Intradialytic glucose infusion increases polysulphone membrane permeability and post-dilutional haemodiafiltration performances

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Abstract

Introduction. During real-time monitoring of the ultrafiltration coefficient (Kuf) in haemodiafiltration (HDF), it was noticed that the ultrafiltration performance of polysulphone membrane dialysers increased when hypertonic glucose (D50%) was administered through the venous blood return.

Methods. This observation was explored in six non-diabetic chronic dialysis patients during 48 HDF sessions using 1.8 m² polysulphone membrane dialysers. In all six patients, 24 sessions were performed with glucose supplementation (as a continuous D50% (500 g/l) infusion at 40 ml/h) and 24 sessions without supplementation.

Results. Glucose supplementation led to a marked increase in Kuf from 22.8 ± 2.2 (without D50%, n = 24) to 32.1 ± 3.9 ml/h/mmHg (with D50%, n = 24) (P < 0.0001). An increase in percentage reduction ratios for urea and creatinine were also consistently observed during the sessions with glucose administration (from respective mean values of 75 ± 5 and 68 ± 4% to 79 ± 4 and 74 ± 10%). Mean double-pool Kt/V, calculated from serum urea concentrations, rose from 1.65 ± 0.24 (n = 24) to 1.86 ± 0.24 (n = 24) (P < 0.005). Similar results were observed in a subgroup of 18 HDF sessions (nine with glucose and nine without) monitored with an on-line urea sensor of spent dialysate. No detrimental effects were induced at any time.

Conclusions. We conclude that intravenous glucose administration during high-flux HDF using polysulphone membranes increases significantly both ultrafiltration capacity and dialysis dose delivery.

Keywords: haemodiafiltration performances; glucose dialysis dose; membrane permeability

Introduction

In the last decade, the introduction of highly permeable membranes has allowed a widening of the spectrum of solute elimination [1–3]. Haemodiafilters equipped with highly permeable membrane are required for convective-based renal replacement modalities such as high-flux haemofiltration (HF) and haemodiafiltration (HDF), to enhance solute clearance and molecular-weight spectrum of solute removed.

In vivo haemodiafilter performances are commonly assessed on two parameters: solute clearances and ultrafiltration coefficient. On one hand, clearances (Kd) permit evaluation of the solute clearing capacity of a haemodiafilter with selected solute marker. Kd gives an indirect insight to the solute membrane permeability. Normalized body clearance known as the ‘dialysis dose’ or Kt/V is one of the most convenient dialysis indices to evaluate objectively the dialysis efficacy. On the other hand, ultrafiltration coefficient (Kuf) permits evaluation of the water plasma filtering capacity of a haemodiafilter. Kuf is a surrogate of haemodiafilter hydraulic permeability that represents the ultrafiltration rate (UFR in ml/h) and transmembrane pressure (TMP in mmHg) ratio. Hydrostatic and osmotic forces both interact to regulate ultrafiltration rate water and transfer across the artificial membrane [4]. In vivo UFR can be altered consequently due to the protein concentration–polarization effect occurring at the surface of the membrane, particularly with high ultrafiltration rate (100–150 ml/min) [5–7]. Recently, on-line continuous Kuf monitoring has proved useful for real-time assessment of hydraulic permeability changes of the module device during HDF [8]. While evaluating this system in vivo, it was noticed that Kuf changed suddenly during the HDF session with high-flux polysulphone when glucose was infused intravenously to the patient to correct or to prevent hypotensive episodes. This empirical observation prompted us to assess the effects of i.v. glucose administration on high-flux HDF performances and to evaluate its impact on ‘dialysis dose’ delivery.
Subjects and methods

Study design

The study consisted of two main parts. In the first part, 24 HDF sessions were performed with glucose supplementation in all six patients; in the second part, 24 HDF sessions were performed in the same patients without glucose administration. Each patient underwent two consecutive sessions without glucose followed by two consecutive sessions with glucose. Glucose, as D50%, was infused in the venous bubble trap continuously with an electric syringe throughout the session at a rate of 30 or 40 ml/h (total of 60 g) for dialysis duration lasting 4 and 3 h respectively. Note that glucose infusion started immediately after connecting the arterial line 3–5 min before the HDF programme was set on the dialysis monitor.

Patients

Six stable haemodialysis dialysis patients were included in this study after informed consent was obtained. The group consisted in four men and two women of a mean age of 41.3 ± 22.4 years. Diabetes mellitus was an exclusion criterion.

Dialysis modality

HDF was performed with 2008E and 4008D volumetric control machines (Fresenius, Bad Homburg, Germany) allowing on-line production of replacement fluid from fresh ultrapure dialysate, as described previously [9]. Dialysate and replacement solutions contained glucose at a concentration of 5.55 mmol/1 (1.0 g/l). The blood flow rate was maintained at 350 ml/min and the mean dialysate flow rate was 630 ± 19 ml/min (both were maintained at the same level for a given patient). Session duration was kept constant in each case, varying between 3.0 and 4.0 h. No change in heparin regimen was allowed during the study for the same urea sensor-monitoring device (UM1000, Baxter Healthcare Corporation, McGraw Park, IL, USA) connected on-line to the HDF adequacy

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In each part of the study, nine HDF sessions were monitored with a urea sensor (UM1000, Baxter Healthcare Corporation, McGraw Park, IL, USA) connected on-line to the effluent stream (spent dialysate plus ultrafiltrate).

Recorded parameters and calculations

Kuf was monitored at bedside during the session (DIB08E, Fresenius), as recently described [8]. This data acquisition system allows continuous determination of Kuf throughout the entire session. Kuf is calculated from the ratio of total UFR and TMP measured by the dialysis monitor. In this case, TEMP represents the numerical sum of the pressure values recorded in blood and dialysate compartments.

Blood was sampled pre- and immediately post-session for urea, creatinine, glucose, sodium, osmolality, and total protein concentrations, as well as haematorcit determination. In addition, serum glucose concentration was measured simultaneously at the dialyser inlet and outlet, and in the spent dialysate, after 1 h of dialysis. Haematorcit level was also measured from inflow and outflow blood sampling. Pre- and post-session dialysate weight, total and net ultrafiltration, and pre- and post-session mean arterial pressure (MAP) were recorded. Percentage reduction ratio (%) for urea and creatinine and double-pool equivalent Kn/V urea were assessed at each session. Body single-pool Kn/V was calculated according to the equation from Garred et al. [10]:

\[
\text{Kn/V} = (\text{LnR} + 3\text{ABW/BD})/(1 - 0.01786t_{\text{HD}})
\]

where R, ABW, BW, and t HD represent respectively ratios between pre- and post-dialysis serum urea concentrations, pre- to post-dialysis change in body weight (kg), dry body weight (kg), and session time (h).

Kn/V was then corrected for rebound according to the equation from Daugirdas et al. [11]:

\[
\text{Kn/V}_{\text{cr}} = \text{Kn/V} - 36*\text{Kt}/V_{\text{sp}}/\text{BW} + 0.03
\]

The on-line urea sensor (UM1000) on the spent dialysate line provides the solute removal index (SRI) and body Kn/V; both parameters were recorded for the sessions monitored with this device.

Validation of dialysis quantification indexes

On-line direct dialysis quantification based on a urea sensor-monitoring device (UM1000, Baxter Healthcare Corporation, McGraw Park, IL, USA) connected on-line to the effluent stream (spent dialysate plus ultrafiltrate).

<table>
<thead>
<tr>
<th>Patient no. and infusion status</th>
<th>Net UF (1/session)</th>
<th>Total UF (1/session)</th>
<th>Urea pre (mmol/l)</th>
<th>Creat pre (mmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Without</td>
<td>3.1 ± 0.8</td>
<td>27.1 ± 0.8</td>
<td>26.5 ± 4.5</td>
<td>895 ± 38</td>
</tr>
<tr>
<td>With</td>
<td>3.5 ± 0.3</td>
<td>27.5 ± 0.3</td>
<td>29.3 ± 6.0</td>
<td>850 ± 91</td>
</tr>
<tr>
<td>Patient 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Without</td>
<td>3.5 ± 0.3</td>
<td>27.5 ± 0.3</td>
<td>31.0 ± 5.7</td>
<td>1083 ± 80</td>
</tr>
<tr>
<td>With</td>
<td>3.1 ± 0.8</td>
<td>27.1 ± 0.8</td>
<td>33.7 ± 7.4</td>
<td>1195 ± 129</td>
</tr>
<tr>
<td>Patient 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Without</td>
<td>2.4 ± 0.5</td>
<td>23.4 ± 0.5</td>
<td>27.2 ± 7.1</td>
<td>1025 ± 48</td>
</tr>
<tr>
<td>With</td>
<td>2.7 ± 0.8</td>
<td>23.7 ± 0.8</td>
<td>27.8 ± 3.1</td>
<td>1151 ± 154</td>
</tr>
<tr>
<td>Patient 4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Without</td>
<td>2.0 ± 0.0</td>
<td>23.0 ± 0.0</td>
<td>15.7 ± 0.7</td>
<td>570 ± 0</td>
</tr>
<tr>
<td>With</td>
<td>1.7 ± 0.8</td>
<td>24.7 ± 0.8</td>
<td>18.4 ± 3.4</td>
<td>629 ± 76</td>
</tr>
<tr>
<td>Patient 5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Without</td>
<td>2.7 ± 0.1</td>
<td>26.7 ± 0.1</td>
<td>13.3 ± 1.3</td>
<td>863 ± 43</td>
</tr>
<tr>
<td>With</td>
<td>2.9 ± 0.3</td>
<td>26.9 ± 0.3</td>
<td>21.0 ± 4.6</td>
<td>892 ± 35</td>
</tr>
<tr>
<td>Patient 6</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Without</td>
<td>2.8 ± 0.3</td>
<td>26.8 ± 0.3</td>
<td>26.2 ± 0.3</td>
<td>839 ± 31</td>
</tr>
<tr>
<td>With</td>
<td>2.2 ± 0.3</td>
<td>26.2 ± 0.3</td>
<td>29.8 ± 10.2</td>
<td>915 ± 108</td>
</tr>
</tbody>
</table>

Results

Results are presented as mean ± SD. Where appropriate, paired Student’s t-test, and Spearman correlation coefficient were applied for statistical analysis.
Intradialytic glucose infusion and post-dilutional HDF performances 513

Healthcare) was employed to validate the calculation of the blood-side dialysis indexes. Body \( \text{Kt/V} \) calculated from the Daugirdas double-pool equivalent formula using pre- and post-dialysis urea concentrations has been plotted against the body \( \text{Kt/V} \) measured by the UM 1000. As shown in Figure 1, a strong positive linear relationship \( (r=0.94, P=0.01) \) exists between body \( \text{Kt/V} \) obtained from blood and dialysate-side methods. The same relationship was found between urea reduction rate and SRI \( (r=0.92, P<0.05) \) in the subgroup of 18 HDF sessions concerned (including nine without and nine with glucose supplementation).

Consequences of glucose administration

Instantaneous glucose mass transfer \( (J_{\text{Glu}}, \text{mmol/min}) \) using inlet \( (\text{Glu}_{\text{inlet}}) \) and outlet \( (\text{Glu}_{\text{outlet}}) \) dialysate glucose concentrations and dialysate flow \( (QD) \) were measured 60 min after the start of HDF session as \( J_{\text{Glu}} = QD \times (\text{Glu}_{\text{outlet}} - \text{Glu}_{\text{inlet}}) \). Instantaneous glucose fluxes were used to calculate the net glucose mass balance (mmol/session and g/session) achieved through the overall HDF session \( (\text{Glu mass}=J_{\text{Glu}} \times t_{\text{HDF}}) \) assuming that operational conditions remained constant.

Membrane permeability changes

Coefficient of ultrafiltration \( (K_{\text{uf}}) \) of the HF80s haemodiafilter was significantly increased with glucose infusion. The mean value for the \( K_{\text{uf}} \) increased from \( 22.8 \pm 2.2 \text{ ml/h/mmHg} \) (without glucose, \( n=24 \) sessions) to \( 32.1 \pm 3.9 \text{ ml/h/mmHg} \) (with glucose, \( n=24 \) sessions) \( (P<0.0001) \). A typical example of such \( K_{\text{uf}} \) changes observed in patient no. 1 during HDF session with and without glucose administration is reported in Figure 2. As shown, the plot of the continuous monitoring of \( K_{\text{uf}} \) evidences a significant and permanent difference of the two exponential curves, the upper curve being obtained with glucose administration and the lower curve obtained without glucose infusion.

Dialysis dose delivered with HDF sessions with and without glucose administration is presented in Figure 2. As shown, significant increases in urea and creatinine percentage reduction ratios and body \( \text{Kt/V} \) were consistently observed with glucose supplementation. Interestingly, UM1000 monitoring confirmed the significant difference in body \( \text{Kt/V} \) delivery and SRI during HDF sessions with glucose as compared to those without. Mean \( \text{Kt/V} \) increased from \( 1.3 \pm 0.2 \) (without glucose) to \( 1.7 \pm 0.2 \) (with glucose) \( (P<0.005) \) and mean SRI, from \( 70 \pm 6\% \) (without glucose) to \( 78 \pm 4\% \) \( (P<0.005) \).

Serum glucose concentrations tended to increase post-HDF as a consequence of the glucose supplementation. However, no severe hyperglycaemia or tolerance problems were observed at any time. Serum glucose pre- and post-HDF rose from \( 4.9 \pm 0.8 \) to \( 6.6 \pm 1.5 \text{ mmol/l} \) for the 24 sessions without glucose, and from \( 5.1 \pm 1.3 \) to \( 8.6 \pm 2.3 \text{ mmol/l} \) for the 24 with glucose infusion \( (P<0.05) \). Glucose concentrations measured simultaneously at dialyser blood inlet and outlet, and in spent dialysate were respectively as follows: \( 5.0 \pm 0.7, 5.7 \pm 1.0 \) and \( 5.5 \pm 0.3 \text{ mmol/l} \) in the absence of glucose infusion and \( 9.1 \pm 0.6, 7.1 \pm 0.6 \) and \( 8.1 \pm 0.5 \text{ mmol/l} \) in the presence of glucose supplementation. Results comparing HDF sessions without and with glucose infusion are presented in Table 2. As shown, a virtually nil net glucose mass balance \( (5.9 \pm 35 \text{ mmol/session} \) or \( 1.1 \pm 6.3 \text{ g/session}) \) was noted in HDF sessions without glucose infusion while HDF sessions with glucose infusion induced a \( 346 \pm 88 \text{ mmol/session} \) \( (62.4 \pm 16 \text{ g/session}) \) negative glucose mass transfer.

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**Fig. 1.** Relationship between \( \text{Kt/V} \) calculated from blood-side measures using the equation from Garred and \( \text{Kt/V} \) provided by UM1000 for a subgroup of 18 HDF sessions; the correlation coefficient was 0.94.
Fig. 2. Real-time ultrafiltration coefficient profile as monitored by the on-site data acquisition system; these figures concern high-flux polysulphone membranes with or without hypertonic glucose administration.

Fig. 3. Mean urea and creatinine percent reduction ratios (%) and single-pool Kt/Vurea obtained during the 24 HDF sessions without hypertonic glucose (hatched bars) and the 24 HDF sessions with glucose administration (dark bars).

Table 2. Glucose mass transfer during HDF session estimated from instantaneous glucose dialysance measured 1 h after starting HDF session

<table>
<thead>
<tr>
<th></th>
<th>Artery (mmol/l)</th>
<th>Vein (mmol/l)</th>
<th>Dialysate (mmol/l)</th>
<th>Gi mass (mmol/session)</th>
<th>Gi mass (g/session)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HDF without D50%</td>
<td>5.0 ± 0.7</td>
<td>5.7 ± 1.0</td>
<td>5.5 ± 0.3</td>
<td>5.9 ± 35.0</td>
<td>1.1 ± 6.3</td>
</tr>
<tr>
<td>HDF with D50%</td>
<td>9.1 ± 0.6</td>
<td>7.1 ± 0.6</td>
<td>7.5 ± 2.4</td>
<td>346.6 ± 88.9</td>
<td>62.4 ± 16.0</td>
</tr>
</tbody>
</table>

Dialysate/infusate glucose concentration 5.55 mmol/l.
Haematocrit level was measured at 34% at dialyser blood inlet, and at 37% at dialyser outlet in the absence of glucose infusion, and respectively at 34 and 36% with glucose supplementation.

Total protein concentration was 66 g/l at dialyser inlet and 70 g/l at dialyser outlet in the absence of glucose infusion as compared to 67 g/l and 71 g/l respectively with glucose supplementation.

Furthermore, there was no significant change in serum sodium concentrations and blood osmolality (pre- vs post-HDF and sessions without vs with glucose). Pre-/post-session serum sodium was 139 ± 2 /413 ± 1 mmol/l in HDF without glucose, and 141 ± 2 /137 ± 2 mmol/l in HDF with glucose (P = NS (non significant) without vs with glucose). Pre-/post-bloom osmolality was 312 ±9 /6301 ± 3.5 without glucose, and 314 ± 10.1 /301 ± 3.9 mosm/kgH2O (P = NS with glucose vs with glucose). Arterial blood pressure tended to be better maintained in HDF sessions when glucose was administered, although it did not reach statistical significance. Mean arterial pressure pre- and post-HDF declined from 101 ± 23 to 78 ± 16 mmHg for the 24 sessions without glucose, and from 97 ± 20 to 83 ± 17 mmHg for the 24 sessions with glucose.

Discussion

During haemodialfiltration, water flux results from the driving force generated mainly by the hydrostatic pressure and the retentive forces resulting from osmotic and oncotic pressures, hydraulic membrane permeability, and protein membrane interaction [4,12]. Ultrafiltration coefficient (Kuf) is a convenient marker that depicts in a simple way the complex scenario of water flux occurring during in-vivo HDF sessions. Kuf is used in this study as a surrogate of filter hydraulic permeability permitting assessment of the membrane permeability changes. According to HDF conditions, Kuf results from a complex interaction between two types of phenomenon: on the one hand, Kuf is proportional to the driving force, that is the transmembrane pressure resulting from hydrostatic pressure generated through the filter, while on the other, Kuf is inversely proportional to the osmotic pressure exerted by the proteins, the hindrance of the membrane and the resistance arising from protein concentration–polarization effect (known as protein cake or second layer). It is well known that enhancing transmembrane pressure exerted through the membrane increases ultrafiltration performance up to a plateau value known as flow-controlled regime. It has also been shown that, at this stage, enhancing blood flow is beneficial to ultrafiltration performance. Higher blood flow increases membrane shear stress, displaces the gel-polarized layer, and partly reverts the system to a pressure-controlled regime. However, it was not suspected that intravenous glucose administration might have an impact on hydraulic permeability of high-flux polysulphone membrane in HDF. Interestingly, the Kuf increase incidentally noticed during glucose administration, either as bolus or as continuous infusion, is confirmed by continuous Kuf monitoring allowed by our on-site system. It is also of interest to note that the Kuf increase is an early phenomenon occurring when glucose-enriched blood comes back to the patient and/or to the contact of the haemodiafilter membrane.

Enhancement in solute clearances by 10–15% with glucose administration observed in our study appears to be most probably a consequence of an increase in membrane water and/or solute fluxes. It translated into improved performance of HDF as evidenced by a significant increase of percentage reduction ratios, Kt/V, and SRI. Although not assessed in the present study, an evaluation of middle molecule elimination during HDF (with or without glucose supplementation) appears suitable to appreciate more extensively the overall gain in performance brought about by the glucose infusion. It is admitted that the formation of a protein layer onto the membrane under high convective flux affects its hydraulic permeability and decreases Kuf and solute removal by reducing the sieving coefficient of the membrane [5–7]. Blood viscosity, which is largely dependent on plasma protein concentration and haematocrit, has an impact on the magnitude of this phenomenon [13–15]. Accordingly, one may wonder if the protein coating that develops at the surface of a polysulphone membrane during high-flux HDF could be altered by glucose administration, thereby reducing its negative impact on water and/or solute permeability.

This preliminary study does not provide a clear explanation for the enhanced ultrafiltration capacity of a polysulphone filter concomitant on glucose infusion in the venous blood return. A mechanical phenomenon is not likely since the continuous infusion should not affect pressure equilibrium in the dialyser. Anyway, the dialysis machine would keep the ultrafiltration at the desired rate by self-adjusting the transmembrane pressure and, indirectly, pressure in the dialysate compartment. It is recognized that an increase in dialysate sodium may result in a higher serum osmolality, permitting a better refilling of the intravascular compartment from interstitial and intracellular spaces [16,17]; this becomes potentially useful for haemodynamically unstable patients during haemodialysis [18]. On the other hand, glucose infused directly into the patient’s blood induces a brisk increase in serum osmolality, accelerating refilling of the intravascular compartment, and consequently contributing to an increase in hydrostatic gradient across the membrane. Indeed, the observed tendency toward higher glycaemia with glucose supplementation would support this hypothesis. On-line volaemia monitoring was not performed during the course of this study, suppressing, by the way, arguments that would favour this hypothesis. However, glucose osmotic effect in non-diabetic patients (who easily release insulin and rapidly transfer glucose into cells) is not as sustained as for other osmotic agents, nor as in diabetic subjects. In the absence of prolonged hyperglycaemia, this explanation...
appears difficult to admit. Finally, the observed phenomenon could instead be attributed to a direct effect of glucose, and several hypotheses may be formulated. First, glucose may act as an osmotic agent that, freely passing the haemodiafilter membrane, induces a solvent drag effect, facilitating water and small solute removal. Negative glucose mass balance observed during an HDF session with glucose infusion may substantiate this hypothesis. Second, glucose may have a direct effect at the membrane level. Indeed, glucose may be adsorbed onto the membrane, reducing the protein-layer formation. Alternatively, glucose may interact with the chemical and/or electrical structure of the polysulphone membrane itself, altering its permeability performance. Nevertheless, the fact that the Kuf increase is a very early phenomenon favours a role of glucose acting as a solvent drag agent more than interfering at the membrane level with the protein-layer formation. Further, in-vitro and in-vivo studies exploring the role of glucose on haemodiafilter performance changes are needed for a better definition and understanding of such phenomena.

From a haemodynamic perspective, intradialytic administration of an osmotic agent such as hypertonic glucose can improve tolerance to haemodialysis and is routine practice in many units. Even if our objective was not to evaluate this particular aspect, a slight tendency toward better preserved post-session MAP was noted in patients receiving glucose supplementation, although it did not reach significance.

We conclude that glucose infusion in the venous blood return during haemodiafiltration using polysulphone membranes increases ultrafiltration performance by as much as 40% with a concomitant increment in dialysis dose by 10–15%. This preliminary observation favours the osmotic role of glucose and its solvent drag action. However, a direct role of glucose at the membrane surface altering the protein layer formation and the membrane permeability may not be completely ruled out. Further studies are required to evaluate the impact of glucose on different types of membrane and to elucidate its beneficial role on haemodiafiltration performance.

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References


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