CRITICAL CARE FOCUS

AETIOLOGY AND OUTCOME OF SEVERE COMMUNITY-ACQUIRED PNEUMONIA IN CHILDREN ADMITTED TO A PAEDIATRIC INTENSIVE CARE UNIT

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Objective. To determine the aetiological agents and outcome of severe community-acquired pneumonia (SCAP) in children admitted to the paediatric intensive care unit (PICU) at Kalafong Hospital, Pretoria.

Patients and methods. An audit was done after a protocol was implemented to identify the aetiological agents in children with life-threatening SCAP admitted to the PICU from the emergency room. The following investigations were done as per protocol: blood culture, culture of the tracheal aspirate, immunofluorescence and culture of the nasopharyngeal aspirate, microscopy and culture of the gastric juice for *Mycobacterium tuberculosis*, and determination of HIV status. The following data, documented prospectively, were obtained from patient records: date of admission, age, gender, weight, duration of ventilation, duration of stay in the PICU, survival or death, and severity of illness as determined by means of the score for acute neonatal physiology (SNAP) or paediatric risk of mortality (PRISM) score depending on the child's age.

Results. Twenty-three children were admitted over a 1-year period (1 November 1994 - 30 October 1995). Their median age was 10 weeks (range 2 weeks - 5 years) and the sex distribution was equal.

Two children were HIV-infected. Twenty children received mechanical ventilation for a median period of 6.5 days (range 2 - 16 days). Aetiological agents were identified in 15/23 children (65%).

Respiratory syncytial virus (RSV) was the most common pathogen, identified in 7/23 children. *Klebsiella pneumoniae* was the most common bacterial pathogen, identified in 5 children (2 blood cultures and 3 tracheal aspirates).

Tuberculosis was not diagnosed. The mean PRISM score was similar in survivors and children who died. The case fatality rate was 30%. The 7 children who died had a median arterial oxygen tension/fraction of inspired oxygen (PaO_2/FiO_2) ratio of 94 (range 32 - 111) and the 16 survivors had a median ratio of 146 (range 51 - 252) ($P = 0.01$) on admission. Both HIV-infected children died and postmortem examination showed a pneumonia due to *Pneumocystis carinii* and cytomegalovirus.

Conclusions. SCAP occurs in very young children. One or more pathogens were isolated in 65% of cases. Viral pathogens predominated, with RSV being the most common. The yield of positive blood cultures was low at 17%. *Streptococcus pneumoniae* and *Haemophilus influenzae* were not found. The case fatality rate was 30% and death was more likely with a low PaO_2/FiO_2 ratio on admission.

S Afr Med J 2002; **92:** 907-911.

Pneumonia is the major cause of death in childhood, accounting for approximately 5 million deaths per year in children younger than 5 years, mainly in developing countries,[1-4] and surpassing diarrhoea as a cause of death.[5] Since the majority of developing countries do not have paediatric intensive care units (PICUs) to manage children with severe community-acquired pneumonia (SCAP) at risk of dying, a paucity of published data is available addressing the issues of the aetiology and outcome of these children when admitted to an intensive care unit (ICU).[3,6] Published data from developed countries are also not available because apart from children with bronchiolitis, SCAP is an extremely rare cause of admission to ICUs in these countries.

Identification of the most common bacterial pathogens in children with SCAP is important to aid in the decision regarding empirical antibiotics. However, it is difficult to identify bacterial pathogens in childhood pneumonia. Distinctive clinical and radiographic appearances are not indicative of specific pathogens in the majority of children.[7,8] Even though the most likely aetiological agent can be deduced from the age of the child, i.e. bacteria predominating in neonates and viruses in older children up to 5 years, with bacteria again predominating in children older than 5 years,[4,9] these findings vary depending on geographical location, time of year, and the diagnostic methodology employed.[10] This is illustrated by the fact that in poor communities the prevalence of bacterial pneumonia is higher than that of viral pneumonia in all age groups.[11,12] Blood cultures are most commonly used to identify bacterial pathogens, but the yield is notoriously low at 10 - 20%.[4,7,8] Lung aspirates are more sensitive than blood cultures and because they do not require sophisticated

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technology, have been used in developing countries to identify bacterial pathogens.[11-13] This method remains the gold standard for the identification of bacterial pathogens, but is seldom justified in cases of childhood pneumonia.[14] Antibody assays are insensitive in the aetiological diagnosis of bacterial pneumonia, particularly in children younger than 2 years of age because they respond poorly to polysaccharide antigens.[14] An additional diagnostic modality is antigen detection. However, this method is expensive and available only in sophisticated laboratories. It has been used in research studies which showed that *Streptococcus pneumoniae* and *Haemophilus influenzae* are the most common bacterial pathogens in the causation of childhood pneumonia in developed[14] and developing countries.[15] However, non-typeable *H. influenzae* remains unidentified by means of this method and its role in the causation of childhood pneumonia is probably underestimated.[14] Culture of induced sputum is an alternative to determine the bacterial aetiology of pneumonia.[16]

Guidelines regarding empirical antibiotic therapy in community-acquired pneumonia cover the most common bacterial pathogens in the different age groups,[4,9,10] but it is not known whether these guidelines can be extrapolated to children with SCAP in intensive care settings in developing countries. Although data from adult ICUs show that early empirical therapy directed at likely pathogens is more likely to improve outcome than the identification of the pathogen *per se*,[17] this evidence is not available for children.

Respiratory viruses are important pathogens in childhood pneumonia. These can be identified rapidly and with relative ease by immunofluorescence on nasopharyngeal aspirates,[14] provided that a virology service is available. However, viral and bacterial pathogens may occur concurrently, with a virus infection having set the stage for a secondary bacterial infection.[14,18]

A protocol was implemented to determine the aetiological agents of SCAP in children with respiratory failure admitted to the PICU at Kalafong Hospital in Pretoria. This study reports on an audit of the aetiological agents identified over a 1-year period after the protocol was initiated, and the short-term outcome of the children for this period.

PATIENTS AND METHODS

The 6-bed PICU at Kalafong Hospital is one of two referral centres in the Pretoria region for critically ill children between 0 and 12 years from poor communities. Children are referred from hospitals within a radius of approximately 200 km of Kalafong Hospital. The number of children admitted annually averages 330 - 350, of whom approximately 20% are non-neonates. The unit is labelled a PICU/neonatal intensive care unit (NICU) and will hereafter be referred to as the PICU.

The protocol was applied to children with SCAP admitted directly to the PICU from the emergency room. Direct admission to the PICU was indicated if intubation was carried out in the emergency room as a life-saving procedure or if the respiratory rate of a child exceeded 80 - 90 breaths per minute, with apnoea anticipated as a result of exhaustion. Radiological findings after admission to the PICU had to be compatible with pneumonia for definite enrolment into the study. After stabilising the child in the PICU the following investigations as per protocol were carried out to determine the aetiological agents: a blood culture using the BACTEC 9240 continuous monitored system, a tracheal aspirate for culture of bacterial and viral pathogens, a nasopharyngeal aspirate for immunofluorescence, followed by culture to identify respiratory viruses (respiratory syncytial virus (RSV), adenovirus, influenza, para-influenza, measles and cytomegalovirus (CMV)). Gastric juice was examined by means of rhodamine-auramine fluorochromic staining, followed by culture for *Mycobacterium tuberculosis*. All mothers were counselled regarding HIV testing of their children. If permission was obtained, screening was performed after admission to the PICU for HIV seropositivity using an enzyme-linked immunosorbent assay (ELISA) test. A positive test was confirmed by a second ELISA test, followed by a DNA-PCR test. All mothers received post-test counselling and HIV-infected children were included in the audit.

Severity of illness was determined after a stay of at least 8 hours in the PICU as is the standard procedure. The score for acute neonatal physiology (SNAP)[19] was used for children younger than 1 month, and the paediatric risk of mortality (PRISM) score[20] for children 1 month and older. The arterial oxygen tension/fraction of inspired oxygen (PaO_2/FiO_2) ratio was determined using the PaO_2 obtained on the first blood gas analysis after admission to the PICU and thereafter as a parameter of the SNAP or PRISM score. The duration of mechanical ventilation and stay in the PICU were documented. The outcome was determined as survival until discharge from the PICU.

RESULTS

Twenty-three children were admitted over a 1-year period (1 November 1994 - 31 October 1995). Their median age was 10 weeks (range 2 weeks - 5 years) and the sex distribution was equal. Six of these children were ex-prematures. Their median chronological age was 5.5 weeks (range 3 - 12 weeks) and their median weight on admission was 2.3 kg (range 1.9 - 3 kg). All children received HIV testing and 2/23 were infected.

The mean SNAP for children younger than 1 month ($N = 5$) was 15. One of these children, with a SNAP of 27, subsequently died. The mean PRISM score for the children older than 1 month ($N = 17$) was 11.8.

Twenty children received mechanical ventilation for a median period of 6.5 days (range 2 - 16 days). The median

period of ventilation for the 13 children who survived was 5 days (range 2 - 11 days) compared with 9 days (range 4 - 16 days) for the 7 children who died. The PaO_2/FiO_2 ratio was determined in 21 children on admission and ranged between 32 and 252, with a median value of 111. The median was 146 (range 51 - 252) for the survivors. The mean PaO_2/FiO_2 of the 7 children who died was 94 (range 32 - 111) ($P = 0.0054$). The median length of stay in the PICU for the group as a whole was 6 days (range 2 - 19 days). The median length of stay for the children who survived was 5.5 days (range 2 - 14 days), and 9 days (range 4 - 19 days) for the children who died. Aetiological agents were identified in 15/23 children (65%). RSV was the most common pathogen (7/15) — the single pathogen in 4 children and in combination with a viral or bacterial pathogen in 3 children. A CMV was identified in 2 patients as a single pathogen, and in combination with bacterial pathogens in 1 child. *Klebsiella pneumoniae* was the most common bacterial pathogen, identified in 5 children, 2 of whom had positive blood cultures. In 1 child serological investigation for adenovirus showed the presence of immunoglobulin G (IgG) and immunoglobulin M (IgM) antibodies suggestive of an adenovirus infection. No other pathogen was identified in this child. Four of 23 children (17%) had positive blood cultures on admission. Three of the 4 children with positive blood cultures died, including the 2 HIV-infected children. The clinical features of the 7 children with RSV pneumonia are given in Table I. All these children were admitted from April to August 1995. Three children were ex-prematures. One of the 7 children died. The characteristics of the children with RSV pneumonia were compared with those of the rest of the study group (Table II). The median age of the children with RSV pneumonia was 4 weeks (range 3 - 12 weeks), while the median age of children in whom no RSV was identified was 11 weeks (range 2 - 240 weeks) ($P = 0.05$). The median PaO_2/FiO_2 ratio and mortality did not differ between the two groups.

Tuberculosis was not identified in any of the children.

Table II. Characteristics of children with RSV pneumonia compared with the rest of the study group

	RSV ($N = 7$)	Non-RSV ($N = 16$)	P-value
Age (weeks) (median, range)	4 (3 - 12)	11 (2 - 240)	0.050
Ventilation (days) (median, range)	6 (2 - 10)	7 ($N = 13$) (2 - 16)	NS
Length of stay (days) (median, range)	7 (4 - 13)	6 (2 - 19)	NS
Prematures	3	3	NS
PaO_2/FiO_2 (median, range)	125.5 (51 - 250)	111.0 (32 - 252)	NS
Mortality (%)	14	37.5	NS

NS = not significant.

Seven of the 23 children died during their stay in the unit, with a case fatality rate of 30.4%. Twenty children were ventilated, of whom 3 had severe co-morbidity (two HIV-infected, 1 pulmonary agenesis). Of the remaining 17 children, 13 survived, probably because of the availability of an intensive care facility.

The children who died were compared with those who survived with regard to age, PRISM score, SNAP, PaO_2/FiO_2 ratio, HIV status and prematurity (Table III). The median age was not significantly different in the children who died compared with the survivors. Severity of illness as determined by the PRISM score was similar in the two groups. Death was more likely in children with a median PaO_2/FiO_2 ratio of 94 or less on admission ($P = 0.01$). The 2 children who were HIV-infected had positive blood cultures, and both died.

Consent for a postmortem examination was obtained for 5 of the 7 children who died, including the 2 HIV-infected children. The predominant pathology in all the children was in the

Table I. Characteristics of children with RSV pneumonia

Month of admission	Age (weeks)	Prematurity	PaO_2/FiO_2	Mechanical ventilation (days)	Stay in PICU (days)	Outcome
April	3	No	100	7	13	Alive
April	4	No	Unknown	6	7	Alive
April	12	Yes	109	4	4	Dead
May	3	No	51	10	11	Alive
May	5	No	250	2	4	Alive
May	10	Yes	150	4	6	Alive
August	3	Yes	142	7	12	Alive

Table III. Characteristics of survivors and children who died

	Survivors (N = 16)	Deaths (N = 7)	P-value
Age (median, range)	5 weeks (2 - 180)	15 weeks (3 - 240)	NS
PRISM (mean, range)	11.27 (N = 11) (1 - 22)	12.83 (N = 6) (8 - 22)	
SNAP (mean, range)	12 (N = 4) (11 - 13)	27 (N = 1)	
PaO_2/FiO_2 (median, range)	146 (N = 14) (51 - 252)	94 (32 - 111)	0.01
HIV-infected children	0	2	
Prematures (N = 6)	5	1	

NS = not significant.

lungs. Two of the 3 HIV-negative children had a viral pneumonia complicated by a bacterial pneumonia. The child with unilateral pulmonary agenesis had a severe bacterial haemorrhagic pneumonia. Both HIV-infected children had pneumonia due to *Pneumocystis carinii* as well as a CMV infection.

Discussion

During the 1-year period of this audit SCAP was the reason for admission in at least 30% of children older than 28 days admitted to the unit. All these children had severe pneumonia, the complications of which were life-threatening at the time of admission to the emergency room. The median age was 10 weeks, suggesting that young children are prone to SCAP, with 6/23 born prematurely.

The severity of illness of the 17 children older than 1 month, as determined by the PRISM score, was similar for the children who died and survivors. PRISM scores in the low ranges in disadvantaged children may not be a true reflection of severity of illness since they have a higher mortality than predicted.[21] The reason for this finding is not clear.[21] One of the 7 children younger than 1 month who died had a SNAP of 27, while the mean SNAP of the survivors was 12. Data from the PICU at Kalafong Hospital showed that children with a SNAP higher than 20 have a greater risk of dying (S D Delport — unpublished data). The median PaO_2/FiO_2 ratio on admission was 94 in the 7 children who died and is significantly lower (P = 0.01) than the median value of 146 for the 14 survivors. Severe hypoxaemia on admission to the PICU may therefore be a grave prognostic indicator for survival. This finding has been described by others in a similar population.[6]

Viruses were identified in 11/23 children — in isolation (6/11), in combination with another virus (2/11), or in combination with bacteria (3/11). The child with the adenoviral pneumonia also had a bacterial pneumonia which was subsequently diagnosed on postmortem examination. RSV was the most common viral pathogen (7/11). It occurred either in isolation (4/11) or in combination with another viral or bacterial pathogen (3/11). RSV pneumonia occurred only in autumn and winter. Young children and ex-prematures had RSV pneumonia, which is an expected finding.[22,23] The mortality rate of these infants was 14%, lower than the mortality rate of 30% for the group as a whole. The duration of ventilation (median 6 days, range 2 - 10 days) and length of stay (median 7 days, range 4 - 13 days) were similar to those for the rest of the study group.

A bacterial pathogen cultured from blood was found in only 4/23 children (17%); 2 of these were from the HIV-infected children. This low yield of growth on blood cultures is in keeping with findings of other studies.[4,7,8] *K. pneumoniae* was the most common bacterial pathogen isolated (5/23) — from blood on 2 occasions and from a tracheal aspirate on 3 occasions. A similar study to ours from a developing country but carried out in an adult ICU reports *K. pneumoniae* to be the second most common bacterial pathogen.[24] The bacterial pathogens traditionally regarded as being the most important in the causation of childhood pneumonia — *S. pneumoniae* and *H. influenzae* — were not isolated during the time period of the audit. Possible reasons are the insensitivity of blood cultures in detecting bacteria, and exposure to antibiotics before admission. The latter possibility could not be excluded with certainty in our study since a history is unreliable. Prior exposure to antibiotics can only be excluded by testing the serum for antibiotic activity.[25]

Severe hypoxaemia after admission, as reflected by a low PaO_2/FiO_2 ratio, may be a predictor of a grave outcome in terms of survival.

Bacterial pathogens are rarely isolated from blood cultures. *S. pneumoniae* and *H. influenzae* are not identified in children with SCAP. Possible reasons are that the diagnostic methods available at the local laboratory are not sensitive enough or that SCAP in children from developing countries is caused by bacteria other than *S. pneumoniae* and *H. influenzae*, such as Gram-negative bacilli.[16] RSV is the most common pathogen identified in children with SCAP.

Our findings relating to bacterial pathogens do not clarify the issues regarding empirical antibiotic therapy on admission. This highlights the need for prospective studies to identify the bacterial pathogens in children with SCAP admitted to ICUs in developing countries. Sputum induction in unintubated children and blind bronchoalveolar lavage (BAL) in intubated children are useful techniques[26] to identify bacterial pathogens. Blind BAL compares well with bronchoscopic BAL[27] and open-lung biopsy,[28] and this method should replace tracheal aspirates.

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EFFECT OF 1% AND 2% PROPOFOL ON BLOOD LIPIDS DURING LONG-TERM SEDATION

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Objectives. To compare the effects of 1% and 2% propofol on the maximum and average lipid levels, the relative frequency of hyperlipidaemia, the propofol dose required to achieve an equivalent degree of sedation, the pharmacodynamic effects at the required infusion rates, and the effect on respiratory function.

Design. Open, randomised, parallel group, multicentre comparison study.

Setting. Intensive care units (ICUs) at the Faculty of Medicine, University of Stellenbosch and at Vergelegen Medicity, Somerset West.

Subjects. Patients who were artificially ventilated for at least 72 hours in the ICUs and who required sedation or analgesia.

Outcome measures. Continuous intravenous infusion of 1% or 2% propofol to provide an administration rate in the range of 1 - 4 mg/kg/h. The initial infusion rate was about 2 mg/kg/h, adjusted to achieve the appropriate level of sedation.

Results and conclusions. Seventy-five patients were enrolled in the study, of which 72 were evaluable for safety analysis and 58 were evaluable for efficacy analysis. The total daily dose of propofol (ml/day) in the 2% propofol group was about 60% of that in the 1% propofol group, indicating that the lipid load in the 2% propofol group had only slightly more than half the lipid load in the 1% propofol group. Thirteen of 27 patients (48%) in the 2% propofol group had abnormally

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