Plasma hepcidin and trace elements in chronic renal failure

K.Yamaya[1], S. Tsuboi[1], C. Tsutaya[1], M. Sutoh[1], Y. Hashimoto[1], H. Saitoh[1], T. Funyu[1]
Y. Shimonaka[2], K. Sera[3], S. Goto[4], S. Hatakeyama[5] and C. Ohyama[5]

[1]Oyokyo Kidney Research Institute
90 Yamazaki Kozawa Hirosaki 036-8243, Japan

[2]Chugai-pharmaceutical Co. Ltd.
200 Kajihara Kamakura Kanagawa 247-8530 ,Japan

[3]Cyclotron Research Center, Iwate Medical University
348-58 Tomegamori, Takizawa, Iwate 020-0173, Japan

[4]Takizawa Laboratory, Japan Radioisotope Association
348-1 Tomegamori, Takizawa, Iwate 020-0173, Japan

[5]Department of Urology, Hirosaki University Graduate School of Medicine
[5] Zaifu-cho Hirosaki 036-8562, Japan

Abstract

Iron deficiency and anemia are commonly in chronic renal failure. Hepcidin, a peptide hormone synthesized in the liver is a key homeostatic regulator of iron metabolism.

It is known that the trace transition elements are also related to anemia and iron revel,however, little is known about the interaction of hepcidin with trace elements.

We examined the association between plasma hepcidin and iron, zinc, selenium and copper concentrations in 150 regular hemodialysis patients. Hepcidin and trace elements (iron, zinc, selenium and copper) in blood were measured respectively by using mass spectrometry and PIXE method.

As a result, although Epo medication or the HB level did not influence each metal concentration, the significant relationships were found between hepcidin and iron ($r=0.22$, $p<0.05$) or zinc ($r=-0.21$, $p<0.05$). Moreover the significant relationships were found between total iron-binding capacity (TIBC) and zinc , selenium or copper .

In conclusion, a possibility that the hepcidin level influenced the iron level was suggested. And a possibility that zinc, selenium and copper level also influenced the hepcidin level was suggested.