflammatory lung injury following fat embolism and pulmonary infarction resulting from microvascular occlusion.[2]

Although ACS may mimic bacterial pneumonia clinically and radiologically, its progression differs markedly from that of pneumonia in children with normal haemoglobin phenotypes. [2] Clinical manifestations range from mild, self-limiting illness to rapidly worsening respiratory failure that necessitates urgent life-saving intervention.[3] Early diagnosis and prompt application of evidence-based treatment are, therefore, essential to prevent both acute complications and long-term sequelae.[3] This review discusses current approaches to managing ACS in children with SCD, with emphasis on practical treatment strategies suitable for resource-limited settings.

Literature Search Strategy

A literature search was conducted in PubMed using the keywords "acute chest syndrome" and "sickle cell disease," yielding 1,599 articles. The search was further refined by incorporating the terms "children" and "treatment," thereby reducing the results to 859 and 232 articles, respectively. Limiting the search to articles published between 2020 and 2025 produced 27 studies. Following exclusion of case reports and publications not directly aligned with the review objectives, 10 original and review articles published in English were selected. Data were extracted from these articles, together with relevant existing guidelines on ACS management, to inform this narrative review.

Epidemiology

Acute chest syndrome is the second most frequent cause of hospitalisation related to sickle cell disease, surpassed only by vaso-occlusive pain crises.[3,4] It also remains the leading cause of death among individuals with SCD.[4] Data from the 1994 Cooperative Study of Sickle Cell Disease (CSSCD),[5] which prospectively followed 3,751 patients for at least two years, showed that 28% (1,085 patients) experienced at least one episode of ACS, accounting for a total of 2,100 events. The incidence was higher among patients with sickle cell anaemia and HbS-β^+ thalassaemia (12.8 and 9.4 episodes per 100 patient-years, respectively) compared with those with HbSC and HbS-α^+ thalassaemia phenotypes (5.2 and 3.9 episodes per 100 patient-years, respectively). [5]

An inverse relationship between ACS incidence and age was observed, with the highest rates reported in children aged 2–4 years (25 episodes per 100 patient-years) and lower rates in adults (9 episodes per 100 patient-years).[5] The CSSCD further demonstrated age-related variation in clinical presentation, with younger children more frequently presenting with fever and cough, and rarely reporting chest pain, while adolescents and

adults typically presented with dyspnoea and severe chest pain.[5]

Pathophysiology and aetiology

Acute chest syndrome (ACS) has a multifactorial aetiology, with recognised triggers including pulmonary infection, fat embolism, pulmonary vascular infarction, and alveolar hypoventilation. Acting independently, or in combination, these factors reduce alveolar oxygen tension, promoting haemoglobin S (HbS) polymerisation, red cell sickling, and pulmonary vascular obstruction. The resulting impairment in pulmonary perfusion further exacerbates hypoxia, establishing a self-perpetuating cycle of hypoxia, HbS polymerisation, pulmonary vaso-occlusion, and pulmonary infarction. [4,6,7]

Pulmonary infection

Pathogenic organisms are identified in approximately 38% of cases from different clinical specimens.[6] An infectious cause is more frequently observed in children, with *Mycoplasma pneumoniae*, *Chlamydophila pneumoniae*, *Staphylococcus aureus*, *Streptococcus pneumoniae*, and *Haemophilus influenzae* being the most commonly isolated pathogens.[3,8] Additionally, respiratory viruses such as influenza A and respiratory syncytial virus (RSV) have been associated with the condition.[3,8,9]

Fat embolism

Vaso-occlusive episodes (VOE) within the bones lead to bone marrow necrosis and the release of fat emboli. These emboli enter the bloodstream and lodge in the pulmonary vasculature, initiating a cascade in which activated secretory phospholipase A2 cleaves fatty acids from phospholipids, releasing arachidonic acid.[6] Arachidonic acid is subsequently metabolised to form inflammatory leukotrienes and prostaglandins, which mediate alveolar inflammation and pulmonary vascular endothelial injury.[7]

Microvascular pulmonary infarction

In situ microvascular occlusion and pulmonary infarction are also associated with ACS and may be secondary to pulmonary intravascular sickling.[3] There is evidence that upregulation of vascular cellular adhesion molecule-1 (VCAM-1) by hypoxia, fat embolism and nitric oxide dysregulation, plays a role in the development of ACS by inducing respiratory endothelial red cell adhesion, thereby contributing to pulmonary vaso-occlusion.[9]

Hypoventilation

Vaso-occlusive pain involving the rib, spine, abdomen or resulting from opioid narcosis, is associated with hypoventilation, which may result in ACS. Thoracic pain secondary to bone infarction can cause splinting, hypoventilation and atelectasis, resulting in intra-pulmonary sickling.[3] Opioids, which are commonly prescribed for moderate to severe vaso-occlusive pain in children, may suppress the cough reflex and respiratory drive, promoting further hypoventilation. [10] Hypoventilation can also occur post-operatively following general anaesthesia.[10]

Asthma

A pre-existing diagnosis of asthma is associated with an increased incidence of ACS in children. [3] Children with concomitant asthma and sickle cell disease have been reported to have increased episodes of ACS, stroke, and the need for blood transfusions.[3]

Clinical features

The clinical manifestations of ACS vary with age, with fever, cough, and dyspnoea predominating in children.[3] Hypoxaemia is not universally present, but serves as an important marker of disease severity and outcome.[3,8] Early symptoms often precede radiographic abnormalities; therefore, a normal initial chest radiograph does not exclude the diagnosis.[3] In a substantial proportion of cases, ACS is not apparent at the time of admission, as nearly half of affected children initially present with vaso-occlusive pain and subsequently develop features of ACS within 72 hours of symptom onset.[3] ACS may also complicate the postoperative period, particularly in children who did not receive preoperative transfusions.[3] The clinical course can be unpredictable, with rapid deterioration occurring

even in those with initially mild symptoms. Consequently, vigilant monitoring for the development and progression of ACS throughout hospitalisation is essential, especially in patients presenting with chest pain. [3]

Diagnosis and evaluation

ACS is commonly defined operationally as the presence of a new pulmonary infiltrate on chest imaging in combination with one or more of the following: fever, cough, dyspnoea, or chest pain. [3,8] Initial evaluation focuses on confirming the diagnosis, identifying reversible or contributory factors, assessing disease severity, and guiding decisions regarding the appropriate level of care. [3]

Chest radiography remains a key diagnostic tool and should be obtained in all suspected cases;[3,8] however, its unavailability should not delay the initiation of treatment when clinical suspicion is high. [3] Early radiographic changes may be subtle, and repeat imaging is warranted if symptoms persist or worsen despite an initially normal study. [3]

Point-of-care lung ultrasonography is increasingly being explored as an alternative diagnostic modality due to its portability, lack of exposure to ionising radiation, and ease of bedside use.[10] In a study by Cohen et al. involving 191 children with SCD, ACS was identified in 17% of participants. Lung ultrasonography demonstrated an overall diagnostic accuracy of 92%, with sensitivity and specificity of 88% and 93%, respectively, compared with chest radiography. [10]

Continuous pulse oximetry is recommended to monitor oxygen saturation. However, the reliability of pulse oximetry in SCD has been questioned because anaemia and vaso-occlusive episodes shift of the oxyhaemoglobin dissociation curve to the right. [7] Serial measurements within the same patient are therefore more informative.

Arterial blood gas analysis should be performed in hypoxaemic patients, with co-oximetric measurements of carboxyhaemoglobin and methaemoglobin considered the gold standard in severe ACS. [3,7]

Baseline laboratory investigations should include blood grouping and antibody screening for HbS-negative donor blood, complete blood count, inflammatory markers (ESR or CRP), blood cultures, targeted respiratory investigations such as sputum culture or polymerase chain reaction (PCR) testing, and nasopharyngeal viral PCR panels.[3,6,8] Bronchoalveolar lavage is not routinely indicated due to its invasive nature and is typically reserved for patients requiring intubation and mechanical ventilation.[3] Where feasible, serological testing for atypical respiratory pathogens such as *Mycoplasma pneumoniae*, *Chlamydophila pneumoniae*, and *Legionella* species should be performed.[3,6]

In malaria-endemic regions, peripheral blood smears for malaria parasites are recommended, as *Plasmodium falciparum* infection is a recognised co-morbidity in children with SCD. [3,8]

Additional investigations may include liver and renal function tests, echocardiography in severe cases to assess cardiac function or pulmonary hypertension, and computed tomography pulmonary angiography (CTPA) when acute pulmonary embolism is suspected. [3]

Treatment

Management of ACS is multimodal and should be initiated promptly, guided by standardised institutional protocols. [3,8] The primary therapeutic goal is the prevention or reversal of acute respiratory failure. Core components of modern ACS management include respiratory support, blood transfusion, pharmacotherapy, supportive measures, and strategies to reduce post-discharge recurrence.[3,4,7,8] Multidisciplinary collaboration involving paediatric haematology, respiratory or critical care teams, physiotherapy, and transfusion services has been shown to improve outcomes.[4,7] Current evidence-informed management approaches are summarised below (Figure 1).

Respiratory Support

Optimal oxygen saturation targets in ACS are not well defined; however, a practical approach is to maintain SpO_2 ≥95% with continuous or at least 4-hourly monitoring to enable early detection of clinical deterioration.[3,8] Low-flow oxygen delivered via nasal cannula or simple face mask is often adequate for children with mild disease who can be managed on the general paediatric wards.[9] Patients with moderate to severe ACS should be

managed in high-dependency or intensive care settings.[9] Emerging evidence suggests that early use of non-invasive respiratory support (NIV), such as humidified high-flow nasal cannula (HFNC), continuous positive airway pressure (CPAP), or bi-level positive airway pressure (BiPAP), may reduce the need for endotracheal intubation in severe ACS.[6,9] Although randomised trials comparing HFNC with low-flow oxygen or other non-invasive modalities are lacking, HFNC is increasingly favoured due to better tolerability in children, ease of use, wide availability, and an acceptable safety profile.[9]

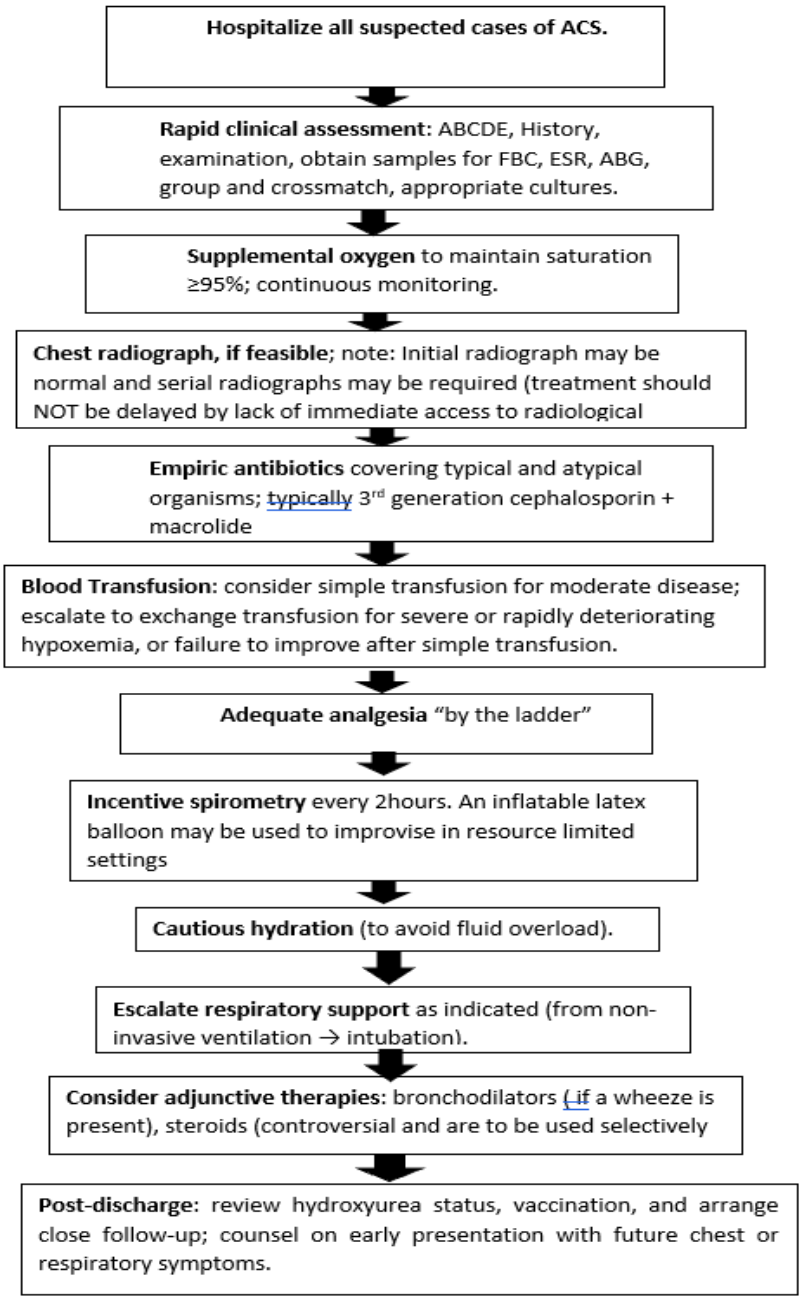

Figure 1: Practical approach to a child presenting with suspected ACS (summary algorithm)

Blood transfusion

The evidence base for transfusion therapy in ACS remains limited.[1,3] A 2020 Cochrane review identified only one eligible randomised controlled trial (the multicentre PROACTIVE study), which was underpowered to draw firm conclusions regarding transfusion efficacy.[1] Consequently, current transfusion practices rely heavily on expert consensus and guideline recommendations, which advocate early transfusion in children with severe ACS.[3,8]

Blood transfusion often results in rapid improvement in clinical status, radiographic findings, and oxygenation, and may be life-saving in severe disease.[3,7] Both simple (top-up) and exchange blood transfusions (EBT) improve oxygen delivery; however, exchange transfusion additionally removes sickled erythrocytes and replaces them with normal donor cells, thereby reducing ongoing vaso-occlusion.[7] All transfused blood in ACS must therefore be HbS-negative.[3]

Concerns have been raised regarding simple transfusions due to the risk of hyperviscosity, which may predispose to complications such as stroke.[9] Manual exchange transfusion carries a lower but still present risk of hyperviscosity, while automated exchange transfusion is often unavailable in resource-limited settings.[7,8] Current guidelines recommend simple transfusion for patients with mild to moderate Hypoxaemia and haemoglobin levels <9 g/dL, while exchange transfusion is indicated for severe Hypoxaemia, clinical deterioration despite initial transfusion, or haemoglobin levels ≥9 g/dL.[3,8] Early initiation of transfusion is advised, as respiratory failure can develop rapidly, and early intervention may prevent progression.[3] Simple transfusion is typically administered at 10 mL/kg of packed red cells, targeting a haemoglobin concentration of 10–11 g/dL,[7,8]. In comparison, EBT requires 40 mL/kg of packed cells in children weighing less than 50kg.[3]

Pharmacotherapy

Antimicrobial therapy: Empirical broad-spectrum antibiotics targeting both typical and atypical respiratory pathogens are recommended in all cases of ACS. [3,8] Common regimens include a third-generation cephalosporin combined with azithromycin, clarithromycin, or vancomycin. [3,8,9] Antiviral therapy, such as oseltamivir, is advised in suspected or confirmed influenza infection, particularly in unvaccinated children. [3,9]

Bronchodilators: These agents may be beneficial in patients with a history of asthma or clinical evidence of bronchospasm. However, current evidence supporting their routine use in ACS is limited. [11]

Corticosteroids: The role of corticosteroids in ACS remains contentious. While some studies suggest reduced morbidity and transfusion requirements, concerns regarding adverse outcomes such as haemorrhagic stroke and rebound vaso-occlusive episodes following withdrawal limit their widespread use. [3,7]

Inhaled nitric oxide: Although inhaled nitric oxide has been explored as a therapeutic option, randomised trials have not demonstrated significant benefit in reducing vaso-occlusive episodes or ACS incidence. [7] As such, routine use is not currently supported by evidence. [3,7]

Anticoagulation: Anticoagulants, particularly low-molecular-weight heparins such as tinzaparin, have been investigated for their potential to reduce vaso-occlusion, especially when fat or thromboembolism is suspected. [7] However, evidence remains insufficient to recommend routine use in paediatric ACS.

Supportive treatment

Adequate pain control

Severe chest pain can lead to thoracic splinting and reduced ventilation,[3] which may progress to ACS. Adequate analgesia facilitates deeper breathing and decreases the risk of ACS in patients with vaso-occlusive chest pain.[7] Current guidelines advocate a “two-step” analgesic ladder in children: mild pain is managed with paracetamol and/or non-steroidal anti-inflammatory drugs (NSAIDs), while moderate to severe pain requires opioid analgesics, most commonly morphine.[3,7,8] Caution is required to prevent opioid-induced hypoventilation, which may increase the risk of ACS.[3] Patient-controlled

analgesia is recommended to minimise the risk of opioid narcosis.[7]

Cautious hydration

Fluid management in ACS should be individualised and guided by the patient's fluid balance and cardiopulmonary status [7] Intravenous crystalloid administration should be cautious, as patients with ACS are prone to pulmonary oedema.[3,7] In children with ACS, conservative hydration using approximately 75% of daily maintenance fluid is advised.[7]

Incentive spirometry

Incentive spirometry is effective in preventing and reducing atelectasis resulting from hypoventilation.[3] Evidence from a randomised controlled trial shows that its use in patients admitted with acute chest or back pain significantly reduces progression to ACS.[6] All children with ACS should be encouraged to perform incentive spirometry at a rate of ten breaths every two hours.[3] Where spirometers are unavailable, blowing inflatable latex balloons at a similar frequency is an acceptable alternative.[8]

Post-discharge care

Hydroxyurea: Current recommendations support the use of hydroxyurea in all children with sickle cell disease (SCD) from the age of nine months.[4] Hydroxyurea has been shown to reduce recurrent episodes of ACS and other SCD-related complications.[4] A randomised, double-blind trial in sub-Saharan Africa demonstrated fewer ACS episodes in children receiving dose-escalated hydroxyurea up to 30 mg/kg/day compared with a fixed dose of 20 mg/kg/day, indicating greater efficacy at maximally tolerated doses.[4] Hydroxyurea is recommended following a single severe episode of ACS, and for patients with recurrent ACS who are not yet receiving this therapy.[3]

Chronic transfusion therapy

There is limited evidence supporting chronic transfusion for the prevention of recurrent ACS. It is noteworthy, however, that findings from the Stroke Prevention Trial in Sickle Cell Anaemia (STOP) showed a reduction in ACS episodes among children receiving chronic transfusion compared with those receiving standard care.[12]

Preoperative blood transfusion:

A randomised trial comparing preoperative simple transfusion with no transfusion was terminated early due to a significantly higher incidence of ACS in the non-transfused group.[4] Consequently, prophylactic preoperative simple transfusion targeting a haemoglobin level of approximately 10 g/dL is recommended for patients with SCD undergoing surgery, particularly when general anaesthesia is required.[4]

L-glutamine and crizanlizumab therapy

Emerging evidence suggests that newer SCD therapies, including L-glutamine and the monoclonal P-selectin antibody, crizanlizumab, may reduce the frequency of ACS and other vaso-occlusive events in children with SCD.[4,6]

Vaccination

Booster vaccinations are recommended for all patients with SCD from the age of two years.[13] Vaccination status should be reviewed before discharge following an ACS episode, and any outstanding age-appropriate vaccines should be scheduled.[3,8] Recommended booster vaccine schedules for children with SCD are summarised in Table I.

Haematopoietic stem cell transplantation (HSCT)

Although no comparative trials are evaluating HSCT versus standard therapy for ACS prevention, observational studies indicate substantial benefit in preventing recurrent ACS. Current guidelines suggest considering HSCT for eligible children with recurrent ACS who have not responded adequately to hydroxyurea, chronic transfusion therapy, or newer targeted treatments.[3,8,1]

Prognosis

ACS is associated with considerable morbidity and mortality. With appropriate and timely care, most children recover without sequelae. Recurrent episodes may, however, occur, which increase the risk of chronic pulmonary disease later in life.

Table I: Recommended Immunization Schedule for children with SCD[13]

	Pneumococcus	Meningococcus and H. influenzae type b	Influenza virus
Under 2 years	Routine immunisation	Routine immunisation	Annual
Age 2-5 years (fully immunised)	Single-dose PPV	Booster Hib and Men C vaccines given 2 months apart	Annual
Age 2-5 years (No/partial vaccination)	Two doses of PCV two months apart, followed 2 months later by PPV	Same as above	Annual
Age > 5 yrs (fully vaccinated)	Single-dose PPV	Same as above	Annual
Age > 5 years (unvaccinated)	Single-dose PPV	Same as above	Annual
Reinforcing immunisation	Single dose PPV every 5 yrs	Men C every 5 years	Annual

Schedule summarised from Salisbury D, Ramsay M, Noakes K. Immunisation against infectious diseases (The Green Book). London: Department of Health; 2006.13.
Hib - Haemophilus influenzae Type b; MenC - Meningococcus group C; PCV - Pneumococcal Conjugate Vaccine; PPV - Pneumococcal Polysaccharide Vaccine.

Conclusion

Acute chest syndrome is associated with significant morbidity and mortality in children with SCD. Contemporary management of this condition requires a combination of respiratory support, transfusion therapy, the use of antimicrobials, analgesics and incentive spirometry. Novel acute disease interventions, such as the use of anticoagulants and inhaled NO, have been described but remain under investigation.

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CLINICAL QUIZ

A 12-year old presents with frequent passage of watery stools and excessive sweating. The parents are worried that her "eyes are getting bigger". The pictures below show the face of the girl six months after the onset of symptoms [A] and nine months after the commencement of treatment [B].

A

B

Questions

1. **Which clinical feature is most commonly observed in the condition?**

a. Breathlessness
b. Depression
c. Somnolence
d. Weight loss despite increased appetite

2. **Which of the following laboratory findings is typically expected in the condition?**

a. Low TSH, high free T4
b. High TSH, low free T4
c. High TSH, high free T4
d. Normal TSH and free T4

3. **Which of the following best explains the pathogenesis of the eye sign?**

a. Increased secretion of aqueous humour
b. Autoimmune-mediated proliferation of orbital fibroblasts
c. Direct stimulation of extraocular muscles by hormones
d. Deposition of calcium salts in orbital tissues

4. Which of the following is an unusual ocular complication of the condition?
a. Dry, gritty eyes
b. Excessive watering
c. Blurred, double vision
d. Cataract

5. **Which of these statements is not valid with regards to management options in the condition?**
a. A combination of carbimazole and carvedilol for a minimum of two years is recommended
b. Osteoporosis is a common unwanted effect of anti-thyroid therapies
c. At the onset of radioactive iodine therapy, it is recommended that anti-thyroid medications should also be administered.
d. Exophthalmos is an indication for surgery

6. If the patient develops a chest pain, which of the following pathologies should receive prime attention?
a. Nervousness
b. Dyspepsia
c. Cardiac arrhythmias
d. Pericardial effusion

Key:

Question	***Answer***
1	D
2	A
3	B
4	D
5	B
6	C

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