thebmj

BMJ 2017;356:j686 doi: 10.1136/bmj.j686 (Published 2017 March 02)

PRACTICE

CLINICAL UPDATES

Community acquired pneumonia in children

Iram J Haq *registrar and clinical research associate in paediatric respiratory medicine*[1 2], Alexandra C Battersby *registrar in paediatric immunology and infectious diseases*[3], Katherine Eastham *consultant paediatrician*[4], Michael McKean *consultant in paediatric respiratory medicine and clinical director*[2]

[1]Institute of Cellular Medicine, Newcastle University, Newcastle upon Tyne, UK; [2]Department of Paediatric Respiratory Medicine, Great North Children's Hospital, Newcastle upon Tyne; [3]Department of Paediatric Immunology and Infectious Diseases, Great North Children's Hospital, Newcastle upon Tyne; [4]Department of Paediatrics, Sunderland Royal Hospital, Sunderland, UK

Correspondence to: I J Haq iram.haq@newcastle.ac.uk

In 2015, community acquired pneumonia (CAP) accounted for 15% of deaths in children under 5 years old globally and 922 000 deaths globally in children of all ages.[1] It is defined as a clinical diagnosis of pneumonia caused by a community acquired infection in a previously healthy child.[2] Clinical assessment can be challenging; symptoms vary with age and can be non-specific in young children, and aetiology is often unknown at presentation.

This article will provide an update on CAP management in otherwise healthy children outside the neonatal period and summarises recommendations from the British Thoracic Society guidelines for UK practice.[2] Similar international guidelines, including the World Health Organisation and Infectious Diseases Society of America guidelines, have some treatment variations, probably dependent on drug availability, cost, and antibiotic resistance patterns.[3 4]

How common is CAP?

Around 14.4 per 10 000 children aged over 5 years and 33.8 per 10 000 under 5 years are diagnosed with CAP annually in European hospitals.[5 6] CAP is more common in the developing world, estimated at 0.28 episodes per child per year and accounting for 95% of all cases.[7] Incidence data varies and may be explained by variation in diagnostic criteria. A bias exists towards hospital based studies, which potentially underestimates overall incidence. Children aged 5-16 years are underrepresented in the literature, making assessment of CAP prevalence in this group difficult.

In otherwise healthy children, those less than 5 years old are at greatest risk. Boys have a higher incidence across all ages.[5] Other risk factors include prematurity, immunodeficiency, chronic respiratory disease, and neurodisability.

What causes CAP?

Defining causative organisms is a challenge. Clinical and radiological features do not reliably distinguish between viral and bacterial aetiology, and obtaining cultures from the lower respiratory tract of young children is tricky. More specific but invasive investigations such as pleural aspiration are infrequently indicated and reserved for severe cases. Blood cultures are rarely performed in patients managed in the community, and hospitalised patients demonstrate a poor yield.[8]

Nasopharyngeal secretions are easily obtainable, and the application of more sensitive techniques such as polymerase chain reaction (PCR) has resulted in pathogen identification in 65-83% of reported cases.[9] Although rapid viral detection is now available with multiplex PCR techniques, differentiating bacterial superinfection from colonisation remains difficult.[10]

CAP aetiology varies with age (table 1⇓). Respiratory viruses are common, particularly in infants, accounting for 30-67% of hospitalised cases. Respiratory syncytial virus accounts for 30% of viral aetiology. Other viruses include parainfluenza, influenza, and human metapneumovirus.[11-13] *Streptococcus pneumoniae* is the commonest bacterial cause across all ages, accounting for 30-40% of cases.[9 13] Other bacterial causes include group A streptococcus and, in infants, group B streptococcus. *Staphylococcal aureus* is associated with round pneumonia, a well defined round area of consolidation visible on chest x ray. Despite a well established vaccination programme, *Haemophilus influenza* remains prevalent in the UK, albeit at lower rates.[13] *Mycoplasma pneumoniae* accounts for up to a third of all cases and is a common cause of atypical CAP.[14 15]

Less common pathogens are often related to an underlying health problem—for example, fungi in an immunocompromised child. *Burkhodheria cepacia*, *Aspergillus fumigatus*, and *Pseudomonas aeruginosa* are associated with primary immunodeficiency and cystic fibrosis.[16] Consider aspiration pneumonia in high risk children or if the history is suggestive.

If there has been recent foreign travel, unusual organisms associated with the travel destination and variations with antibiotic resistance are important considerations. Consider atypical organisms if treatment fails.

What you need to know

- Introduction of the pneumococcal conjugate vaccine has significantly reduced rates of community acquired pneumonia (CAP) in the developed world
- Clinical assessment requires careful evaluation of clinical features, severity, and evidence of complications
- Children with mild to moderate symptoms can be managed in the community
- Recommended empirical first line treatment is oral amoxicillin. Intravenous antibiotics are indicated in children who cannot tolerate oral medicines or have septicaemia or complications
- Patients should be reviewed 48 hours after starting treatment to monitor response and for evidence of complications

How is CAP assessed?

Figure 1⇓ summarises the approach for assessment and management of CAP. Assess the likelihood and severity of CAP by measuring fever, tachypnoea, cough, breathlessness, chest wall recession, and chest pain. Respiratory rate and dyspnoea are useful measures of severity and predict oxygen requirement.[2 17] A UK prospective study investigating children with radiologically defined CAP found respiratory rate to be positively correlated with reduced oxygen saturations in children of all ages and dyspnoea in children over 1 year old.[17] Increased work of breathing is associated with radiological changes.[18 19]

It is difficult to distinguish clinically between bacterial and viral aetiologies. Consider bacterial pneumonia in children presenting with persistent or recurrent fever ≥38.5°C over the preceding 24-48 hours with chest wall recession and tachypnoea.[2] Fever and tachypnoea are early features of pneumococcal pneumonia. Cough is not always apparent or required for diagnosis, and may be absent in the early stages of illness. Mycoplasma pneumonia presents with cough and chest pain and is often associated with wheeze, general malaise, arthralgia, sore throat, and headache.

Clinical features vary with age. Local variations in CAP management and definitions can be challenging when comparing studies. Often a combination of clinical signs, rather than individual features, leads to a clinical diagnosis and helps assess severity.

Table 2⇓ lists disease severity markers to help aid management. Mild to moderate severity confers a low risk of complications. Previously well children with only mild symptoms who present directly to community or acute secondary services can be managed safely in the community. Children with severe symptoms require secondary care referral for urgent assessment and may require admission to paediatric intensive care (box 1). Children who present with mild symptoms but have red flag features (box 2) may require secondary care management and need careful assessment.

Assessment in the community

Focus the examination on defining severity and identify children with underlying conditions who are at increased risk. Hypoxaemia increases mortality risk, and oxygen saturations <95% in room air are a key indicator for hospital assessment.[20]

Assessment in hospital

All children require pulse oximetry. Level of C reactive protein is not useful to differentiate viral and bacterial causes, but it can guide investigation and management of CAP complicated by effusions, empyema, or necrosis.[2] Urinary pneumococcal antigen detection has a high sensitivity but very low specificity.[21] If it is available, consider using it as a negative predictor.[2]

Avoid routine chest radiography in children requiring hospital admission.[2] Radiographic appearance correlates poorly with clinical signs and outcome, and there is high inter-observer variability in interpretation.[22 23] Consider radiography in severe cases or where complications such as effusion or empyema are suspected (fig 2⇓).

Investigations recommended by the British Thoracic Society for complicated or severe CAP are summarised in box 3.[2]

How is CAP managed?

Children with clinical features consistent with CAP require antibiotics (box 4). CAP in a fully vaccinated child less than 2 years old (who has received the pneumococcal vaccine) with mild symptoms is unlikely to be bacterial, and antibiotics are not required unless symptoms become more severe.[2]

Antibiotics

British Thoracic Society guidelines recommend amoxicillin as first line treatment.[2] Consider adding a macrolide if there is no improvement or resolution of symptoms after 48 hours. Macrolides are recommended instead of amoxicillin as first line treatment if the child is allergic to penicillin. Dual treatment with amoxicillin and a macrolide may be considered for suspected mycoplasma pneumonia.

Antibiotic resistance is a global issue. Penicillin and macrolide resistance of *Streptococcus pneumoniae* is low in the UK compared with mainland Europe.[2] Second or third line treatment may be required to cover resistant pneumococcal strains or children who have recently travelled to mainland Europe. There is evidence of increasing macrolide resistance of group A streptococcus, with varying rates worldwide.[24]

Several large randomised controlled trials, including the UK PIVOT trial, have shown that oral amoxicillin produces outcomes equivalent to those achieved with parenteral penicillin.[25-27] This was confirmed by a Cochrane review of children hospitalised with severe CAP.[28] However, a UK audit of children requiring hospital admission found that co-amoxiclav was most commonly used.[29] This is probably explained by variations in clinical custom. Amoxicillin is safe to administer orally if tolerated, even in cases of severe CAP. Its treatment efficacy is similar to co-amoxiclav but is better tolerated and more cost effective.[2]

In the absence of guidance for optimal treatment duration, empirical treatment is generally for 7-10 days. The UK CAPIT study will investigate the optimum treatment dose and duration.[30]

Supportive therapies and advice for care givers

For children managed in the community with mild to moderate symptoms, provide safety net advice on signs of deterioration, dehydration, and complications. Offer written information, if available, regarding fever management and what to watch out for. Ask the parents or carers to seek further advice if fever persists or symptoms deteriorate despite 48 hours of antibiotic treatment.

Box 1: British Thoracic Society criteria for referral to paediatric intensive care[2]

Indications for referral

- Development of respiratory failure requiring assisted ventilation
- Pneumonia complicated by septicaemia

Clinical features

- Failure to maintain oxygen saturations >92% with FiO_2 60%
- Clinical features of shock
- Increasing respiratory and heart rates with severe respiratory distress and exhaustion, with or without raised pCO_2
- Recurrent apnoea or slow irregular breathing

British Thoracic Society admission criteria are similar to those of international guidelines in similar resource settings.[4]
FiO_2 = fraction of inspired oxygen. pCO_2 = partial pressure of carbon dioxide.

Box 2: Red flag features for community acquired pneumonia (CAP)

History of underlying comorbidities, including

- Bronchopulmonary dysplasia
- Disorders of mucus clearance (such as cystic fibrosis)
- Congenital heart disease
- Immunodeficiency
- Severe cerebral palsy

Relevant medical history

- History of severe pneumonia (inpatient stay requiring oxygen, paediatric intensive care admission, complications of CAP (such as lung abscess, effusion, empyema)
- Recurrent pneumonia

Box 3: British Thoracic Society recommended investigations for complicated or severe community acquired pneumonia (CAP)[2]

- Bloods (full blood count, urea and electrolytes, C reactive protein, blood culture, anti-streptolysin O titre, serology for viruses, *Mycoplasma pneumoniae* and *Chlamydia pneumoniae*, atypical CAP screen)
- Nasopharyngeal secretions and swabs for viral PCR or immunofluorescence detection
- Chest x ray to assess for effusion or empyema
- Consider pleural fluid for microscopy, culture (including tuberculosis), pneumococcal antigen for PCR, biochemistry, and cytology (if aspiration required)

PCR = polymerase chain reaction.

Box 4: British Thoracic Society recommendations for antibiotic selection in community acquired pneumonia (CAP)[2]

Preferred route of administration

- Oral antibiotics are safe and effective for children even with severe CAP
- Use intravenous antibiotics in children who:
  - Are unable to tolerate oral fluids (such as because of vomiting) *or*
  - Have signs of septicaemia or complicated pneumonia

Which antibiotic?

- Amoxicillin is first line therapy (use macrolides as first line in penicillin allergy)
- Macrolides can be added at any age if there is no response to first line therapy
- Macrolides should be used if *Mycoplasma* or *Chlamydia pneumoniae* are suspected or if disease is severe
- Co-amoxiclav is recommended for pneumonia associated with influenza
- Intravenous antibiotic treatment with amoxicillin, co-amoxiclav, cefuroxime, cefotaxime, or ceftriaxone is recommended for severe pneumonia

In secondary care, children with oxygen saturations <92% in room air require supplemental oxygen to maintain >95% saturation. Oxygen can be administered via face mask, nasal cannulae, or head box (a device that surrounds the head to deliver humidified oxygen to babies). Method of delivery depends on the clinical condition, required volume of inspired oxygen, and practical considerations such as age and feeding. There is no evidence to suggest that any method is superior to others.[2]

Hydration can become compromised in severe CAP due to breathlessness, fatigue, and vomiting. Nasogastric feeds can maintain hydration, but if they are not tolerated because of vomiting or severe illness, intravenous fluid replacement may be required, with daily electrolyte monitoring for sodium depletion or syndrome of inappropriate antidiuretic hormone secretion.

Clinical trials have not shown any benefit from physiotherapy on radiological resolution, length of hospital stay, or symptom

improvement.[31 32] This may not be true during recovery for children with underlying respiratory diseases and impaired mucus clearance.

Spotting complications

Empyema (pus in the pleural space) is the most common complication.[33]Table 3⇓ summarises the clinical features that should arouse suspicion for empyema and lung abscess. A case-control study of children hospitalised in the north east of England with clinical and radiological features of pneumonia revealed that empyema was evident in 25%.[37] In empyema, effusions are initially exudative and become fibro-purulent, loculated, and infected without treatment. Ongoing fibroblastic growth causes formation of a thick peel over the visceral pleura, preventing lung expansion.

Other complications include necrotising pneumonia, systemic sepsis, haemolytic uraemic syndrome, and bronchiectasis following severe or complicated CAP. Offer secondary care referral to those with suspected complications.

What follow-up is required?

Follow-up is not routinely needed in children who recover fully without complications. Children who do not improve in 48-72 hours after starting treatment need reassessment, which can be in the community. Children who have lobar collapse, round pneumonia, or complications of CAP on radiography require follow-up as an outpatient at six to eight weeks with a repeat x ray and clinical assessment.

Reducing CAP incidence

Various public health measures reduce CAP incidence. The current UK vaccination schedule involves doses of pneumococcal conjugate vaccine (PCV) at 2, 4, and 12 months old. *Haemophilus influenzae* type B (Hib) vaccination is given at 2, 3, and 4 months with a booster at 1 year.[38] An annual influenza vaccine is given to children between 2 and 8 years old every September, including children in school years 1, 2, and 3.[38] Additional pneumococcal, and in some cases influenza, vaccination is provided for high risk children with asplenia or splenic dysfunction, cochlear implants (due to the meningitis risk), chronic disease, complement disorders, and immunosuppression.[38 39]

PCV7 protects against seven pneumococccal serotypes: 4, 6B, 9V, 14, 18C, 19F, and 23F. At the time of its introduction to the UK vaccination schedule in 2006, these serotypes accounted for up to 90% of invasive pneumococcal disease in northern America and substantially fewer, up to 15%, of European cases.[40] PCV13 was introduced into the UK schedule in 2010, providing additional cover for serotypes 1, 3, 4, 5 6A, 6B, 7F, 9V, 14, 18C, 19A, 19F, and 23F.

Globally, the WHO recommended routine Hib and PCV vaccination in 2006 and 2007 respectively. By 2016, 98% of countries had introduced Hib into their routine schedule and 68% had introduced PCV.[41] Comparison of different PCV vaccination schedules has shown a marginal seropositivity benefit in those vaccinated with a primary course of three vaccines versus two, without any obvious clinical benefit.[42]

PCV implementation has reduced CAP incidence, admission rates, invasive pneumococcal disease, and radiologically confirmed pneumonia in both developed and low income settings.[43-47] PCV13 introduction has prevented infection by resistant pneumococcal strains including serotype 19A. Hib vaccination has reduced pneumonia rates in the developing world and the UK.[48 49] Canadian data suggest that routine influenza vaccination reduces mortality in all ages and emergency department attendances.[50]

Competing interests: We have read and understood the BMJ Group policy on declaration of interests and have no relevant interests to declare.

Provenance and peer review: Commissioned; externally peer reviewed.

1 World Health Organization. Pneumonia. WHO, 2015.
2 Harris M, Clark J, Coote N, et al. British Thoracic Society Standards of Care Committee. British Thoracic Society guidelines for the management of community acquired pneumonia in children: update 2011. *Thorax* 2011;356(Suppl 2):ii1-23. doi:10.1136/thoraxjnl-2011-200598 pmid:21903691.
3 World Health Organization. Revised WHO classification and treatment of pneumonia in children at health facilities: evidence summaries. WHO, 2014.
4 Bradley JS, Byington CL, Shah SS, et al. Pediatric Infectious Diseases Society and the Infectious Diseases Society of America. The management of community-acquired pneumonia in infants and children older than 3 months of age: clinical practice guidelines by the Pediatric Infectious Diseases Society and the Infectious Diseases Society of America. *Clin Infect Dis* 2011;356:e25-76. doi:10.1093/cid/cir531 pmid:21880587.
5 Clark JE, Hammal D, Hampton F, Spencer D, Parker L. Epidemiology of community-acquired pneumonia in children seen in hospital. *Epidemiol Infect* 2007;356:262-9. doi:10.1017/S0950268806006741 pmid:17291362.
6 Senstad AC, Surén P, Brauteset L, Eriksson JR, Høiby EA, Wathne KO. Community-acquired pneumonia (CAP) in children in Oslo, Norway. *Acta Paediatr* 2009;356:332-6. doi:10.1111/j.1651-2227.2008.01088.x pmid:19006533.
7 Rudan I, Tomaskovic L, Boschi-Pinto C, Campbell H. WHO Child Health Epidemiology Reference Group. Global estimate of the incidence of clinical pneumonia among children under five years of age. *Bull World Health Organ* 2004;356:895-903.pmid:15654403.
8 Davis T, Evans H, Murtas J, Weisman A, Francis JL, Khan A. Utility of blood cultures in children admitted to hospital with community-acquired pneumonia. *J Paediatr Child Health* 2016;doi:10.1111/jpc.13376.
9 Thomson A, Harris M. Community-acquired pneumonia in children: what's new? *Thorax* 2011;356:927-8. doi:10.1136/thoraxjnl-2011-200671 pmid:21933948.
10 Clark JE. Determining the microbiological cause of a chest infection. *Arch Dis Child* 2015;356:193-7. doi:10.1136/archdischild-2013-305742 pmid:25246089.
11 Cevey-Macherel M, Galetto-Lacour A, Gervaix A, et al. Etiology of community-acquired pneumonia in hospitalized children based on WHO clinical guidelines. *Eur J Pediatr* 2009;356:1429-36. doi:10.1007/s00431-009-0943-y pmid:19238436.
12 Michelow IC, Olsen K, Lozano J, et al. Epidemiology and clinical characteristics of community-acquired pneumonia in hospitalized children. *Pediatrics* 2004;356:701-7. doi:10.1542/peds.113.4.701 pmid:15060215.
13 Bowen SJ, Thomson AH. British Thoracic Society Paediatric Pneumonia Audit: a review of 3 years of data. *Thorax* 2013;356:682-3. doi:10.1136/thoraxjnl-2012-203026 pmid:23291351.
14 Principi N, Esposito S, Blasi F, Allegra L. Mowgli study group. Role of Mycoplasma pneumoniae and Chlamydia pneumoniae in children with community-acquired lower respiratory tract infections. *Clin Infect Dis* 2001;356:1281-9. doi:10.1086/319981 pmid:11303262.
15 Baer G, Engelcke G, Abele-Horn M, Schaad UB, Heininger U. Role of Chlamydia pneumoniae and Mycoplasma pneumoniae as causative agents of community-acquired pneumonia in hospitalised children and adolescents. *Eur J Clin Microbiol Infect Dis* 2003;356:742-5. doi:10.1007/s10096-003-1037-9 pmid:14610659.
16 Bylund JD, Goldblatt D, Speert DP. Chronic granulomatous disease: from genetic defect to clinical presentation. In: Pollard AJ, Finn A, eds. *Hot Topics in Infection and Immunity in Children*. Springer, 2005: 67-87doi:10.1007/0-387-25342-4_5.
17 Clark JE, Hammal D, Spencer D, Hampton F. Children with pneumonia: how do they present and how are they managed? *Arch Dis Child* 2007;356:394-8. doi:10.1136/adc.2006.097402 pmid:17261579.
18 Redd SC, Vreuls R, Metsing M, Mohobane PH, Patrick E, Moteetee M. Clinical signs of pneumonia in children attending a hospital outpatient department in Lesotho. *Bull World Health Organ* 1994;356:113-8.pmid:8131246.
19 Cherian T, John TJ, Simoes E, Steinhoff MC, John M. Evaluation of simple clinical signs for the diagnosis of acute lower respiratory tract infection. *Lancet* 1988;356:125-8. doi:10.1016/S0140-6736(88)90683-6 pmid:2899187.
20 Smyth A, Carty H, Hart CA. Clinical predictors of hypoxaemia in children with pneumonia. *Ann Trop Paediatr* 1998;356:31-40. doi:10.1080/02724936.1998.11747923 pmid:9691999.
21 Charkaluk M-L, Kalach N, Mvogo H, et al. Assessment of a rapid urinary antigen detection by an immunochromatographic test for diagnosis of pneumococcal infection in children. *Diagn Microbiol Infect Dis* 2006;356:89-94. doi:10.1016/j.diagmicrobio.2005.10.013 pmid:16530375.
22 Hazir T, Nisar YB, Qazi SA, et al. Chest radiography in children aged 2-59 months diagnosed with non-severe pneumonia as defined by World Health Organization: descriptive multicentre study in Pakistan. *BMJ* 2006;356:629. doi:10.1136/bmj.38915.673322.80 pmid:16923771.
23 Swingler GH, Hussey GD, Zwarenstein M. Randomised controlled trial of clinical outcome after chest radiograph in ambulatory acute lower-respiratory infection in children. *Lancet* 1998;356:404-8. doi:10.1016/S0140-6736(97)07013-X pmid:9482294.
24 Beekmann SE, Heilmann KP, Richter SS, García-de-Lomas J, Doern GV. GRASP Study Group. Antimicrobial resistance in Streptococcus pneumoniae, Haemophilus influenzae, Moraxella catarrhalis and group A beta-haemolytic streptococci in 2002-2003. Results of the multinational GRASP Surveillance Program. *Int J Antimicrob Agents* 2005;356:148-56. doi:10.1016/j.ijantimicag.2004.09.016 pmid:15664485.
25 Atkinson M, Lakhanpaul M, Smyth A, et al. Comparison of oral amoxicillin and intravenous benzyl penicillin for community acquired pneumonia in children (PIVOT trial): a multicentre pragmatic randomised controlled equivalence trial. *Thorax* 2007;356:1102-6. doi:10.1136/thx.2006.074906 pmid:17567657.

Additional educational resources

Resources for clinicians

- Harris M, Clark J, Coote N, et al; British Thoracic Society Standards of Care Committee. British Thoracic Society guidelines for the management of community acquired pneumonia in children: update 2011. *Thorax* 2011;66(suppl 2):ii1-23. doi:10.1136/thoraxjnl-2011-200598
- World Health Organization. Revised WHO classification and treatment of pneumonia in children at health facilities: evidence summaries. 2014. http://apps.who.int/iris/bitstream/10665/137319/1/9789241507813_eng.pdf
- Balfour-Lynn IM, Abrahamson E, Cohen G, et al; Paediatric Pleural Diseases Subcommittee of the BTS Standards of Care Committee. BTS guidelines for the management of pleural infection in children. *Thorax* 2005;60(suppl 1):i1-21. doi:10.1136/thx.2004.030676
- Public Health England. The complete routine immunisation schedule. 2016. www.gov.uk/government/publications/the-complete-routine-immunisation-schedule

Resources for patients

- NHS Choices. Pneumonia. www.nhs.uk/Conditions/Pneumonia/Pages/Introduction.aspx
- NHS Choices. Vaccinations: When to have vaccinations. www.nhs.uk/conditions/vaccinations/pages/vaccination-schedule-age-checklist.aspx

How patients were involved in this article

The *BMJ* did not ask the authors to involve patients in the creation of this article.

26 Addo-Yobo E, Chisaka N, Hassan M, et al. Oral amoxicillin versus injectable penicillin for severe pneumonia in children aged 3 to 59 months: a randomised multicentre equivalency study. *Lancet* 2004;356:1141-8. doi:10.1016/S0140-6736(04)17100-6 pmid:15451221.

27 Hazir T, Fox LM, Nisar YB, et al. New Outpatient Short-Course Home Oral Therapy for Severe Pneumonia Study Group. Ambulatory short-course high-dose oral amoxicillin for treatment of severe pneumonia in children: a randomised equivalency trial. *Lancet* 2008;356:49-56. doi:10.1016/S0140-6736(08)60071-9 pmid:18177775.

28 Rojas MX, Granados C. Oral antibiotics versus parenteral antibiotics for severe pneumonia in children. *Cochrane Database Syst Rev* 2006;(2):CD004979.pmid:16625618.

29 BTS. 2012-2013 BTS paediatric pneumonia audit. 2013. www.brit-thoracic.org.uk/document-library/audit-and-quality-improvement/audit-reports/bts-paediatric-pneumonia-audit-report-201213/.

30 PERUKI. CAPIT feasibility service evaluation. 2016. www.peruki.org/current-studies.

31 Paludo C, Zhang L, Lincho CS, Lemos DV, Real GG, Bergamin JA. Chest physical therapy for children hospitalised with acute pneumonia: a randomised controlled trial. *Thorax* 2008;356:791-4. doi:10.1136/thx.2007.088195 pmid:18276723.

32 Britton S, Bejstedt M, Vedin L. Chest physiotherapy in primary pneumonia. *Br Med J (Clin Res Ed)* 1985;356:1703-4. doi:10.1136/bmj.290.6483.1703 pmid:3924221.

33 Light RW. Parapneumonic effusions and empyema. *Proc Am Thorac Soc* 2006;356:75-80. doi:10.1513/pats.200510-113JH pmid:16493154.

34 Tan TQ, Mason EO Jr, , Wald ER, et al. Clinical characteristics of children with complicated pneumonia caused by Streptococcus pneumoniae. *Pediatrics* 2002;356:1-6. doi:10.1542/peds.110.1.1 pmid:12093940.

35 Principi N, Esposito S. Management of severe community-acquired pneumonia of children in developing and developed countries. *Thorax* 2011;356:815-22. doi:10.1136/thx.2010.142604 pmid:20965930.

36 Balfour-Lynn IM, Abrahamson E, Cohen G, et al. Paediatric Pleural Diseases Subcommittee of the BTS Standards of Care Committee. BTS guidelines for the management of pleural infection in children. *Thorax* 2005;356(Suppl 1):i1-21. doi:10.1136/thx.2004.030676 pmid:15681514.

37 Elemraid MA, Thomas MF, Blain AP, et al. North East of England Pediatric Respiratory Infection Study Group Newcastle upon Tyne, UK. Risk factors for the development of pleural empyema in children. *Pediatr Pulmonol* 2015;356:721-6. doi:10.1002/ppul.23041 pmid:24692118.

38 Public Health England. The complete routine immunisation schedule. 2016. www.gov.uk/government/publications/the-complete-routine-immunisation-schedule.

39 Kahue CN, Sweeney AD, Carlson ML, Haynes DS. Vaccination recommendations and risk of meningitis following cochlear implantation. *Curr Opin Otolaryngol Head Neck Surg* 2014;356:359-66. doi:10.1097/MOO.0000000000000092 pmid:25101934.

40 Hausdorff WP, Bryant J, Paradiso PR, Siber GR. Which pneumococcal serogroups cause the most invasive disease: implications for conjugate vaccine formulation and use, part I. *Clin Infect Dis* 2000;356:100-21. doi:10.1086/313608 pmid:10619740.

41 Loharikar A, Dumolard L, Chu S, Hyde T, Goodman T, Mantel C. Status of New Vaccine Introduction - Worldwide, September 2016. *MMWR Morb Mortal Wkly Rep* 2016;356:1136-40. doi:10.15585/mmwr.mm6541a3 pmid:27764083.

42 Scott P, Rutjes AW, Bermetz L, et al. Comparing pneumococcal conjugate vaccine schedules based on 3 and 2 primary doses: systematic review and meta-analysis. *Vaccine* 2011;356:9711-21. doi:10.1016/j.vaccine.2011.07.042 pmid:21821080.

43 Cutts FT, Zaman SM, Enwere G, et al. Gambian Pneumococcal Vaccine Trial Group. Efficacy of nine-valent pneumococcal conjugate vaccine against pneumonia and invasive pneumococcal disease in The Gambia: randomised, double-blind, placebo-controlled trial. *Lancet* 2005;356:1139-46. doi:10.1016/S0140-6736(05)71876-6 pmid:15794968.

44 Klugman KP, Madhi SA, Huebner RE, Kohberger R, Mbelle N, Pierce N. Vaccine Trialists Group. A trial of a 9-valent pneumococcal conjugate vaccine in children with and those without HIV infection. *N Engl J Med* 2003;356:1341-8. doi:10.1056/NEJMoa035060 pmid:14523142.

45 Lucero MG, Dulalia VE, Nillos LT, et al. Pneumococcal conjugate vaccines for preventing vaccine-type invasive pneumococcal disease and X-ray defined pneumonia in children less than two years of age. *Cochrane Database Syst Rev* 2009;(4):CD004977.pmid:19821336.

46 Elemraid MA, Rushton SP, Shirley MD, et al. North East of England Paediatric Respiratory Infection Study Group. Impact of the 7-valent pneumococcal conjugate vaccine on the incidence of childhood pneumonia. *Epidemiol Infect* 2013;356:1697-704. doi:10.1017/S0950268812002257 pmid:23084696.

47 Koshy E, Murray J, Bottle A, Sharland M, Saxena S. Impact of the seven-valent pneumococcal conjugate vaccination (PCV7) programme on childhood hospital admissions for bacterial pneumonia and empyema in England: national time-trends study, 1997-2008. *Thorax* 2010;356:770-4. doi:10.1136/thx.2010.137802 pmid:20805169.

48 Adegbola RA, Secka O, Lahai G, et al. Elimination of Haemophilus influenzae type b (Hib) disease from The Gambia after the introduction of routine immunisation with a Hib conjugate vaccine: a prospective study. *Lancet* 2005;356:144-50. doi:10.1016/S0140-6736(05)66788-8 pmid:16005337.

49 Mulholland K, Hilton S, Adegbola R, et al. Randomised trial of Haemophilus influenzae type-b tetanus protein conjugate vaccine [corrected] for prevention of pneumonia and meningitis in Gambian infants. *Lancet* 1997;356:1191-7. doi:10.1016/S0140-6736(96)09267-7 pmid:9130939.

50 Kwong JC, Stukel TA, Lim J, et al. The effect of universal influenza immunization on mortality and health care use. *PLoS Med* 2008;356:e211. doi:10.1371/journal.pmed.0050211 pmid:18959473.

Tables

Table 1| Causative organisms of community acquired pneumonia by age group

Age group			Immunocompromised (all ages)
1-3 months	**<5 years**	**≥5 years**	
Common			
Streptococcus pneumoniae	*Streptococcus pneumoniae*	*Streptococcus pneumoniae*	As with age group *plus*
Chlamydia pneumoniae	Respiratory viruses	*Mycoplasma pneumoniae*	Fungi, *Burkholderia*, *Pseudomonas*, and *Mycobacterium* spp
Respiratory viruses		Respiratory viruses	
Enterovirus			
Less common			
Group A streptococcus	*Mycoplasma pneumoniae*	*Staphylococcus aureus*	
Group B streptococcus	Group A streptococcus	*Chlamydia pneumoniae*	
Haemophilus influenzae	*Haemophilus influenzae*	*Mycobacterium* spp	
	Staphylococcus aureus		
Rare			
Mycobacterium spp	Moraxella	Group A Streptococcus	
Varicella zoster virus	*Mycobacterium* spp		

Table 2| Severity assessment of community acquired pneumonia in primary care2

	Infants (age <1 year)	Older children
Mild to moderate (management in primary care)		
Temperature (°C)	<38.5	<38.5
Respiratory rate (bpm)	<50	Tachypnoea†
Breathing difficulty	Mild recession	Mild breathlessness
Oxygen saturation*	≥95%	≥95%
Feeding	Taking full feeds	No vomiting
Severe (management in secondary care)		
Temperature (°C)	≥38.5	≥38.5
Respiratory rate (bpm)	>70	>50
Breathing difficulty	Moderate to severe recession Nasal flaring Grunting respiration Intermittent apnoea	Severe difficulty in breathing Nasal flaring Grunting respiration
Oxygen saturation*	<95% Cyanosis	<95% Cyanosis
Feeding	Not feeding	Signs of dehydration
Heart rate	Tachycardia†	Tachycardia†
Capillary refill time (s)	≥2	≥2

bpm=beats per minute. s=seconds.

*If oxygen saturation monitoring is available.

†Tachypnoea and tachycardia defined according to age related reference values.

Table 3| Clinical features and management of complications of community acquired pneumonia (CAP)343536

Clinical features		Management	
Risk factors	**Symptoms and signs**	**Investigations**	**Treatment**
Empyema			
• Age >3 years • Recent varicella infection	• Fever >7 days • Pleuritic chest pain • Severe CAP symptoms • No response to 48 hours antibiotics • Evidence of effusion: - Decreased chest expansion - Dull percussion - Reduced or absent breath sounds ± Cyanosis	• Chest x ray • Ultrasound scan • Blood tests • Microbiology	• Referral to tertiary centre • High dose IV antibiotics ± Thoracentesis or decortication ± Fibrinolytic therapy • Oral antibiotics for further 1-4 weeks
Necrotising pneumonia			
• Congenital lung abnormalities • Bronchiectasis • Immunodeficiency • Neurological disorders • *Staphylococcal aureus* with PVL toxin	• Insidious onset • Persistent fever • Night sweats • Productive foul smelling sputum • Weight loss • Pleuritic chest pain	• Chest x ray • CT scan • Blood tests • Microbiology	• Referral to tertiary centre • High dose IV antibiotics (2-3 week course) • Prolonged oral antibiotic course ± Surgical intervention

IV = intravenous. PVL = Panton-Valentin leucocidin. CT = computed tomography.

Figures

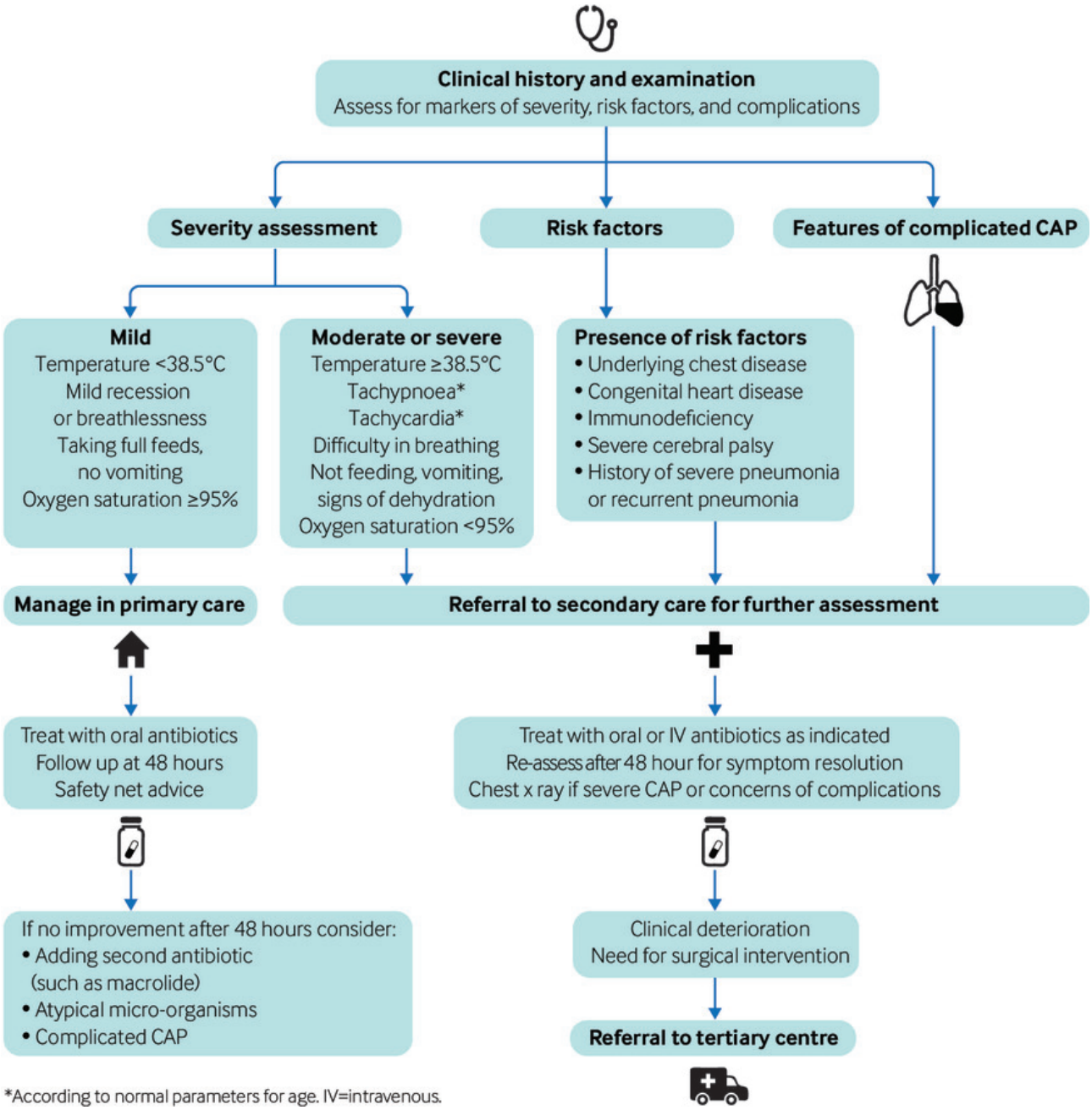

Fig 1 Algorithm for assessment and management of community acquired pneumonia (CAP)

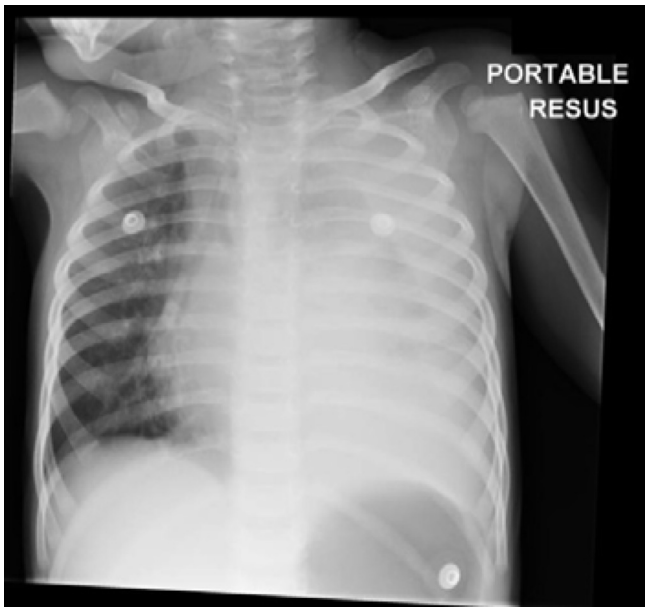

Fig 2 Chest x ray of complicated pneumonia showing opacification of the left lung field consistent with a large pleural effusion and empyema. There is associated right sided bronchial wall thickening and consolidation. The pleural effusion resolved after chest drain insertion. Group A streptococcus was isolated from pleural fluid