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Influence of Enteral Nutrition on Postoperative Hyponatremia

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Abstract

Introduction: Postoperative hyponatremia is still not fully understood. Some of the explanations presented are based on dilutions with low-sodium fluids, the influence of anti-diuretic hormone, the desalination process, and the "sick cell syndrome" with re-distribution of sodium and increased cell membrane permeability, but the authors of these trials are still looking for further evidence.

Purpose: To provide information on postoperative hyponatremia in combination with early postoperative enteral nutrition.

Methods: S-sodium, s-potassium, and s-creatinine were analysed daily during a random double blind prospective trial in which 30 patients received Nutridrink® and 30 patients received a placebo (water) through a nasoduodenal tube from the day of the operation to the 4th postoperative day. The patients were mobilised from the 1st postoperative day and weighed every day on the same scales. Twenty-four hour urine was collected one day preoperatively, the day of the operation, and postoperatively on days 1 through 5 and the urinary creatinine was measured.

Multivariate analysis of variance for 5-7 related means (MANOVA) was used in a combined design with nutrition and placebo as between-subject factors and combined with univariate F tests.

Results: S-sodium values were significantly lower and s-potassium values significantly higher in the placebo group on postoperative days 2 and 3, although the two groups received the same amount of intravenous sodium, and the placebo group received significantly less potassium.

Conclusions: The result indicates that early postoperative enteral nutrition ensures the energy supply to the cell (Na/K pump) in the postoperative period and prevents sodium being redistributed intracellularly during postoperative stress.

Keywords: Postoperative enteral nutrition, Hyponatremia, Surgery, Sodium, Potassium

Introduction

One hypothesis about postoperative hyponatremia concerns the postoperative administration of sodium-free or sodium-low intravenous fluids in the postoperative period [1,2] while the antidiuretic hormone is functioning [1,3]. Aronson et al. mentioned impaired urinary dilution capacity as being a factor in hyponatremia [3]. Another explanation for hyponatremia could be the desalination process with the excretion of hypertonic urine [1]. Trials exploring these hypothesises have concluded that even when no potentially hypo-osmolar fluids [2] or near-isotonic fluids were infused, postoperative hyponatremia occurred within 24 hours of the induction of anaesthesia [1]. It was concluded that plasma-sodium falls following surgery of any severity and that such falls occur even when exogenous dilution cannot be implicated as a cause [1,2]. Finally, new results have shown that hypo- or dysnatraemia has a negative effect on outcome [4,5]. The results presented cover the relationship between postoperative hyponatremia and early postoperative enteral nutrition and were obtained during a randomised trial, which examined the influence of enteral nutrition on postoperative complications [6].

Materials and Method

S-sodium, s-potassium, and s-creatinine were analysed daily during a prospective, double blinded and placebo controlled trial. Sixty patients were randomly allocated to two comparable groups of 30 patients each: a "nutritional" group and placebo roup.

All patients scheduled for elective major gastrointestinal surgery at the Department of Surgical Gastroenterology, Glostrup University Hospital, Denmark, were considered for inclusion, irrespective of nutritional status. Patients with diabetes mellitus, inadequate renal or hepatic functions, or inflammatory bowel disease were excluded.

All patients gave informed preoperative consent to participation before inclusion. Randomisation was done after exploring the abdomen to ensure the viability of the intended procedure.

A nasoduodenal feeding tube (Flocare 125 cm, Ch. 10) was positioned in the 2nd to 3rd part of the duodenum at the end of the operation. The patients in the "nutritional" group received nutritional supply with Nutridrink (orange taste), postoperatively within four hours, 600 ml on the day of surgery, increasing by 400 ml each following day until the fourth postoperative day. Nutridrink (Nutricia, The Netherlands) contains 150 Kcal/100 ml, 5 g protein/100 ml, sodium 80 mg/100 ml, potassium 150 mg/100 ml and is hyperosmolar (390 mOsm/l). The patients in the placebo group received the same quantity of orange flavoured water, no energy content, vitamins, or trace elements. All patients were permitted to eat a normal diet from the fifth postoperative day. Tea, coffee, and water were allowed from the day of

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operation. Isotonic saline and glucose was given intravenously until the patient was able to drink sufficiently. All fluids were recorded.

All patients received prophylactic antibiotics (penicillin, metronidazole, and gentamicin) and surgery was performed according to the routine of the department.

All patients had combined thoracic epidural and general anaesthesia. Postoperative pain relief was achieved with a continuous infusion of 0.2 mg/h morphine and 10 mg/h bupivacaine in the epidural catheter for at least 48 hours postoperatively.

The patients were mobilised from 1st postoperative day and weighed every day on the same scales. Twenty-four hour urine was collected one day preoperatively, the day of the operation, and postoperatively on days 1 through 5 and urinary creatinine was measured.

Ethics

The study complied with the Helsinki Declaration II.

Trial registration: The protocol of this study was approved by the Copenhagen County Scientific Ethics Committee (H-KA-93174). The study was initiated in 1993, before it was mandatory to register the trial on a public database.

Statistics

All results are indicated by mean values and standard deviation. According to the power of the study, it was chosen that 2 alpha and beta=0.05 and MIREDIF=SD in changes in blood samples meaning 30 patients in each group.

Multivariate analysis of variance for 5-7 related means (MANOVA) was used in a combined design with nutrition and placebo as between-subject factors and combined with univariate F tests. A p value <0.05 was considered significant. SPSS Release 10 for Windows was used for data analysis. This means that all measurements on a single parameter from preop day to POD 5 were primarily compared between groups and if any significance was found tests for difference every day was done.

Results

Table 1 shows the demographic data. No significant differences were found.

Intravenous and oral supplies are shown in Table 2.

The multivariate analyses of repeated measurements showed no significant difference between the treatment groups, irrespective of supply The univariate analyses showed that the placebo group consumed a significantly larger amount of product on postoperative day 1 (POD 1) and a significantly higher total supply on postoperative day 2 (POD 2).

	Nutrition group (range)	Placebo group (range)
Age (median)	66.5 years (27-93)	61.5 years (27-80)
Male/female	18/12	20/10
Height/weight (mean)	169.5 cm/71.2 kg	172 cm/68.5 kg
Patients with malign disease	19	20
Number of malnourished patients	4	5
Operation length (median)	170 minutes (60-365)	165 minutes (60-365)
Number of severe complications	6	6

Table 1: Demographic data.

In Table 3 the MANOVA shows no difference between the treatment groups in the amount of diuresis, weight changes (exact change in kilogram or percentage compared to preoperative weight), urine-creatinine, or deficit calculated for each day (difference between daily total supply and daily diuresis). There was a significant difference in serum-creatinine in the treatment groups in the multivariate analysis, the nutrition group varying less from the preoperative value.

The univariate analyses showed a significantly larger amount in the daily secretion of urine-creatinine in the nutrition group on postoperative day 4 (POD 4).

In Table 4, the results of the MANOVA showed significant differences between the nutrition group and the placebo group in s-sodium and s-potassium from the preoperative day through postoperative day 5 (POD 5). The univariate tests showed significant differences on POD 2 and POD 3, the nutrition group having an almost unchanged s-sodium compared to preoperative value and the placebo group having a significantly lower value compared to the nutrition group. Table 4 also shows the input of sodium and potassium to be significantly different.

The univariate test showed that the placebo group received significantly more sodium on POD 1 and the nutrition group received significantly more potassium every day from the day of operation through POD 5.

Discussion

A fluid resuscitation with crystalloid solutions is normally used in major abdominal surgery. Hyponatremia (s-sodium level<136 mmol/L) is the most frequent electrolyte disorder and is associated with an increase of morbidity and mortality [4,5].

During the day of operation, the patients in this study received more than seven litres of mainly isotonic sodium chloride. They had a positive deficit of almost 5 litres and on POD 1 a weight gain on 1.5-3.0% of their preoperative weight. This is in accordance with a study of vascular surgery patients, which demonstrated that patients who received crystalloid solutions preoperatively and postoperatively had a weight gain POD 1 of 2.3-4.5 kilogram. This trial also demonstrated that weight changes paralleled the changes in the extracellular volume (ECV) [7].

Although the patients received isotonic sodium chloride, the placebo group suffered a significant decrease in s-sodium on POD 2 and POD 3 and developed hyponatraemia on POD 2, while the nutrition group showed no sign of low s-sodium in the same period. In total, our patients in both groups received more than 1400 mmol sodium from the day of the operation through POD 5. The results of decreased s-sodium-despite the use of isotonic fluid in the placebo group -are comparable to results obtained in other trials using isotonic fluids [1,2]. There was no difference in urine production, indicating that a change in kidney function due to the increased desalination process in the placebo group was not the reason for hyponatremia on POD 2 and POD 3. Naturally one of the weaknesses of the study is that it is unable to demonstrate the output of sodium in the urine. The lower values of s-creatinine in the placebo group could indicate a dilution of ECV in this group.

Another explanation for the postoperative hyponatremia is the "sick cell syndrome" in which -during surgery-intracellular solutes may leak out of cells because of increased membrane permeability leading to increased osmolality, cellular water shift, and redistribution hyponatremia.

	Day of operation Mean (SD) ml/day	POD 1 Mean (SD) ml/day	POD 2 Mean (SD) ml/day	POD 3 Mean (SD) ml/day	POD 4 Mean (SD) ml/day	POD 5 Mean (SD) ml/day
Intravenous supply						
Placebo	6388 (2136)	2222 (1116)	1725 (1045)	1001 (972)	550 (963)	739 (1330)
Nutrition	6423 (2413)	1868 (1174)	1314 (1037)	1084 (1084)	648 (1345)	396 (1205)
Extra water						
Placebo	80 (196)	436 (534)	540 (649)	575 (551)	513 (655)	409 (939)
Nutrition	171 (438)	631 (712)	544 (704)	565 (872)	433 (588)	247 (541)
Product						
Placebo	583 (74)	964 (92)†	1196 (442)	1005 (699)	755 (595)	93 (271)
Nutrition	571 (114)	837 (319)	986 (473)	810 (638)	591 (590)	203 (405)
Total supply						
Placebo	7057 (2066)	3658 (1352)	3572 (1133)†	2694 (1092)	1908 (881)	1278 (1516)
Nutrition	7195 (2392)	3361 (1338)	2962 (962)	2514 (1289)	1818 (1364)	937 (1455)

†p<0.05 comparison between groups (univariate F test)

Table 2: Information of supply from the day of operation to postoperative day 1-5.

	Preoperative measurement (baseline) Mean (SD)	Day of operation Mean (SD)	POD 1 Mean (SD)	POD 2 Mean (SD)	POD 3 Mean (SD)	POD 4 Mean (SD)	POD 5 Mean (SD)
Diuresis l/day							
Placebo	1.9 (1.0)	2.2 (0.9)	1.9 (1.0)	2.8 (1.2)	2.3 (1.0)	1.9 (1.3)	1.7 (1.0)
Nutrition	1.8 (1.1)	2.4 (1.1)	1.9 (1.0)	2.4 (1.2)	1.7 (1.0)	1.5 (1.1)	1.4 (0.9)
U-creatinine mmol/day							
Placebo	5.8 (3.4)	5.1 (2.2)	7.7 (5.6)	4.4 (2.7)	5.8 (3.9)	5.8 (4.3)†	7.3 (3.7)
Nutrition	6.2 (3.0)	5.2 (3.0)	7.7 (5.1)	5.9 (5.0)	7.5 (4.4)	9.4 (6.5)	10.9 (8.8)
S-creatinine* mmol/l							
Placebo	0.080 (0.018)	0.071 (0.019)	0.080 (0.021)	0.076 (0.018)	0.078 (0.021)	0.087 (0.023)	0.091 (0.026)
Nutrition	0.085 (0.021)	0.072 (0.019)	0.084 (0.025)	0.083 (0.028)	0.081 (0.029)	0.082 (0.031)	0.087 (0.025)
Weight change Kg							
Placebo			1.22 (1.68)	1.26 (1.66)	0.48 (1.53)	- 0.98 (1.58)	-1.62 (2.12)
Nutrition			2.09 (1.91)	1.93 (2.17)	1.16 (1.53)	0.26 (2.18)	-0.33 (2.06)
Weight change Percent (%)							
Placebo			1.55 (2.29)	1.65 (2.15)	0.62 (2.27)	-1.30 (2.40)	-2.05 (2.70)
Nutrition			3.01 (2.40)	2.83 (3.45)	1.82 (2.46)	0.64 (3.67)	-0.20 (3.05)
Deficit kg/day							
Placebo		4.95 (1.95)	1.79 (1.67)	0.82 (1.14)	0.45 (1.13)	-0.03 (1.45)	-0.50 (1.62)
Nutrition		4.97 (2.55)	1.75 (1.46)	0.78 (1.38)	1.01 (1.21)	0.60 (1.67)	-0.35 (1.71)

Weight change compared to preoperative weight. Deficit: total input (intravenous supply, product and oral water) minus diuresis

*p<0.05 (repeated measures multivariate test (Pillai's trace)) †p<0.05 comparison between groups (univariate F test)

Table 3: Diuresis and weight charges.

	Preoperative measurement (baseline) Mean (SD)	Day of operation Mean (SD)	POD 1 Mean (SD)	POD 2 Mean (SD)	POD 3 Mean (SD)	POD 4 Mean (SD)	POD 5 Mean (SD)
S–sodium* mmol/l							
Placebo	138 (2.8)	140 (3.3)	138 (3.4)	134 (2.5)‡	135 (2.6)†	136 (3.6)	136 (3.4)
Nutrition	139 (2.0)	141 (3.2)	140 (2.8)	138 (2.7)	137 (3.5)	137 (3.8)	137 (2.9)
S–potassium* mmol/l							
Placebo	4.02 (0.36)	3.36 (0.43)	4.01 (0.41)	4.02 (0.28)‡	3.99 (0.28)‡	4.07 (0.52)	3.98 (0.55)
Nutrition	4.07 (0.50)	3.35 (0.41)	3.81 (0.41)	3.63 (0.44)	3.67 (0.38)	3.75 (0.50)	3.85 (0.46)
Input sodium§ mmol/day							
Placebo		898 (241)	233 (141)†	125 (107)	62 (88)	42 (83)	69 (124)
Nutrition		923 (283)	158 (133)	150 (108)	101 (108)	72 (130)	29 (76)
Input potassium* mmol/day							
Placebo		7.7 (19.1)‖	27.8 (28.2)‡	16.3 (23.1)‖	8.1 (17.8)‖	0.9 (4.7)‖	2.6 (10.3)†
Nutrition		27.7 (14.0)	48.6 (25.3)	49.7 (26.8)	41.2 (31.2)	30.5 (28.4)	14.2 (26.8)

*p<0.005; §p<0.05 (repeated measures multivariate test (Pillai's trace))

†p<0.05; ‡p<0.005; ‖p<0.0005 comparison between groups

Table 4: S-sodium and s-potassium and daily supply of colloids.

A study of patients, who had undergone hip arthroplasty, was not able to demonstrate that hyponatremia was related to a positive electrolyte free water balance. The study demonstrated that patients developing postoperative hyponatremia showed a postoperative increase of the osmotic gap in plasma and a greater postoperative osmotic gap in urine. It suggested that the release of osmotically active solutes leads to cellular water shift from intracellular to extracellular spaces, and concluded that these data might support the clinical relevance of the sick cell syndrome in the postoperative context [8].

The "sick cell syndrome" was also demonstrated in critically ill patients. A study found that hyponatremia with an increased osmolar gap was linked to an accumulation of endogenous solutes. The authors did not have any explanation as to the nature of the endogenous solutes accounting for the increased osmolar gap and concluded that this question remains to be determined [9].

In the study presented, an increase in bodyweight, which paralleled ECV, was found. In the placebo group, hyponatremia on POD 2 and POD 3 indicating a redistribution of sodium was also found.

An explanation for the sick cell syndrome, the trial concluded that the increased distribution volumes for ions physiologically and predominantly restricted to the extracellular space indicated a change in the permeability of the cell membrane [10].

In an animal model for acute hyponatremia, the basis was electrolyte loss and a small negative balance for water, the ECVvolume did not contract which suggested that water shifted from the ICV to the ECV. The authors concluded that hyponatremia was associated with cell swelling only if its cause was a positive water balance and/or a loss of Na from the ECV [11]. All patients in the study gained weight because of a massive water input the day of operation. Although they received a large amount of sodium, the patients in the placebo group had hyponatremia on POD 2 and POD 3 indicating cell swelling.

The change in cell membrane permeability alone does not explain these findings. The S-potassium was significantly higher in the placebo group on POD 2 and POD 3 although the patients in this group received significantly less potassium than the nutrition group. The patients in the nutrition group showed lower values of s-potassium from the day of operation through POD 5. The input of potassium was not comparable to that in a normal diet (i.a. 50-100 mmol/day) but was still much higher than the input in the placebo group. Unfortunately, the amount of potassium in the urine was not measured. However, it would seem unreasonable to conclude that the potassium secretion in the nutrition group explained the significant lower values associated with the higher input. In the placebo group, the only source of potassium was intracellular except for the small intravenous input.

The normal regulation of cell volume depends on the Na-K pump, which actively pumps sodium out of the cells. A lack of energy supply to the Na-K pump can result in a dysfunction in pumping sodium out of the cell and the cell will consequently leak potassium to the ECV. A dysfunction of the Na/K pump could result in hyponatremia because of re-distribution to ICV and an increase in the extracellular potassium level. This is in accordance with the findings in the placebo group.

There was none of these colloid changes in the early postoperative nutrition group indicating that early postoperative enteral nutrition ensures the energy supply to the cell in the postoperative period and protects against hyponatraemia in surgical patients.

Conclusion

Early postoperative enteral nutrition protects against hyponatraemia compared to "no food". This could be due to the energy supply to the Na-K pump.

Trial Registration

The protocol for this study was approved by the Copenhagen County Scientific Ethics Committee (H-KA-93174). The study was initiated in 1993, before it was mandatory to register the trial on a public database.

Key messages

Early postoperative enteral nutrition ensures energy for the Na-K pump.

Early postoperative enteral nutrition protects against postoperative hyponatraemia.

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