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Glucose-charged dialysate for children on hemodialysis: acute dialytic changes

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Abstract. Glucose has been omitted from hemodialysates in the recent past. Currently, there is a tendency to include glucose in dialysates at physiological concentrations between 100 and 200 mg/dl (5.56–11.12 mmol/l). In adult patients, this induces, over the dialysis session, a significant uptake of glucose, with some benefits, i.e., avoidance of caloric loss, but also with some metabolic risks, i.e. decreased dialytic potassium removal secondary to an insulin-dependent intracellular potassium shift. We have performed a crossover study in five stable children (mean age 11.7 years) with normal fasting glucose on chronic bicarbonate hemodialysis. The dialysis prescription of 3-h sessions was changed only in terms of the glucose dialysate concentration, being either glucose free or containing 9.17 mmol/l (165 mg/dl) glucose; dialysates were potassium free. Twenty sessions were analyzed for each group by whole dialysate collection (glucose, potassium, phosphate) and serum concentration analysis during and post dialysis (glucose, potassium, phosphate, insulin). Glucose-free dialysis was associated with a patient net glucose loss of 113 ± 12 mmol/session (nearly 20 g). Conversely, with the glucose-charged dialysate a small uptake of glucose was noted [13.8 ± 2.1 mmol/session (nearly 2 g)]. At the end of the session, serum glucose was lower with the glucose-free dialysate (4.64 ± 0.52 mmol/l) than the glucose-charged dialysate (6.11 ± 0.92 mmol/l). Conversely, serum insulin was higher with the glucose-charged dialysate (38 ± 17 mU/l) than the glucose-free dialysate (19 ± 9 mU/l). There were no significant differences either for dialytic removal of potassium (70 vs. 73 mmol/session) or phosphate (20 vs. 22 mmol/session), with and without glucose dialysates. Our study, contrary to previously published data in adults, demonstrated that in children the use of a physiological concentration of glucose in the dialysate (165 mg/dl) avoids dialytic glucose loss without a significant decrease in dialytic potassium removal.

Key words: Glucose-charged dialysate – Potassium

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Introduction

In the early years of hemodialysis, high dialysate glucose levels were prescribed to induce osmotic ultrafiltration. These were not infrequently associated with severe hyperglycemia and a hyperosmolar syndrome, comprising agitation, thirst, and headaches [1]. In recent years, however, the vast majority of children have been treated with glucose-free dialysates. Currently there is a tendency to use dialysates containing physiological concentrations of glucose. This new trend [2] has some benefits, i.e., avoidance of hypoglycemia-related symptoms [3] and even provision of caloric supplementation, but may also be associated with some risks, either acute metabolic risks, i.e., decreased dialytic potassium removal secondary to an insulin-dependent intracellular (IC) potassium shift [4], or chronic metabolic risks, i.e., dyslipidemia and glucose intolerance [5]. Because of the important risks associated with hyperkalemia, we have measured the acute change in potassium with and without glucose-charged dialysate in children.

Patients and methods

We performed a crossover study in five children (mean age 11.7 years) on chronic bicarbonate hemodialysis, three sessions per week. These patients were stable with normal fasting glycose; no patient received antihypertensive treatment with β-adrenergic blockers. Midweek sessions were analyzed for 8 consecutive weeks. Each child was alternately dialyzed with or without glucose in the dialysate. The dialysis sessions were performed in the morning, after a standard breakfast, eaten at the unit at least 1 h before dialysis. The patients fasted thereafter until the end of the dialysis session. The dialysis prescription was changed only in terms of the glucose dialysate concentration, being either glucose-free or containing 9.17 mmol/l (165 mg/dl) glucose. The dialysis session duration was 3 h: dialysates contained bicarbonate (35 mmol/l) and were potassium free (0 mmol/l). The prescribed dialysis dose was kept constant over the study period, adjusted to an equilibrated KT/V of 1.3 [6].

Collection of the whole dialysate allowed calculation of the mass balance, especially for glucose, potassium, phosphate, and proteins, for the entire session, for the period 0–45 min, and for the period 45–180 min. Blood samples were taken from the arterial blood access line

Table 1. Time-related values for serum glucose, insulin, potassium, phosphate, and pH with (n = 20 sessions) and without (n = 20 sessions) glucose-charged dialysate[a]

Dialysis time (min)		Session			Rebound	
		t_0	t_{45}	t_{180}	t_{180+30}	t_{180+60}
Glucose	Without	5.99±1.10	6.22±1.27	4.64±0.52	4.72±0.61	4.83±0.62
(mmol/l)	With	6.15±1.03	7.68±0.77*	6.11±0.92*	5.06±0.89*	4.93±0.79
Insulin	Without	22±8	ND	19±9	ND	23±7
(mU/l)	With	24±13		38±17*		21±12
Potassium	Without	4.26±0.36	3.39±0.51	3.01±0.36	3.28±0.42	3.43±0.45
(mmol/l)	With	4.40±0.70	3.38±0.28	2.98±0.28	3.35±0.24	3.56±0.29
Phosphate	Without	1.94±0.33	1.29±0.27	1.08±0.14	1.34±0.16	1.50±0.12
(mmol/l)	With	1.84±0.39	1.21±0.25	1.04±0.14	1.31±0.12	1.44±0.11
pH	Without	7.38±0.05	7.42±0.04	7.47±0.05	7.47±0.04	7.48±0.03
	With	7.40±0.03	7.42±0.01	7.48±0.04	7.47±0.04	7.47±0.03

*P <0.01 vs. without glucose-charged dialysate at the same dialytic time
[a] Results are given as mean ± SD for n sessions

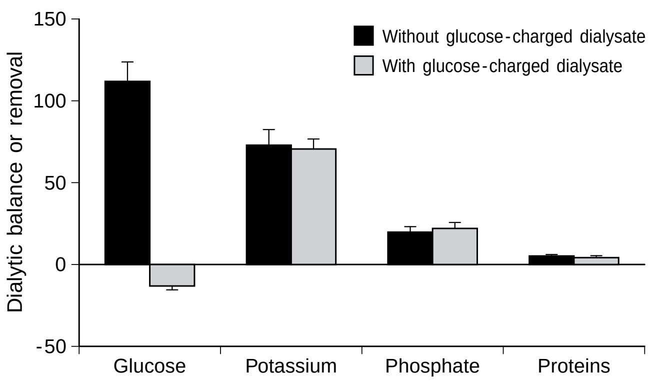

Fig. 1. Dialytic glucose balance (mmol/session), potassium and phosphate (mmol/session) and protein removal (g/session) over the whole session with (n = 20) or without (n = 20) glucose-charged dialysate. Results given as mean±SD

prior to dialysis (t_0), 45 min after initiation of the session (t_{45}), at the end of the 3-h session (t_{180}), i.e., after 3 min of low blood flow, 50 ml/min [6], and during the so-called post-dialytic rebound period, at 30 min and 60 min postdialysis (t_{180+30}, t_{180+60}). During this post-dialytic period, dialysate flow was stopped and extracorporeal circulation was maintained at a low blood flow, i.e., 50 ml/min.

Blood samples were used for determination of urea, creatinine, glucose, potassium, pH, bicarbonate, phosphate, and insulin by immunoassay (Abbott, IMX). From analysis of the total dialysate removed and of the blood level variations over the period of dialysis, the contribution of the IC compartment to dialytic potassium removal was calculated, asumming 60% of the total body water to be IC, total body water to be 60% of the body weight, and taking pre- and post-dialysis serum concentrations as representative of extracellular (EC) concentrations at those times [7]:

Total mass removed = (IC+EC) mass removed
EC mass removed = (body weight × 60%) × 60% × (K_0–K_{180}).

Within group, i.e., with (n = 20) or without (n = 20) glucose-charged dialysate, time-related values were evaluated using Student's t-test for paired data. Analysis of variance was used to compare the effect of including or excluding glucose in the dialysate on the blood profiles and on the quantities removed in the dialysate. Changes were considered statistically significant for P values ≤0.01. Values are given as mean plus or minus standard deviation for n observations. Pearson's correlation coefficients were calculated to determine the significance of linear relationships among variables. Patients were informed of the study objectives and verbal acceptance was obtained.

Table 2. Total measured and calculated dialytic potassium and glucose removal with (n = 20 sessions) and without (n = 20 sessions) glucose-charged dialysates

		Total	Intracellular	Extracellular
Dialytic potassium removal (mmol/session)	Without	73±10	34.7±7.4	38.3±6.8
	With	71±6	31.8±4.3	39.1±5.2
Dialytic glucose balance (mmol/session)	Without	113±12	glucose patient loss	
	With	13.8±2.1	glucose patient gain	

Results

Glucose-charged dialysate induced a significant increase in serum glucose 45 min after initiation of dialysis (Table 1), an increase maintained during the entire dialysis period and the initial post-dialysis period (t_{180+30}). No intra- or post-dialytic episodes of hypoglycemia were noted. The serum insulin levels differed only at the end of the dialysis session (Table 1), with a significant increase with glucose-charged dialysate. Despite the use of potassium-free dialysate, no symptomatic episodes of hypokalemia were noted in any of the 40 analyzed sessions. The lowest end-dialysis serum potassium recorded was 2.4 mmol/l, which correlated with the lowest pre-dialysis value of 4 mmol/l. No differences were observed in serum phosphate or pH over time between the two groups. With glucose-free dialysate (Fig. 1), children had a negative glucose balance (patient loss) of 113±12 mmol/session. In contrast with the glucose-charged dialysate, children had a positive glucose balance (patient gain) of 13.8±2.1 mmol/session. The dialysate glucose concentration influenced neither the potassium nor the phosphate mass removed (Fig. 1). No differences were noted either in mass removed for the entire dialysis session or for the periods from 0 min to 45 min or from 45 min to

180 min. The calculated compartmental potassium mass removed did not differ between the glucose-charged dialysate (IC 31.8 ± 4.3 mmol and EC 39.1 ± 5.2 mmol) and the glucose-free dialysate (IC 34.7 ± 7.4 mmol and EC 38.3 ± 6.8 mmol) (Table 2). Dialytic potassium removal was positively correlated with the serum potassium at the start of the session both with (r = 0.879, P < 0.001, $y = 0.054x \pm 0.74$) and without (r = 0.797, P < 0.001, $y = 0.037x \pm 0.4226$) glucose-charged dialysate. The amount of protein removed by dialysis was not significantly different with (3.7 ± 0.6 g/session) and without (4.3 ± 0.9 g/session) glucose-charged dialysate.

Discussion

For many years, the hemodialysate used for children remained constant, until a decade ago when bicarbonate was substituted for acetate [2, 8]. More recently, problems of maintaining adequate nutrition have led many dialysis centers to include glucose in the dialysate, but only a few reports have mentioned the potential risks associated with the use of glucose, especially the reduction of dialytic potassium removal secondary to glucose-related insulin secretion [4].

In our study we clearly demonstrated that glucose-free dialysate led to a significant loss of glucose of 113 mmol ± 12 mmol/session per patient, i.e., 20 g of glucose and 80 kcal/session per patient. A negative glucose balance during dialysis should be avoided, more to maintain the physiological state [9] than to guard against malnutrition. Addition of glucose to the dialysis fluid may help the energy balance [9], but it does not appear, in our study, to significantly reduce dialytic protein removal [10]. Inclusion of glucose in the dialysate could be recommended for children, but the concentration should be adjusted bearing in mind the benefits and the risks [1, 2, 4, 5]. Using a glucose dialysate concentration of 9.17 mmol/l (165 mg/dl), the glucose increase was low, 13.8 ± 2.1 mmol/session. Serum glucose and insulin profiles differed in the groups with and without glucose-charged dialysate (Table 1). Nevertheless, there was no significant potassium insulin-dependent shift from the EC to the IC space with glucose dialysate, as measured by similar dialytic potassium removal with and without glucose-charged dialysate (Fig. 1). The quantitative importance of the glucose charge or the potential insulin resistance described in uremia [11] could explain these results. In adults [4], using an 11.1 mmol/l (200 mg/dl) glucose dialysate concentration over a 4-h bicarbonate hemodialysis session, dialytic potassium removal was significantly lower with glucose-charged dialysate (54 ± 1224 mmol/session) than glucose-free dialysate (72 ± 26 mmol/session). Therefore, in our opinion the recommended glucose dialysate concentration should be in the physiological range and should be adapted to the dialysis dose.

Dialytic phosphate removal was not influenced by the glucose dialysate concentration in our study, but higher glucose concentrations inducing higher serum insulin levels should be able to trap phosphate in the cells, thus decreasing dialytic phosphate removal [7]. In order to optimally analyze dialytic potassium removal, we used a potassium-free dialysate, despite the known potential risks [8, 12]. Although serum potassium was not significantly increased at the beginning of the dialysis session (Table 1), clinical symptoms of hypokalemia or profound hypokalemia were not observed at the end of the 40 dialysis sessions. These results suggest that the usual dialysate prescribed for children containing 2 mmol/l potassium [2, 6] should be reduced for many patients, allowing an improvement of dialytic potassium removal.

In conclusion, our study supports the use of a glucose-charged dialysate in children, but the choice of the glucose concentration is important. It should, in our opinion, be in the physiological range, high enough to avoid dialytic glucose loss but also low enough to avoid patient glucose charge. In children with a dialysis dose of 1.3. KT/V, a glucose level of 9.17 mmol/l (165 mg/dl) in the dialysate was able to achieve this goal without acute metabolic risks, particularly without decreased dialytic potassium removal.

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References

1. Mendelssohn S, Schwartz CD, Yudis M, et al (1967) High glucose concentration dialysate in chronic hemodialysis. Trans ASAIO 13:249–253
2. Van Stone JC (1994) Individualization of the dialysate prescription in chronic hemodialysis. Dial Transplant 23(11):624–663
3. Pietsman SJ, Agranal BN (1977) Spontaneous hypoglycemia in end stage renal failure. Nephron 19:131–139
4. Ward RA, Wathen RL, Williams TE, Harding GB (1987) Hemodialysate composition and intradialytic metabolic, acid base and potassium changes. Kidney Int 32:129–135
5. Swamy AP, Cestero RVM, Campbell RG, Freeman RB (1976) Long term effect of dialysate glucose on the lipid levels of maintenance hemodialysis patients. Trans ASAIO 21:54–59
6. Buur T, Bradburg MG, Smye SW, Brocklebank JT (1994) Reliability of hemodialysis urea kinetic modeling in children. Pediatr Nephrol 8:574–578
7. Fischbach M, Hamel G, Simeoni U, Geisert J (1992) Phosphate dialysis removal (PDR): enhancement of phosphate cellular clearance by biofiltration (with acetate free buffer dialysate). Nephron 62:155–160
8. Avis L, Carney S, Gillies A, Jones B, Nanra R, Shaw R, Trevillion P, Webb C (1996) An optimal potassium, calcium and magnesium dialysate for general use? Dial Transplant 25:354–379
9. Wathen RA, Keshavish P, Hommeyer P (1978) The metabolic effects of hemodialysis with and without glucose in the dialysate. Am J Clin Nutr 31:1870–1875
10. Gutierrez A, Bergstrom J, Alvestrand A (1994) Hemodialysis associated protein catabolism with and without glucose in the dialysate fluid. Kidney Int 46:814–122
11. Mak RHK, De Fronzo RA (1992). Glucose and insulin metabolism in uremia. Nephron 61:377–381
12. Hau S, Mc Elroy PA, Nootens J, Beach M (1989) Safety and efficiency of low potassium dialysate. Am J Kidney Dis 13:137–143