短 報

ABSORPTION OF METALLIC MERCURY FROM THE INTESTINE AFTER RUPTURE OF MILLER-ABBOT BALLOON

ミラー・アボット ゾンデ破損時の
金属水銀の腸管吸収

The Miller-Abbot balloon containing a considerable amount of metallic mercury has been used in the treatment for patients suffered from some of intestinal obstructive conditions. In the procedure of treatment, the balloon may come into rupture¹⁾. By Sollman²⁾, the use of metallic mercury given in doses 100 to 500 g for the treatment of paralytic ileus, usually does not produce symptoms of poisoning but sometimes cause diarrhoea and stomatitis; and exceptionally, severe and even fatal poisoning is resulted.

Whether the lesion in the epithelium of intestine is necessary for the absorption of metallic mercury is still disputable³⁾, but the absorption is claimed to occur in Krais's report¹⁾. Bornman et al. demonstrated in rats that an oral administration of 2.5 g of metallic mercury induced the tenfold or more increase of mercury contents in blood and kidney over normal values⁴⁾.

We have estimated mercury contents in the red cell, plasma and urine from a patient with ileus, in whom a rupture of Miller-Abbot balloon happened and about 10 ml (134.6 g) of metallic mercury leaked in the stomach.

Mercury was estimated by the method of Magos and Cernik⁵⁾, which estimates only the inorganic mercury.

Results

Results are shown in the figure and summarized as follows.

1) The mercury content in red cells stays almost constant up to 122 hours after the leakage of mercury.

2) Since the early period after the leakage, plasmatic mercury contents are higher than erythrocytic ones. This distribution pattern of mercury in the blood has to be noted, because it resembles that of inorganic mercury in the blood of workers exposed to mercury vapour⁶⁾.

A peak of plasmatic mercury level is observed at around 50 hours after and a gradual rise thenceforth is paralleled with the change of urinary

mercury content.

3) As general tendency, urinary mercury contents increase until 240 hours after the leakage. In the early period, the level is already higher than the normal upper limit of 20 $\mu\text{g/l}$ in lay populations without any particular exposure to mercurials.

Discussion

The physician in charge followed the mercury by X-ray observations. The mercury moved from the stomach to the small intestine after the fortunate recovery from ileus, scattered to a lot of droplet, attached to the mucous membrane, and stayed there for about one week. The patient and the physician noticed the fecal excretion of metallic mercury at one week after the leakage.

Because the physician ceased to sample the specimens at 10 days after the leakage by his judgement that there was no danger of poisoning, we could not check if there would be further increase of plasmatic and urinary mercury contents. As far as the present data are concerned, the absorption of mercury from the intestine is not negligibly small but large enough to call our attention to the possibility of poisoning.

We should have estimated the mercury content in the plasma or urine longer, because the mercury level found in the urine was over 300 $\mu\text{g/l}$ (the alarming level for workers exposed to mercury vapour to reduce their mercury exposure thereafter, at 240 hours after the leakage. To prevent a possible danger in such a large dose of metallic mercury as over 100 g, the estimation of mercury for longer period than present one may be recommended.

No increase in the erythrocytic mercury level, in contrast to the elevation of plasmatic mercury contents, means that the transportation of inorganic mercury depends on the plasma. The parallelism between the plasmatic and urinary mercury contents is also a worthy finding to understand the excretory mechanism through the kidney.

References

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Fig. Changes of Mercury Contents in Red Cells, Plasma and Urine from a Patient, in Whose Stomach Metallic Mercury leaked from the Miller-Abbot Balloon.