EFFECTS OF INTRAOPERATIVE FLUID THERAPY DURING CRANIOTOMY ON POSTOPERATIVE GLYCOLYSIS AND SERUM ELECTROLYTE LEVELS

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SUMMARY

There is a controversy in administering fluid therapy containing glucose intraoperatively to neurosurgical patients. Patients with intracranial mass already face the risk of cerebral hypoperfusion. To study the effects of intraoperative glucose administration of postoperative outcome, different fluid therapy was administered to each of three groups of patients undergoing craniotomy. One group received physiological saline (PS), the second group received Ringer solution without lactate (R), and the third group received 5% dextrose solution (D). Venous blood levels of glucose, lactic acid, pyruvic acid, Bhydroxybutyric acid (βHBA), sodium, potassium, chrolide, acetone, and arterial pCO2, pO2, and bicarbonate were estimated at induction of anaesthesia and 6, 12, 24, 48 hours postoperatively. Urinary acetone was also estimated. Obtained values were compared between the groups and their relation to each other were analyzed within each group. Blood glucose rose in all three groups but no significant difference was found in the glucose levels among the three groups, contrary to what has been expected.

Key Words: Craniotomy, fluid therapy, neurosurgery, intraoperative glucose.

INTRODUCTION

There have been different opinions regarding glucose administration during intracranial surgery. Glucose administration produces protein sparing in starving individuals, decreases fat and protein mobilization during a short fast, and provides free water (1,2). Glucose administration during neurosurgery has been preferred for these reasons. However, recent animal and human studies suggest that hyperglycemia exacerbates ischemic brain damage and may, therefore harm patients whose operations are associated with periods of decreased cerebral perfusion (3-5). Decreased cerebral perfusion to levels that may produce ischemia may occur during brain tumor resection with its attendant brain retraction and hypotension (6). During ischemia, high glucose levels in the brain tissue may lead to neurological and histopathological injury. Anesthesia and surgical stress can cause an increase in cortisol levels, which may in turn cause an increase in serum glucose content (7). In addition to being a stress response, hyperglycemia may directly influence outcome. In patients with intracranial tumor or aneurism, focal cerebral hypoperfusion may occur (8). This may be due to local tissue pressure, vasospasm or edema. The applied anesthetic and surgical procedure also exacerbates the situation (3,4). Glucose administered to such a patient is converted to pyruvate and lactate because of insufficient oxygen supply and finally cellular acidosis and irreversible cell injury occurs (9-13).

The aim of this study was to observe the postoperative biochemical effects of different fluid therapies administered intraoperatively on blood glucose and electrolyte levels in craniotomy patients and to show refraining from glucose can be beneficial for the patients and mininimizes the risks. To this end patients undergoing craniotomy were given three different fluid therapies and blood glucose, electrolytes, pyruvate, lactic acid, βHBA were monitored.

SUBJECTS AND METHODS

Thirty adult craniotomy patients ages between 21-76 were included in our study. Patients were selected randomly among those who did not have renal, cardiovascular or endocrine system disorders; those that required blood transfusion were not included in the study. In addition those patients who were on steroid therapy during the preoperative period were excluded. Age, body weight, duration of surgery and anesthesia are given in Table I. All patients were premedicated with 0,5 mg atropine sulphate forty-five minutes prior to surgery.

Patients were divided randomly into three groups; one group received only Physiological Saline (PS), the second group received only Ringer Solution without Lactate (R), and the third group received 1000 ml 5% Dextrose (D) followed by PS. Blood levels of glucose, lactic acid, pyruvic acid, βHBA, sodium, potassium, chloride, pCO2, pO2, bicarbonate, and acetone were estimated, urinary acetone levels were also monitored at induction of anesthesia and 6, 12, 24, 48 hours postoperatively. All patients received 0.08 mg/kg pancuronium bromide, 5 mg/kg sodium thiopental iv. during induction period and then they were tracheally intubated. They were also administered 1 mg/kg furosemide, 0.5 mg/kg 20% mannitol. After the induction period, anesthesia was continued with 2 l/min oxygen, 4 l/min nitrous oxide and 1 MAC isoflurane in all patients and they were mechanically ventilated so as to sustain 10 ml/kg tidal volume. Arterial and venous pressures were constantly monitored and the volume of urine was measured. All patients received 200 ml PS postoperatively as soon as the surgery ended. Blood was taken 6, 12, 24, 48 hours after the start of 5% dextrose solution postoperatively. Fluid diet was started after 24 hours.

Arterial blood was taken with heparin for blood gas analysis which was performed with Nova Biomedical Stadt Profile 3. All other parameters were measured in venous blood. βHBA determination was performed in serum by Sigma kit enzymatically. Boehringer kits were used in the determination of pyruvate and lactate. Serum glucose was measured with Abbott Spectrum by hexokinase method, Serum chloride levels were measured by Technicon RA 1000. Serum sodium and potassium measurements were done with KNA 1 Ion selective electrode potentiometer. Acetone in serum and urine was estimated with Rothera's reagent.

Statistics: Chi square test, One Way Anova, Kruskal Wallis nonparametric Anova test, multiple regression analysis were used in the evaluation of the results.

RESULTS

No significant difference was found in average age or body weight of the patients included in the study, duration of anesthesia and operation time between the three groups (Table I). Average blood glucose and pyruvate levels had increased in all sampling periods as shown in Table II. Increase of blood glucose levels in the R and the PS groups can be explained to be due to surgical stress (14). Average lactate values showed an increase at all times in the R group but only at 6., 12., 24. hours in the D group.

Average glucose, pyruvate, lactate, and βHBA values are shown in Table II. Among the blood gas values, only pO2 rose due to mechanical hyperventilation with oxygen enriched gas which caused both a rise in pO2 and a fall in pCO2. As a result of respiratory alkalosis due to hyperventilation, low levels of pCO2 and HCO3 were seen in all groups. Although all the electrolyte values were in the normal range, sodium and chloride showed an increase in the R and the PS groups.

DISCUSSION

Average values for each parameter at all sampling times were compared between and within groups with the Anova test (Table II). Comparisons within groups revealed significant differences only between average glucose values in the R group at different time intervals when compared to the induction value. A second but less significant relation was observed between the average glucose values in the S group (within group) at 6 hrs. Contrary to our expectation, no significant differences were found between the groups in the average blood levels of glucose and glycolytic metabolites (Table II). Correlation between the parameters within each group was statistically determined by Multiple regression test (Table III). A strong multiple correlation was found between the urinary acetone and serum glucose, pyruvate, lactate, and BHBA altogether (r=0.9540) for the R group at 6 hrs and 12 hrs (r=0.9779). One to one analysis showed the expected reverse relation between the urinary acetone and serum glucose (r= -0.8684) for the R group at 12 hrs. A strong multiple correlation was obtained for the D group at 48 hrs between urinary acetone and serum glucose, lactate, pyruvate, and BHBA taken together (r=0.9713). Correlation between lactate and pyruvate was found to be strong (r=0.9379) in the S group at 48 hrs while it was less strong (r=0.8663) in the D group.

As a result, type of intraoperative fluid therapy had no significant effect on blood glucose levels, glucose metabolites and blood gas levels in this study. Hypoglycemia and increase in blood βHBA was not observed in the PS and the R groups. Risk of enhancing ischemic damage by giving glucose is a major consideration during surgical procedures where the risk of brain ischemia is increased. Patients with poor neurological outcome have shown to be those with significantly higher serum glucose than patients with good neurological outcome (7). It is well known that carbohydrate metabolism is profoundly affected by anesthesia as a result of increasing symphathetic activity. Increase in blood levels of cortisol, epinephrine and growth hormone as a result of surgical stress also leads to hyperglycemia (15). To conclude, we believe that it is appropriate not to administer glucose intraoperatively. Monitoring blood glucagon, insulin, free fatty acids, calcium levels, and plasma osmolality can give a deeper insight into the situation.

Table I- Average age, body weight, duration of surgery and anesthesia: (Mean + SD)

	Group 1	Group 2	Group 3
Age (y)	45 (12.4)	44 (10.7)	46 (14.8)
Weight (kg)	62.2 (19.3)	64.2 (24.4)	65.3 (17.3)
Surgery (m)	210.0 (86.4)	230.0 (56.4)	225.0 (86.6)
Anesthesia (m)	270.0 (64.2)	240.0 (94.7)	245.0 (37.1)

Group 1: Physiological saline; Group 2: Ringer: Group 3: Dextrose y: age in years;m:minutes
Values in paranthesis are standard deviations

Table II- Average serum glucose, lactate, pyruvate, and βHBA values for ± SD for the Physiologic Serum (S), Ringer (R), Dextrose (D) groups.

	Induction	6 hrs	12 hrs	24 hrs	48 hrs
Glucose S	93.6* ± 12.0	155.8* ± 44.7	142.4 ± 29.4	152.0 ± 22.2	158.0 ± 42.9
Glucose R	89.9+#$ ± 13.2	181.7+ ± 59.9	164.3 # ± 56.6	141.1 ± 41.9	165.0 $ ± 44.5
Glucose D	101.7 ± 22.4	150.8 ± 70.9	146.2 ± 49.9	157.2 ± 39.5	159.1 ± 42.4
Lactate S	27.0 ± 11.7	31.5 ± 16.4	25.4 ± 16.4	20.4 ± 7.4	26.3 ± 9.7
Lactate R	21.6 ± 7.8	32.0 ± 13.9	31.4 ± 16.2	26.5 ± 11.2	23.9 ± 5.4
Lactate D	18.4 ± 5.3	33.0 ± 17.4	29.6 ± 13.7	24.6 ± 10.5	20.4 ± 7.2
Pyruvate S	0.8 ± 0.4	1.6 ± 1.0	1.5 ± 0.7	1.3 ± 0.7	1.1 ± 0.4
Pyruvate R	0.8 & ± 0.4	2.0 & ± 0.7	1.3 ± 0.7	1.1 ± 0.5	1.2 ± 0.4
Pyruvate D	0.9 ± 0.1	1.5 ± 1.0	1.5 ± 0.7	1.3 ± 0.5	1.1 ± 0.5
βHBA S	5.6 ± 3.8	4.7 ± 3.7	5.1 ± 2.9	4.3 ± 2.3	4.2 ± 2.4
βHBA R	5.1 ± 3.4	4.8 ± 3.1	4.5 ± 2.6	5.1 ± 2.4	4.2 ± 1.6
βHBA D	3.4 ± 1.7	4.4 ± 2.1	3.7 ± 1.8	3.3 ± 2.4	3.6 ± 2.2

Significant differences found by nonparametric Kruskal Wallis Anova Test
* = p<0.05 +=p<0.001 #=p<0.05
$ = p<0.001 &=p<0.01

Table III- Results of the regression analysis:

HOUR	GROUP	DEPENDENT VAR	INDEPENDENT VAR	r
6	R	Urinary Acetone	Glu, Pyr, Lac, HBA	0.954
12	R	Urinary Acetone	Glu	- 0.868
12	R	Urinary Acetone	Glu, Pyr, Lac, BHA	0.978
12	PS	Pyr	Lac	0.838
48	R	Urinary Acetone	Glu, Pyr, Lac, BHA	0.866
48	D	Pyr	Lac	0.868
48	D	Urinary Acetone	Glu, Pyr, Lac, BHA	0.971
48	D, R, PS	Pyr	Lac	0.906

r: correlation coefficient

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