Introduction

Candida spp. are commonly isolated from humans, particularly in neonates and young infants below 3 years of age (Kozinn et al., 1958; Reyes et al., 1962; Soeprihatin, 1962). They often occur as saprophytes on mucocutaneous surfaces and in the gastrointestinal tract.

Significant infections with Candida spp. appear, however, to be relatively rare except in the presence of predisposing conditions such as malnutrition, impaired cellular mediated immunity or on immunosuppressive therapy, prolonged antibiotic therapy, low birth weight infants, diarrhoea, etc. (Mata et al., 1972; Oro Dewanoto et al., 1968; Suprapti Thib et al., 1968).

The excessive number of Candida spp. in the gastrointestinal tract may contribute to the production of diarrhoea by their effects on intestinal absorption of sugar (Burke et al., 1977) and fluid and electrolytes (Thelen et al., 1978).

If this condition occurs in malnourished children, the reduction of intake of food and water will increase the degree of malnutrition. Previous studies have shown the ability of such children to secrete intestinal immunoglobulins to be unimpaired (Bell et al., 1976), while depression of cell-mediated immunity in vivo and in vitro had been reported in malnutrition (Geethhuysen et al., 1971; Chandra, 1972; Ferguson et al., 1974).

This condition then will cause the increase of the numbers of Candida spp. in the gastrointestinal tract, so that it became a vicious cycle as shown in figure 1.

![Diagram](image)

The purpose of this paper is to present some results of studies in the relationship between malnutrition, gastrointestinal candidiasis and diarrhoeal diseases in Indonesian children.

Investigations

Several investigations regarding intestinal candidiasis in children with malnutrition and diarrhoea have been done. They consist of:

1. Studying the duodenal and gastric content of 21 malnourished Indonesian children with diarrhoea.
2. Studying the oropharyngeal microflora of 28 malnourished Indonesian children.
3. Isolation of Candida spp. from the gastrointestinal tract in malnourished children.
4. Investigating the frequency of intestinal Candidiasis of infants and children with diarrhoeal diseases.

Abstract

One hundred and thirteen newborns admitted to the nursery ward of the Child Health Department from June 1, 1976 until December 31, 1976 were selected for this study. They were, 34 normally delivered term infants, 36 SGA with birth-weight ranging 1750-2400 gm and 30 high risk term infants and 23 premature. Blood glucose was examined at 1, 3 and 6 hours after birth using O-Toluidine method.

It was found that in the normal term infants there was a significant increase of mean blood glucose level at 3 hours after birth (p<0.05), followed by a slight decrease at 6 hours of age. In small for gestational age and premature infants a slight increase was observed at 3 hours of age, followed by a moderate but insignificant decrease at 6 hours of age. In the high-risk-term-infants, almost no fluctuation of blood glucose level was detected throughout the study.

This study shows that 6-hour-fasting period for normal term, S.G.A. and premature infants with birth weight ranging from 1750-2500 grams, and high-risk-term infants is justified to be safe with regard to the risk of hypoglycemia.

Received 20th. February 1979.
Introduction

Hypoglycemia in the newborn has been considered a serious condition that could lead to central nervous system damage, including death. It is reported a high incidence of mental impairment among premature infants receiving delayed feeding, in spite of a lower incidence of symptomatic hypoglycemia.

Early feeding is stated to be a simple method in the prevention of neonatal hypoglycemia especially among premature infants (Smallpiece and Davies, 1964). Wu and Telfan (1966), also reported that mean blood sugar of early-fed premature infants were higher than of late fed ones.

In ours, a 6 hour-fasting period has been taken as the limit before the first oral feeding is given to normal term, high-risk-term, and mild-low-birth-weight infants (Birth weights ranging from 1750-2500 grams).

This prospective study was performed to confirm whether a 6 hour-fasting-period is a safe duration regarding the possibility of the occurrence of hypoglycemia with or without clinical symptoms in the above mentioned newborn infants.

Material and Methods

The 113 vigorous infants (Apgar Score 7 or more), who were selected for this study, were admitted to the neonatal ward, Department of Child Health Gunung Wenang Hospital Medical Faculty, Sam Ratulangi University Manado, Indonesia, from June 1, 1976 until December 31, 1976.

They consisted of the following:

— 34 normally delivered term infants.
— 30 high risk term infants.
— 26 small for gestational age infants.
— 23 premature infants.

In this study, the small for gestational age and premature infants who were chosen had birth weights ranging from 1750-2500 grams.

The gestational age was determined from the last menstrual period. Blood glucose concentration was determined at 1, 3 and 6 hours after birth before the first feeding was given. Capillary blood, taken from a heelprick, was examined using O-Toluidine methods by one of the authors (R.E.J.R.).

From those 4 groups of infants, the mean gestational age, birth weight, the mean blood glucose at 1, 3 and 6 hours and the incidence of hypoglycemia can be seen in table 1.

Terminology:

The uniformity of terminology is of paramount importance for coming to the same conclusions of every study.

Terminology used in this study were as follows:

Normal term infants: Vigorous baby of normal pregnancy with a birth weight of 2500 grams, and a gestational age of 37 weeks.

Premature infants: Newborn infant with a birth weight of less than 2500 grams and a gestational age of 37 weeks or less.

Small for gestational age term infant:

The newborn with a birth weight of less than 2500 grams and a gestational age of 37 weeks or more.

Normal high-risk-infant:

Vigorous baby of high risk pregnancy with a birth weight of 2500 grams or more, and a gestational age of 37 weeks or more. Only newborn infants born with S.C. were chosen.

Hypoglycemia:

A true blood glucose of 30 mg/100 ml or less in term and 20 mg/100 ml or less in small for gestational age and premature infants, with or without clinical symptoms during 72 hours of age.

Results

Table 1 shows the mean blood glucose concentration of each group.

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>High risk</th>
<th>Small for gestational age</th>
<th>Premature</th>
</tr>
</thead>
<tbody>
<tr>
<td>No of infants</td>
<td>34</td>
<td>30</td>
<td>26</td>
<td>23</td>
</tr>
<tr>
<td>Male/Female</td>
<td>10/24</td>
<td>6/20</td>
<td>23/15</td>
<td>13/10</td>
</tr>
<tr>
<td>Mean Birth Weight (gram)</td>
<td>3250 ± 475.3</td>
<td>3260 ± 436.6</td>
<td>2302 ± 146.5</td>
<td>2192 ± 183.5</td>
</tr>
<tr>
<td>Mean gestational age (months)</td>
<td>3950 ± 0.95</td>
<td>3927 ± 1.02</td>
<td>3946 ± 0.81</td>
<td>3953 ± 2.1</td>
</tr>
<tr>
<td></td>
<td>(38 - 40)</td>
<td>(38 - 40)</td>
<td>(38 - 40)</td>
<td>(30 - 37)</td>
</tr>
<tr>
<td>Mean Blood Glucose (1 hour) (mg/100 ml) (Mean + SD)</td>
<td>52.3 ± 22.6</td>
<td>57.9 ± 21.1</td>
<td>56.8 ± 22.4</td>
<td>46.4 ± 22.4</td>
</tr>
<tr>
<td>Mean Blood Glucose (3 hours) (mg/100 ml) (Mean + SD)</td>
<td>62.4 ± 17.3</td>
<td>56.2 ± 21.96</td>
<td>59. ± 17.7</td>
<td>50.5 ± 17.9</td>
</tr>
<tr>
<td>Mean Blood Glucose (6 hours) (mg/100 ml) (Mean + SD)</td>
<td>60. ± 18.8</td>
<td>59.9 ± 21.08</td>
<td>53. ± 21.6</td>
<td>46.1 ± 16.7</td>
</tr>
<tr>
<td>Hypoglycemia:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 hour</td>
<td>3 (8.8%)</td>
<td>3 (10%)</td>
<td>—</td>
<td>2 (8.7%)</td>
</tr>
<tr>
<td>3 hours</td>
<td>—</td>
<td>2 (6.7%)</td>
<td>1 (3.8%)</td>
<td>1 (4.3%)</td>
</tr>
<tr>
<td>6 hours</td>
<td>3 (8.8%)</td>
<td>1 (3.3%)</td>
<td>1 (3.8%)</td>
<td>1 (4.3%)</td>
</tr>
</tbody>
</table>

It was found that there was a significant increase of mean blood glucose level at 3 hours after birth in the normal term infants (p < 0.05), followed by a slight decrease at 6 hours of age. In the mild low-birth-weight infants, a slight increase was observed at 3 hours postnatally, followed by a moderate decrease at 6 hours of age which was not significant. Almost no fluctuation of blood glucose
level was observed in the high risk term infants. The linear correlation can be seen in Figure 1.

**Discussion**

The mean blood glucose curves of normal term, small for gestational age, and premature infants were similar at 3 hours of age when the mean blood glucose level increased, and again at 6 hours of age when the level decreased.

Contrary to the above findings, in normal-high risk infants, the mean blood glucose curve was almost flat. The mean blood glucose level at 1 and 3 hours of age was almost the same and slightly increased at 6 hours of age (fig. 1).

In term infants the mean blood glucose level falls rapidly to about 50 mg/100 ml after birth (Cornblath and Reisner, 1965; Shelly and Nelligan, 1966). This value is similar to the result of this study where the mean blood glucose level of normal term infants at 1 hour of age was 52.3 mg/100 ml. Then at 3 hours of age the mean blood glucose level was 62.4 mg/100 ml. This is brought about by the sufficient glycogen reserve in their liver, the proper enzymatic and hormonal system, and the increased metabolic rate due to the alteration of ambient temperature.

In small for gestational age and premature infants, the mean blood glucose level increased insignificantly from 56.8 mg/100 ml and 46.4 mg/100 ml at 1 hour of age to 59 mg/100 ml and 50.5 mg/100 ml at 3 hours of age respectively. And then at 6 hours of age decreased respectively to 53 mg/100 ml which was lower than that of 1 hour of age, and 46.1 mg/100 ml which was the same level as that of 1 hour of age. This phenomenon might be due to insufficient glycogen reserve, and improper enzymatic and hormonal systems.

It seems that the risk of the occurrence of hypoglycemia is much higher in small for gestational age infants after 6 hours of fasting period, since the mean blood concentration at 6 hours drop to a level lower than that of 1 hour of age.

The mean blood glucose level of small for gestational age and normal term-high risk infants at 1 hour of age was almost the same and was higher than normal term infants. Shelly and Nelligan (1966) stated that hypoxia occurring during delivery could effect a relatively high blood glucose concentration in most babies at birth. However, we also believe that the higher level of blood glucose in both groups may be due to some metabolic changes, caused by some conditions of environmental stress during intra-uterine life or shortly before delivery.

Despite the fluctuation, normal term and high-risk term infants had a higher mean blood glucose level at 6 hours of age than at 1 and 3 hours of age. But in small for gestational age infants this value at 6 hours of age was lower than at 1 and 3 hours of age. So, this study proved that 6 hours of fasting for small for gestational age and premature infants is still a safe period regarding
the risk of hypoglycemia. However, vigilance remains necessary since the total incidence of hypoglycemia at 1.3 and 6 hours of age was 7%, 4% and 5% respectively.

Luchchenco and Bard (1971) reported that 11.4% of infants with blood glucose less than 30 mg/100 ml in a general nursery population occurred before the first feedings at 6 hours of age.

The institution of oral glucose solutions shortly after birth might be necessary in an attempt to prevent asymptomatic hypoglycemia.

Surprisingly, the incidence of asymptomatic hypoglycemia in small-for-gestational age in this study were lower than those reported in western literature.

It is well documented in western literature that the incidence of hypoglycemia is very high in small-for-gestational age infants (Luchchenco and Bard, 1971; de Leeuw and de Vries, 1976; Campbell et al., 1967; and Blum et al., 1969). Only 3.8% of our small-for-gestational age were hypoglycemic at 3 and 6 hours of age, and none of them was hypoglycemic at 1 hour of age.

From this finding, we concluded that many factors account for the development of hypoglycemia rather than a lack of glycogen reserve only.

REFERENCES


