ORIGINAL ARTICLE

Blood Gas Analysis in Neonatal Tetanus

Taslim S.Soetomenggolo, Dwi Putro Widodo, Jimmy Passat, Sofyan Ismael

(Department of Child Health, Medical School, University of Indonesia, Jakarta)

ABSTRACT We reviewed the results of arterial blood gas analysis in 127 patients with neonatal tetanus on admission, and in 52 of such patients on the day before they died. All patients were hospitalized at the Department of Child Health, Cipto Mangunkusumo Hospital, Jakarta. On admission, most patients showed uncompensated metabolic acidosis. The mortality of patients with pH of less than 7 was 100%. There was no significant difference between the mortality of patients with pH 7.35-7.45 and those with pH of less than 7.35. Analysis of acid-base balance indicated that venilatory failure was the most common finding in 52 patients who subsequently died. We recommend using intravenous fluid containing a combination of 5% dextrose and sodium bicarbonate with 4 : 1 (vol/vol) ratio from the first day of hospitalization to reduce the possibility of the development of ongoing metabolic acidosis in patients. **[Paediatr Indones 1994; 34:44-7]**

Introduction

Patients with neonatal tetanus usually suffer from trismus and muscle spasms. The trismus causes the patients unable to take their meal, while spasms of respiratory muscles and larynx make breathing difficulty leading to hypoxia. These two conditions will eventually lead to anaerobic metabolism resulting in lactic

Received for publication: July 26, 1993. For correspondence: T. S. Soetomenggolo, MD, Department of Child Health, Medical School, University of Indonesia, Jalan Salemba 6, Jakarta 10430, Indonesia. Tel 62-21-390-7741; Fax 62-21-390-7743.

acid accumulation (lactic acidosis).¹ Patients with severe neonatal tetanus may develop complications such as aspiration pneumonia or bronchopneumonia.²⁻⁵ Respiratory muscle and laringeal spasms, and bronchopneumonia can cause respiratory failure that may lead to death. Metabolic acidosis and respiratory failure are difficult to detect clinically; blood gas analysis is the only way to determine such alterations accurately.⁶

This study aimed to determine blood gas alterations in patients with neonatal tetanus on admission and few hours before the patients died.

Methods

Arterial blood gas analysis was performed in 127 patients with neonatal tetanus on admission, and in 52 patients on the day the patients died. All patients were hospitalized at the Department of Child Health, Cipto Mangunkusumo Hospital, Jakarta.

On the day of admission the examination was performed before the patients received intravenous fluid drip (IVFD) therapy. All blood samples were analyzed in the Intensive Care Unit Laboratory for pH, PaCO₂, PaO₂, plasma bicarbonate (HCO₃-), base excess (BE) or deficit, oxygen saturation, and standard bicarbonate. The equipment is ABL 2 Acid Base Laboratory, Radiometer Copenhagen by using method described by Astrup et al.⁷

Results

Table 1 shows the mean values of pH, $PaCO_2$, PaO_2 , bicarbonate, and base excess of 127 patients with neonatal tetanus on the day of admission. The results clearly demonstrated that in general patients with neonatal tetanus sufferred from uncompensated metabolic acidosis.

 Table 1. pH and arterial blood gas analysis of 127

 patients with neonatal tetanus on admission

	Mean	SD
pH	7.21	0.00
Pa CO ₂ (mm Hg)	45.7	0.04
Pa O ₂ (mm Hg)	83.2	0.70
HCO ₃ - (mEq/I)	17.1	0.20
BE (mEq/I)	-10.2	0.06

.....

Table 2 shows the results of pH and arterial blood gas analysis classified using criteria suggested by Shapiro et al.⁶ We noted normal blood gas in 12, acute ventilatory failure in 35, uncompensated acidosis in 48, partly compensated acidosis in 24, and compensated acidosis in 8 patients.

 Table 2. Classification of results arterial blood gas

 analysis in 127 patients with neonatal tetanus on admission

Classification	lo. of patients	%
Normal	12	9.4
Acute ventilatory failure	35	27.6
Uncompensated acidosis	48	37.8
Party compensated acidos	is 24	18.9
Compensated acidosis	8	6.3
Total	127	100.0

Table 3 shows the relationship between the pH on the day of admission and the mortality. Of the 20 patients with normal pH, 9 (45%) died, 63 (65.6%) out of 96 patients with pH of less than 7.35 died, while all 11 patients with pH of less than 7 died. The difference of the mortality between the patients with normal pH and pH less than 7.35 was not statistically significant.

 Table 3. Relationship between pH on the day of admission and mortality

рH	N	recovered	Died	%
7.35 - 7.45	20	11	9	45
< 7.35	96	33	63	65.6
< 7.00	11	0	11	100.0
Total	127	44	83	

X² = 2.99; df = 1; p > 0.05

Taslim S Soetomenggolo et al

Table 4 depicts pH and arterial blood gas analysis of 52 patients few hours before the patients died. The mean results showed acute ventilatory failure with the mean pH = 7.08, PaCO₂ = 77.2 mm Hg, $PaO_{2} = 60.8 \text{ mm Hg}, HCO_{2} = 21.7 \text{mEq/l},$ and base excess = -9.5 mEg/l.

Table 4. pH and arterial blood gas analysis of 52 patients with neonatal tetanus few hours before died

	Mean	SD
pН	7.08	0.00
Pa CO₂ (mm Hg)	77.2	1.20
Pa O₂ (mm Hg)	60.8	0.03
HCO ₃ - (mEq/l)	21.7	0.02
BE (mEq/l)	- 9.5	0.40

Table 5 shows the pH and arterial blood gas analysis of th 52 patients according to Shapiro classification.⁶ Most of From Tables 1 and 2 we can see that on those patients (76.9%) showed acute ventilatory failure.

Table 5. Classification of blood gas status in 52 patients with neonatal tetanus few hours before died

Classification	n	%
Normal	3	5.8
Acute ventilatory failure	40	76.9
Chronic ventilatory failure	1	1.9
Uncompensated acidosis	4	7.7
Partly compensited acidosis	2	3.9
Compensated acidosis	1	1.9
Compensated alkalosis	1	1.9
Total	52	100.0

Table 6 shows the comparison of pH and arterial blood gas analysis of patients with neonatal tetanus on admission and on the day before the patients died. The differences were statistically significant in all blood gas components.

Table 6. Comparison of components of arterial blood gas in patients with neonatal tetanus on admission and at few hours before died

Mean values				
	On ad- mission	Before died	t	P
pН	7.21	7.08	25.60;	< 0.001
PaCO ₂ (mm Hg)	45.7	77.2	41.78;	<0.001
PaO₂ (mm Hg)	83.2	60,8	20.34;	<0.001
HCO ₃ - (mEq/l)	17.1	21.7	11.50;	<0.001
BE (mEq/l)	- 10.2	-9.5	2.65;	<0.01

Discussion

admission most patients suffered from uncompensated metabolic acidosis. For this reason we suggest that on admission the IVFD should contain a combination of 5% dextrose and 1.5% sodium bicarbonate 1.5% with a volume / volume ratio of 4 to 1, instead of a combination of saline and dextrose usually administered.8-10

All of the 11 patients who had pH of less than 7 died (Table 3) in spite of the administration of sodium bicarbonate for correcting the base deficit. This might be due to the severity of the disease. The case fatality rate of patients with pH value between 7.35 and 7.45 was not statistically significant with that of patients with pH of less than 7.35. We presumed that this, at least in part, might be due to sodium bicarbonate treatment.

Tables 4 and 5 clearly demonstrate that ventilatory failure was the most frequently problem affecting severe patients before they died. For these patients the proper management would have been intermittent positive pressure ventilation in the Intensive Care Unit.

In conclusions, our simple data showed that in general patients with neonatal tetanus presented with metabolic acidosis, and as the disease became more severe, respiratory problem developed that might lead to ventilatory failure.

References

- 1. Plum F, Posner JB. Multifocal, diffuse, and metabolic brain diseases causing stupor or coma. In : Plum F, Posner JB, Eds. The diagnosis of stupor and coma; 3rd ed. Philadelphia: Davis, 1972; 177-284.
- Smythe PM. Studies on neonatal tetanus, and on pulmonary compliance of the totally relaxed infant. Br Med J 1963; March 2: 565-71.

- 3. Athavale VB, Pai PN. Tetanus neonatorum clinical manifestations. Trop Pediatr 1965: 67:649-57.
- 4. Rayudu GBS, Grover JM. Neonatal tetanus in a militory hospital. Indian J Pediat 1974; 41:33-9.
- 5. Salimpour R. Cause of death in tetanus neonatorum. Arch Dis Child 1977; 52: 587-94.
- 6. Shapiro BA, Harrison RA, Walton JR. Clinical approach to interpritation. Clinical aplication of blood gases, 2nd ed. London: Year Book Medical Publ. 1978; 133-44.
- 7. Astrup P, Anderson OS, Jorgensen K, Engel K. The acid-base metabolism - A new approach. Lancet 1960; 1:1035-9.
- 8. Husada TJ, Munir A. Failure of high dosage of valium in the treatment of neonatal tetanus. Paediatr Indones 1980; 20:51-6.
- 9. Soetomenggolo TS, Purboyo RH, Hendarto SK. Ismael S. Neonatal tetanus treated with diazepam as single antispasmodic agent. Paediatr Indones 1981; 21:101-6.
- 10. Sugitha N, Suwendra P, Suraatmaja S. High dosage diazepam as single antispasmodic agent in treatment of neonatal tetanus. Paediatr Indones 1983; 23: 163-72.