Blood Cultures from Brazilian Pediatric Outpatients with Community-acquired Pneumonia (CAP)

Childhood community-acquired pneumonia (CAP) is an important cause of hospitalization and mortality in developing countries[1,2] and represents a major source of morbidity worldwide.[3] Blood culture, when positive, is the most feasible way to identify the causative organism.[4] In order to describe the role of blood culture in recovering bacteria and to assess the clinical characteristics of bacteremic patients, we attempted to identify prospectively every child with CAP coming to two Emergency Rooms in Salvador, Northeast Brazil, between September 1997 and October 1999. The diagnosis of pneumonia was based either on cough and tachypnea,[5] and/or crackles and/or radiological findings. Chest X-ray was read by the duty pediatrician. Each case was cross-referenced with the Bacteriology Laboratory log-book of the respective hospital to verify the collection of blood culture. A clinical score was created to assess for severity by summing up the presence of each clinical variable studied: cough, ability to drink, fever (axillary temperature > 37.5°C at consultation), tachypnea, chest retraction, crackles, wheezing, somnolence. Of 3431 cases 54.3 per cent were males. The median age was 1.92 years (range 2 days to 15.5 years, mean 2.87 ± 2.78 years). Tachypnea and crackles were reported in 57.7 and 68.3 per cent of the cases, respectively. Chest X-ray was performed in 93.2 per cent and a radiologically confirmed infiltrate was reported in 96.0 per cent. The rate of blood culture collection was 65.5 per cent. Those patients whose blood was collected for culture were distinctly more ill (mean clinical score for severity 3.47 ± 1.56 for patients with blood culture and 2.87 ± 1.57 for patients without blood culture, $p < 0.000001$, 95 per cent CI 0.49–0.71). The rate of contamination was 4.1 per cent. Antimicrobial use during the 72 h before the blood had been obtained for culture was reported in 17.6 per cent. *Streptococcus pneumoniae* was isolated from one patient who reported the use of trimethoprim-sulfamethoxazole. The proportions of bacterial isolations were quite different in relation to previous antimicrobial use: 0.7 per cent (2/305) and 2.3 per cent (33/1428), $p = 0.06$, but there was no significant difference in contamination rates: 3.9 per cent (12/305) and 4.2 per cent (60/1428), $p = 0.83$. Table 1 shows significant differences among children with and without isolation of pathogenic bacteria from blood. No significant differences were noticed in the frequency of cough, ability to drink, underlying chronic illness, day care center attendance, tachypnea, chest retraction, crackles, wheezing, chest X-ray performance and presence of radiological infiltrate, as well as in the distribution of gender. Table 2 shows the frequency of isolated bacteria stratified by hospital, and the demographic-clinical characteristics of the groups of patients from whom each bacterium was isolated. The rate of isolation (Table 2) was similar to those reported in several studies of the etiology of childhood CAP.[6–10] *Streptococcus viridans* were considered pathogenic as we isolated them in pure culture, and they had been isolated previously from pulmonary parenchyma.[11] *Salmonella* was isolated from the blood of two patients who were successfully treated with Penicillin G. Perhaps the *Salmonella* was in the blood stream in a course of a self-limited infection not related to the pneumonia. Young age,

TABLE 1
Association of the isolation of pathogenic bacteria from blood culture with clinical findings in pediatric outpatients with community-acquired pneumonia

Characteristic	Isolation of pathogenic bacteria		Total	*p* value
	Yes	No		
Age (years)				
Mean ± SD	1.96 ± 1.66	2.92 ± 2.80	2.90 ± 2.78	0.07
Median	1.33	2.00	2.00	
Range	0.08–6.92	0.02–15.50	0.02–15.50	
Somnolence (% nN)	34.1 (14/41)	19.0 (351/1845)	19.4 (365/1886)	0.015
Temp ≥ 37.5ºC[a] (%nN)	62.5 (25/40)	41.5 (815/1966)	41.9 (840/2006)	0.008
Hospitalization (%nN)	59.1 (26/44)	29.1 (640/2202)	29.7 (666/2246)	0.00002

[a] Axillary temperature during the consultation.

TABLE 2
Frequency of isolated bacteria stratified by hospital and demographic and clinical findings[a]

Bacteria	Hospital		Total (n = 2246)	Age (years) Mean ± SD Range	Concomitant disease[b]	Hospitalization[b]	Cough[b]	Tachypnea[b]	Fever[b]	Ability to drink[b]	Chest retraction[b]	Crackles[b]	Wheezing[b]	Somnolence[b]	Chest X-ray result[b,l]
	PCPHO (n = 1542)	AH (n = 2246)													
S. pneumoniae	14 (0.91)	4 (0.57)	18 (0.80)	1.63 ± 1.55 0.17–5.92	4/17[e]	12/18	16/18	12/17	10/17	16/18	10/18	9/18	2/17	8/16	12 alv, 3 int 2 mix, 0 pf
S. aureus	6 (0.39)	2 (0.28)	8 (0.36)	2.07 ± 0.78 1.33– 3.58	1/8[f]	1/8	8/8	7/8	3/7	7/7	4/6	7/8	5/8	0/8	4 alv, 3 int 1 mix, 0 pf
Salmonella[c]	6 (0.39)	0	6 (0.27)	1.82 ± 1.50 0.83– 4.83	3/6[g]	4/6	6/6	3/4	3/5	5/5	4/6	4/6	4/5	1/6	3 alv, 2 int 1 mix, 2 pf
Haemophilus	4 (0.26)	0	4 (0.18)	2.08 ± 2.08 0.58– 5.00	2/4[h]	2/4	4/4	2/4	2/3	4/4	1/4	3/4	1/4	1/4	2 alv, 0 int 0 mix, 1 pf
Streptococcus viridans	4 (0.26)	1 (0.14)	5 (0.22)	3.08 ± 2.47 1.00–6.92	5/5[i]	4/5	5/5	3/4	5/5	5/5	0/5	2/5	2/5	2/5	4 alv, 1 int 0 mix, 0 pf
Group B Streptococcus	0	1 (0.14)	1 (0.04)	0.08	1/1[j]	1/1	1/1	0/1	0/1	NR	NR	1/1	NR	NR	NR
E. coli	1 (0.06)	0	1 (0.04)	0.33	0/1	1/1	1/1	0/1	1/1	1/1	0/1	1/1	0/1	1/1	1 int
E. cloacae	1 (0.06)	0	1 (0.04)	5.08	1/1[k]	0/1	1/1	0/1	1/1	1/1	1/1	1/1	0/1	1/1	1 normal
Total	36 (2.33)[d]	8 (1.14)[d]	44 (1.96)												

[a] Results are reported in n(%);
[b] Number of cases;
[c] *S. enteritidis* (2), *S. cholerasuis* (1), *S. enterica* (1), *Salmonella* sp. (2);
[d] p = 0.06.
[e] asthma (1), allergic rhinitis (1), patent ductus arteriosus and atrial septal defect (1), Down syndrome and congenital heart disease (1);
[f] asthma;
[g] acute diarrhoea (1), hemolytic-uremic syndrome (1), asthma (1);
[h] acute diarrhoea (1), biliary atresia (1);
[i] acute tonsillitis (2), furunculosis (1), hemoglobin AS and malnutrition (1), patent ductus arteriosus (1);
[j] conjunctivitis;
[k] hemoglobin SS;
[l] alv = alveolar, int = interstitial, mix = mixed, pf = pleural fluid;
PCPHO = Pediatric Center Professor Hosannah de Oliveira, AH = Aliança Hospital, NR = not reported.

somnolence, axillary temperature > 37.5°C and hospitalization can be used in the clinical decision to order blood culture.

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Value of Neuron-specific Enolase Levels in Cerebrospinal Fluid in Evaluating the Prognosis of Asphyxiated Neonates

Recently neuron-specific enolase (NSE) has been reported as a biochemical marker of neuronal injury.[1,2] NSE, which is the gamma fraction of enolase, is concentrated in neurons and in neuroendocrine cells and it has been suggested that its level rises in cerebrospinal fluid (CSF) during intracerebral events in neonates.[3–7] We aimed to evaluate the value of NSE concentrations in CSF, to estimate the degree of perinatal asphyxial damage, to determine prognosis, and to identify patients at high risk of developing neurological sequelae.

Thirty-two term asphyxiated neonates were grouped as stages I–III according to Sarnat and Sarnat classification.[8] Serum glucose, lactate dehydrogenase, creatine kinase, alanine transaminase, and aspartate transaminase levels were measured, and cerebral ultrasound scanning was performed in all neonates. Glucose, chloride, albumin and NSE levels were analysed in the CSF of the cases at 72 hours of life. NSE values were measured by radioimmunoassay using ELISA NSE kit and 10 ng/ml was accepted as reference value.

NSE levels were significantly high in asphyxiated newborns (52.88 ± 27.96 ng/ml) according to the reference value (p = 0.001). Also, NSE levels of patients in stages II (45.05 ± 21.04 ng/ml) and III (77.89 ± 22.55) were higher than those of patients in stage I (22.96 ± 6.43 ng/ml) (p = 0.035, p = 0.0001).

Cerebral ultrasound revealed abnormal findings in 21 out of 32 neonates, and the presence of abnormal findings were correlated with high NSE levels in CSF (62.64 ± 28.14 ng/ml, p = 0.015). The highest levels were found in patients with intracerebral hemorrhage.

There was no correlation between CSF NSE levels and serum lactate dehydrogenase, aspartate transaminase, alanine transaminase, and glucose levels in CSF. However, a positive correlation was found between CSF NSE levels and serum creatine kinase (p = 0.04), and CSF albumin (p = 0.03) concentrations.

With respect to the prediction of outcome, NSE