In conclusion we suggest that diagnosis and treatment of 
G. lamblia infection, which can exist together with H. pylori 
infection, is very important to control the dyspeptic compl-
aints in haemodialysis patients.

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Arachidonic acid metabolites as biocompatibility parameters for cellulosic membranes

Sir,

Although the complement pathway of activation has been 
the aspect of biocompatibility that has been most thoroughly 
studied, it is clear that other closely related pathways are 
activated during blood–membrane interaction. The general 
objective of the present study was to investigate the bio-com-
patibility of cuprophane membrane (cellulose based) by 
monitoring the levels of arachidonic acid metabolites. Fifteen 
patients with stable end-stage renal failure, receiving thrice 
weekly haemodialysis, were included in the protocol (M/F 
11/4, mean age 40.2±1.3 years, mean haemodialysis duration 
16.2±0.8 months). All the patients were dialysed with single-
use cellulosic cuprophane haemodialysers (Kawasaki, 
Renak 10 H) with prescribed treatment time 240 min (Qb 
230 ml/min, Qb 500 ml/min). Haemodialysis vascular access 
was a native arteriovenous fistula and only bicarbonate-
containing dialysate was used. The anticoagulation regimen 
was individualized with an initial priming dose between 1000 
and 1500 IU of standard heparin, which was followed by a 
continuous infusion of 1000–1500 IU/h. Heparin adminis-
tration was stopped 30 min prior to the end of treatment. In all 
the patients at the onset of treatment, during the 4h haemodialysis 
and at the end of haemodialysis session the following para-
eters were determined: tromboxane B2 (Tx B2), white 
blood cell (WBC) and platelet (PLT) counts, C5C, C4, 
prostaglandin E2 (PGE2), 6-keto prostaglandin F1a, (6-keto 
PGF1a), leucotriene B4 (LTB4), β-tromboglobulin (β-TG). 
Blood samples for TxB2, WBC, PLT, C5C and C4 were 
obtained from all patients at basal, 15 min, 90 min and at 
the end of haemodialysis, whereas PGE2, 6-keto PGE1a, LTB4, 
β-TG at the onset and at the end of haemodialysis session. 
The data were presented as mean±SD and compared for 
statistical significance. Statistical analyses were performed 
using two-tailed Student t-test. A P value of <0.05 was 
considered to be statistically significant.

Table 1 summarizes the mean values of the parameters 
measured. The plasma TxB2 concentration tended to 
decrease at 15 min (P=NS*), returned to predialytic values at 
90 min (P=NS*) and reached a peak value at 240 min 
(P<0.01**). WBC count declined to 61.2% of predialysis 
value and this difference achieved statistical significance at 
15 min (P<0.01**). There was a small but significant 
decrease in platelet count at 15 min after the onset of dialysis 
and a significant increase at 240 min (P<0.01**). Plasma 
β-TG significantly increased from a mean of 67±10 ng/ml at 
the beginning of dialysis to 76±25 ng/ml at 240 min of 
dialysis (P<0.05). Both C5C and C4 levels remained 
unchanged during haemodialysis. The LTB4 concentration 
reached a maximum of 3–4 times baseline value within 
240 min. The endogenous production of prostaglandin I2 
was assessed by measuring its stable end-products, 6-ketoPGE1a and PGE1a. Its concentration increased within 
240 min of dialysis (P<0.01). Plasma β-TG, as a marker of 
platelet activation, was measured to assess the release of

<table>
<thead>
<tr>
<th>Parameter</th>
<th>0</th>
<th>15 min</th>
<th>90 min</th>
<th>240 min</th>
<th>P values</th>
</tr>
</thead>
<tbody>
<tr>
<td>TxB2 (pg/ml)</td>
<td>82±28</td>
<td>75±18*</td>
<td>80±23*</td>
<td>105±37**</td>
<td>n.s.*</td>
</tr>
<tr>
<td>WBC (1 x 10³/mm³)</td>
<td>7.2±2.3</td>
<td>2.8±1.2**</td>
<td>6.6±1.9</td>
<td>7.4±1.9</td>
<td>&lt;0.01**</td>
</tr>
<tr>
<td>Platelet (1 x 10³/mm³)</td>
<td>198±11</td>
<td>163±9**</td>
<td>188±9</td>
<td>222±10**</td>
<td>&lt;0.01**</td>
</tr>
<tr>
<td>β-TG ng/ml</td>
<td>67±10</td>
<td></td>
<td></td>
<td>76±25</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>C5C mg/dl</td>
<td>24±8</td>
<td>24±5</td>
<td>27±9</td>
<td>27±6</td>
<td>n.s.</td>
</tr>
<tr>
<td>C4 mg/dl</td>
<td>40±6</td>
<td>41±11</td>
<td>38±9</td>
<td>38±9</td>
<td>n.s.</td>
</tr>
<tr>
<td>LTB4 pg/ml</td>
<td>130±103</td>
<td></td>
<td></td>
<td>310±285</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>6-keto PGE1a pg/ml</td>
<td>283±104</td>
<td></td>
<td></td>
<td>332±64</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>PGE1a pg/ml</td>
<td>39±7</td>
<td></td>
<td></td>
<td>54±4</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>
platelet granular contents pointing to membrane–platelet adhesion and/or aggregation. We confirmed reports [4,5] that this protein was elevated before dialysis and rose further during haemodialysis when cuprophane membranes were used. Seyfert et al. [6] showed higher β-TG levels for cuprophane as compared to PAN (AN69). In this study plasma TxB2, the stable metabolite of TxA2, was quantified for blood entering and leaving the dialysers to assess TxA2 generation during platelet–dialyser membrane interaction. Plasma TxB2 levels were shown to be higher during haemodialysis, indicating generation of TxA2. We demonstrated significant transient thrombocytopenia during haemodialysis with cuprophane membranes associated with a rise in plasma TxB2 levels. The time course mirrored the decrease in platelet counts. Moreover, the interaction of blood with cuprophane leads to a rapid and massive complement activation with leukopenia [7]. In the present study WBC count declined significantly at 15 min and this fall in WBC did coincide with the change in C3c and C4 levels. The changes in 6-keto PGF1α levels indicate that the cuprophane membrane induces alterations in blood components that lead to stimulation of PGI2 production by patient vascular endothelial cells that occurred during haemodialysis.

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Tacrolimus can resolve cyclosporin-induced gingival hyperplasia

Sir,

Gingival hyperplasia is a well recognized potentially serious side-effect of cyclosporin therapy occurring in up to 30% of kidney transplant recipients treated with cyclosporin-based immunosuppressive regimens [1]. Poor oral hygiene, dental appliances and concomitant administration of dihydropyridine calcium channel antagonists or phenytoin augment the hyperplastic reaction to cyclosporin [2]. The macrolide antibiotic azithromycin has been shown to partially reverse cyclosporin-induced gingival hyperplasia [3]. Tacrolimus is an alternative immunosuppressive agent to cyclosporin, having a similar mode of action on T-cell function. Gingival hyperplasia has been described rarely in patients treated with primary tacrolimus therapy [4]. A switch of immunosuppres-