EFFECT OF HEMODIALYSIS ON TRACE ELEMENTS IN PATIENTS WITH ACUTE AND CHRONIC RENAL FAILURE

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ABSTRACT

Hemodialysis is being implicated in the development of metabolic disturbances, as complications have been observed and the role of trace metals in their development has been questioned. In 78 renal failure patients who underwent hemodialysis, serum levels of zinc and copper were determined before and after first hemodialysis. Acute and chronic renal failure patients were found to have lower serum zinc levels before hemodialysis and higher levels after hemodialysis, while serum copper levels were found to be higher before hemodialysis and to increase further after hemodialysis. Disturbances in acid-base balance lead to acidosis which can alter the binding, transport and excretion of these elements. Dialysis fluid containing salts of trace elements may have also contributed to this alteration of trace element levels.


Keywords: Blood trace metals, Hemodialysis, Renal failure.

INTRODUCTION

Essential trace metals are involved in a number of metabolic activities, including neuroconduction, transport, excretory processes and serving as cofactors for enzymes. The cells of the proximal renal tubule have an important role in the homeostasis of essential metals, and the kidney is a target site for metal toxicity.¹ Zinc and copper are two of the most intensively investigated and metabolically important trace metal nutrients. For this reason, and because of their close chemical similarity and extensive biological interaction, they are often considered together.² In recent years, intermittent hemodialysis has been successful in extending the life span of patients with renal failure. However, excess or deficiency of trace elements, which was disregarded until several years ago, has been noticed as one of the factors that induce complications in dialyzed patients.³ The accurate determination of trace metals was limited by methodology prior to the last two decades. This resulted in a limited knowledge of the concentrations of metals in many diseases, including renal disease. Fortunately, this situation is being remedied to a major extent by atomic absorption spectrophotometry.⁴ Hence the present study was undertaken to evaluate the alterations in trace element levels due to hemodialysis in renal failure patients.

MATERIAL AND METHODS

The present study was conducted in Government Medical College, Miraj, India. The assessment includes 120 subjects ranging from 20–60 years old of both sexes, out of which 42 cases were healthy controls, 41 cases had chronic renal failure (group 1) and 37 cases had acute renal failure (group 2). Patients’ samples were collected before and after their first hemodialysis. The serum was separated, and zinc and copper levels were estimated by atomic absorption spectrophotometry method.⁵
Effect of Hemodialysis on Trace Elements

Normal control Pre HD CRF Post HD CRF Pre HD ARF Post HD ARF

Fig. 1. Levels of zinc in acute and chronic renal failure before and after first hemodialysis.

RESULTS

As shown in Table I and Fig. 1 serum zinc levels were decreased quite significantly in acute as well as in chronic renal failure patients as compared to the control. The mean zinc levels were increased quite significantly after first hemodialysis as compared to pre-hemodialysis ($p<0.001$) in both groups. Table II and Fig. 2 represent serum copper levels in acute and chronic renal failure before and after hemodialysis.

DISCUSSION

In healthy persons the kidney is a regulatory organ that allows for variations in dietary intake by causing corresponding alterations in excretion. The mechanisms responsible for the trace element disturbances found in renal failure patients are probably multiple and multifactorial. In renal failure, patients have disturbances in acid-base balance and blood pH is acidic, therefore low zinc levels in these patients are believed to be due to the shift of zinc into red cells under acidic conditions. Manifestations of zinc deficiency, including skin lesions and poor wound healing, have been observed in these patients. Zinc present in serum is combined with plasma proteins, especially with albumin. Proteinuria in renal failure leads to excessive excretion of zinc along with protein, hence continued urinary loss of zinc might ultimately lead to low serum zinc levels.

In renal failure patients the renal capacity for excreting excess copper diminishes, which leads to accumulation of this element in the body causing toxicity with neurologic, hepatic and corneal lesions.

Although reports are available concerning the favourable effect of hemodialysis on decreased serum antioxidant activity,⁴ the changes in body composition in chronic hemodialysis patients before and after hemodialysis have also been reported.⁷ Dialysis remarkably contributes to an increase in many trace elements. The dialysis fluids which are used in different dialytic treatments contain variable amounts of trace elements. Among 15 elements determined in hemodialysis fluid, Salvadeo et al.⁸ showed that copper, iron and zinc were present at the highest concentrations. Hence this could reflect the wide range of trace metal concentrations in such patients. In the process of hemodialysis, plasma actively takes up both copper and zinc from the dialysis fluid even against a concentration gradient, and since zinc binds actively to albumin, it is not refluxed back to the dialysis fluid. Acidosis may favour trapping of copper from dialysis salt by sialic acid to synthesize ceruloplasmin, which may lead to increased concentrations of copper in serum. Hence our results indicate that dialysis causes changes in the serum zinc and copper levels in these patients.

Table I. Serum zinc levels in acute and chronic renal failure patients before and after first hemodialysis.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Zn (μg/dL) Pre HD Mean ± S.D.</th>
<th>Zn (μg/dL) Post HD Mean ± S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>72.5 ± 9.8*</td>
<td>86.26 ± 9.21*</td>
</tr>
<tr>
<td>CRF (n=41)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group 2</td>
<td>70.96 ± 10.11*</td>
<td>87.54 ± 9.17*</td>
</tr>
<tr>
<td>ARF (n=37)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal control</td>
<td>99.97 ± 9.12 μg/dL</td>
<td></td>
</tr>
</tbody>
</table>

*Highly significant, $p<0.001$.

Table II. Serum copper levels in acute and chronic renal failure patients before and after first hemodialysis.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Cu (μg/dL) Pre HD Mean ± S.D.</th>
<th>Cu (μg/dL) Post HD Mean ± S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>138.52 ± 9.02*</td>
<td>152.51 ± 10.29*</td>
</tr>
<tr>
<td>CRF (n=41)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group 2</td>
<td>132.12 ± 9.54*</td>
<td>150.10 ± 10.10*</td>
</tr>
<tr>
<td>ARF (n=37)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal control</td>
<td>105.43 ± 12.17 μg/dL</td>
<td></td>
</tr>
</tbody>
</table>

*Highly significant, $p<0.001$.
REFERENCES
