Correspondence

High urinary cadmium concentration in a case of gastric cancer

SIR—It is well known that cadmium (Cd) adversely affects renal tubular function at an early stage and may induce osteomalacia in severe cases.¹ In 1981 and 1982 the 3119 subjects living in a Cd polluted area in Japan and 289 non-exposed controls were examined by collecting morning urine samples (table 1).² Since then several subjects with renal tubular dysfunction have been observed.

Among these cases was one showing an extreme increase of urinary Cd concentration. Such a high Cd concentration has never been found, despite our having studied environmental Cd effects for more than 20 years. The 71 year old woman presented in 1982 with slightly raised Cd and β_2 -microglobulin concentrations in the urine (table 2). One year later the urinary Cd concentration had increased by more than 200 times. This value was higher than the maximum urinary Cd concentration in table 1. Indicators of renal tubular dysfunction such as urinary β_2 -microglobulin, α_1 -microglobulin, and metallothionein also showed pronounced increases (table 2). Serum creatinine was 1.38 mg/dl and glomerular function was maintained.

The cadmium concentration in whole blood was 133.0 $\mu\text{g/l}$. This was also unusually high; the geometric mean and standard deviation of 205 subjects with

Table 1 Urinary cadmium concentrations ($\mu\text{g/l}$) of Cd exposed subjects living in a cadmium polluted area in Japan and controls

Subjects	Sex	No	GM	GSD	Min	Max
Cd exposed	M	1403	4.02	2.023	0.3	37.5
	F	1716	3.86	2.077	0.3	105.5
Controls	M	130	1.84	1.992	0.2	13.6
	F	159	1.69	2.069	0.2	11.0

GM = Geometric mean.

GSD = Geometric standard deviation.

Table 2 Urinary findings

Sample	November 1982 November 1983		
	Early morning urine	Total urine day	Urine per 2 hours
Cadmium ($\mu\text{g/l}$)	6.2	1330.6	2064.3
β_2 -Microglobulin ($\mu\text{g/l}$)	1231	27433	121 621
α_1 -Microglobulin ($\mu\text{g/l}$)			188 962
Metallothionein (ng/l)			> 512

renal tubular dysfunction caused by exposure to Cd in 1983 was $8.4 \pm 1.9 \mu\text{g/l}$.

Between these examinations, she was operated on for advanced gastric cancer. Nine months later she died and no necropsy was performed. In those days the mine which was the source of Cd pollution had already closed and the level of Cd exposure had not changed. It may be that invasion of the stomach cancer, with metastases to the liver and peritoneum, and the operation itself caused the transfer of Cd from these organs to the kidneys leading to increased urinary Cd excretion.

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