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Aluminium in Antacids and Cooking Pots and the Risk of Hip Fractures in Elderly People

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Summary

The relationship between hip fractures and aluminium in antacids and cooking pots was examined in an epidemiological study in Sydney, Australia. A population-based case–control study was conducted and 416 men and women aged 65 years and over were recruited (209 cases and 207 controls). There was a significantly ($p < 0.05$) increased risk of hip fracture associated with use of aluminium cooking pots at age 20 years: the age- and sex-adjusted odds ratio was 1.9 [95% confidence interval (CI) 1.1–3.2]. No association was detected between risk of hip fracture and current use of aluminium cooking pots or use at age 50. There was some suggestion that long-term use of aluminium-containing antacids might increase the risk of hip fracture: the adjusted odds ratio comparing those who had used aluminium-containing antacids for more than 10 years with those who had never used them was 1.8 (95% CI 0.8–4.1). The findings of this study support the need for further research on the association between oral ingestion of aluminium and risk of hip fracture.

Introduction

There is evidence that the age-adjusted incidence of hip fractures is increasing [1]. It has recently been suggested that increasing exposure to aluminium during the twentieth century might be part of the explanation [2, 3]. This hypothesis has not been tested.

The main evidence that aluminium might increase the risk of hip fractures comes from studies of patients on renal dialysis. In 1977, Platts and colleagues noted that home dialysis patients who lived in areas with a water supply high in aluminium were more likely to suffer fractures than patients living in other areas [4]. Subsequently, Ellis *et al.* showed that patients on renal dialysis had higher levels of aluminium in their bones than control subjects [5]. They also found that rats given intraperitoneal injections of aluminium developed osteomalacia. Robertson *et al.* have since shown that aluminium administration results in reduced bone formation in rats [6].

There have been no studies of the effects of aluminium on the risk of hip fracture. Our aim in the research described in this paper was to determine whether or not there was any association between use of aluminium-containing antacids and aluminium cooking pots and risk of hip fracture. This research was part of a recently completed epidemiological study of risk factors for hip fracture in elderly Australians [7].

Subjects and Methods

The methodology for this case–control study is described in detail elsewhere [7]. In summary, we tried to identify all hip fracture cases occurring in a geographically defined population in the western suburbs of Sydney, Australia, during 1990–91 and compared their exposure histories with those of a random sample of people without hip fractures (controls) from the same geographic area.

Cases were selected from the 12 hospitals that we identified as treating patients with hip fractures who lived in the study area. The majority of cases (75%) came from Westmead Hospital, a 1000-bed teaching hospital.

Control subjects living in private homes in the community were selected with an area probability sampling method. Ten census collectors' districts (CDs) from the study area were randomly selected by the Australian Bureau of Statistics (ABS) and all dwellings in each CD were visited (a total of 2560 dwellings). We tried to interview all people aged 75 years and over, as well as a 10% random sample of people aged 65–74 years. Controls were also selected from nursing homes and hostels. Six nursing homes and three hostels were selected at random from nursing homes ($n = 28$) and hostels ($n = 12$) in the study area. Five people aged 75 years or more were selected at random from each selected nursing home and hostel.

An interviewer-administered questionnaire was used to measure exposures of interest. For people with cognitive impairment or various other health problems, a shortened questionnaire was used in an interview with a proxy respondent.

Directly interviewed subjects were asked what their cooking pots were made of at various times during their life: age 20 years (when bone density is increasing); age 50 years (when bone density is decreasing); and at current age. The stewing of acidic fruits (such as rhubarb and tomatoes) in aluminium pots leads to increased aluminium content of these foods [8]; hence, subjects were asked how often they ate stewed fruit at the three ages listed above. Proxy respondents were asked a question about

Table I. Selected characteristics of cases with hip fracture and controls

Characteristic	Cases (n = 209) No. (%)	Controls (n = 207) No. (%)
No. of men	35 (17)	70 (33)
No. of women	174 (83)	137 (67)
Age group (years)		
65–74	41 (20)	35 (17)
75–79	37 (18)	77 (37)
80–84	57 (27)	60 (29)
85–89	45 (21)	25 (12)
90–100	29 (14)	10 (5)
Residence		
Community	130 (62)	166 (80)
Hostel	10 (5)	14 (7)
Nursing home	69 (33)	27 (13)
Proxy respondent needed	84 (40)	30 (14)

frequency of eating fruit cooked in aluminium pots during adult life.

Use of aluminium-containing antacids was assessed in all subjects by inquiring about all medications that were currently used. In addition, information on the name(s) and duration of use of any antacid ever taken for more than 6 months was collected from directly interviewed subjects. Antacids were then classified as aluminium-containing or not. Most antacids used by subjects in this study contained aluminium—92% of currently used antacids and over 80% of antacids ever used.

The reproducibility of reports of use of aluminium pots and many other exposures was tested in a random sample of 63 directly interviewed subjects (28 cases and 35 controls). These subjects were re-interviewed 1–3 months after the original interview. Details of the reproducibility study have been published elsewhere [9].

Data analysis involved crude, stratified, and multivariate methods to produce odds ratios for associations between hip fracture and exposure to aluminium-containing products [10]. Age and sex are known to be strong predictors of hip fracture and so all results were adjusted for age and sex. Proxy status was included in relevant analysis to minimize any measurement bias due to use of proxy respondents. Multiple logistic regression was used to assess aluminium–hip fracture relationships while simultaneously controlling for several confounding variables. The measurement of these confounding variables is described elsewhere [7, 9].

Results

Four hundred and sixteen subjects participated in this study: 209 cases and 207 control subjects. The response rate was 96% for cases and 84% for controls. The age distribution and some other characteristics of cases and controls are shown in Table I. Proxy respondents were required for 40% of cases and 14% of controls; most of the results reported here are based on data from directly interviewed subjects only (125 cases and 177 controls).

The main results for use of aluminium pots are shown in Table II. There was a statistically significant ($p < 0.05$) increased risk of hip fracture for those who reported using aluminium cooking pots at age 20 years (age- and sex-adjusted odds ratio 1.9). There was some suggestion that aluminium pot users at age 20 who ate stewed fruit regularly were at greater risk of hip

Table II. Adjusted odds ratios in directly interviewed study subjects for risk of hip fracture and use of aluminium cooking pots and eating stewed fruit[a]

Variable		No. of cases	No. of controls	Adjusted odds ratios (95% CI)
Regular use of aluminium pots				
Age 20 years:	No	44	97	1.00 (referent)
	Yes	71	74	1.92 (1.13–3.24)
Age 50 years:	No	14	29	1.00 (referent)
	Yes	107	143	1.58 (0.77–3.22)
Current age:	No	34	59	1.00 (referent)
	Yes	82	14	1.11 (0.65–1.90)
Fruit stewed in aluminium pots[b] (times per week)				
Age 20 years:	< 1	13	25	1.00 (referent)
	1–6	40	37	1.44 (0.56–3.67)
	⩾ 7	16	10	2.07 (0.67–6.45)[c]
Age 50 years:	< 1	27	44	1.00 (referent)
	1–6	61	82	1.15 (0.64–2.09)
	⩾ 7	15	17	1.42 (0.55–3.65)
Current age:	< 1	48	70	1.00 (referent)
	1–6	26	36	1.09 (0.54–2.21)
	⩾ 7	4	8	0.71 (0.18–2.90)

[a]Odds ratios are adjusted for age group and sex. CI denotes confidence interval. [b]Only subjects who reported regular use of aluminium pots are included here. [c]Test for trend: $p = 0.24$.

Table III. Adjusted odds ratios for risk of hip fracture and use of aluminium-containing antacids[a]

Variable	No. of cases	No. of controls	Adjusted odds ratios (95% CI)
All study subjects			
Current use of aluminium-containing antacids			
No	192	181	1.00 (referent)
Yes	17	26	0.73 (0.34–1.57)
Directly interviewed subjects only			
Regular use of aluminium-containing antacids (ever)			
Never	80	121	1.00 (referent)
1–2 years	8	18	0.69 (0.28–1.72)
3–9 years	11	11	1.64 (0.62–4.38)
⩾10 years	15	18	1.78 (0.80–4.07[b]

[a]Odds ratios for directly interviewed subjects only are adjusted for age group and sex; odds ratios for all subjects are adjusted for age group, sex and proxy status. CI denotes confidence interval. [b]Test for trend: p = 0.19.

fracture than aluminium pot users at age 20 who did not eat stewed fruit (Table II). The adjusted odds ratios for aluminium pot use at age 50 years and at current age were not statistically significant. Adjusting for number of years of education (a measure of social class) made no difference to these odds ratios.

Proxy respondents were asked to estimate how often the subject ate fruit stewed in aluminium pots during his/her adult life. The adjusted odds ratio comparing those who ate fruit stewed in aluminium pots more than once a week and those who ate it less frequently was 2.6 (95% CI 0.9–7.1).

No relationship was apparent between current use of aluminium-containing antacids and risk of hip fracture (Table III). There was some suggestion, however of an increased risk of hip fracture with long-term regular use of aluminium-containing antacids. The adjusted odds ratio for hip fracture was 1.8 for those who had ever used aluminium-containing antacids for 10 years or more compared with those who had never used these antacids. This association was not statistically significant.

There were ten subjects in this study who had used aluminium pots at all three ages studied and also used aluminium-containing antacids for at least 10 years (high aluminium exposure group). There were 13 subjects who had not used aluminium pots at any of the ages studied and never used aluminium-containing antacids (low aluminium exposure group). The crude odds ratio for hip fracture comparing the high exposure group to the low exposure group was 8.3 (95% CI 1.2–55.1). The age- and sex-adjusted odds ratio was 6.3 (95% CI 0.8–48.7).

The association between aluminium pot use at age 20 years and risk of hip fracture persisted after adjusting for multiple confounders (age, sex, alcohol, body mass index, caffeine intake, cognitive status, dairy product intake, health status, physical activity, psychotropic drug use, and smoking) in a logistic regression model: the adjusted odds ratio was 1.9 (95% CI 0.9–3.7). However, the association between duration of use of aluminium-containing antacids and risk of hip fracture was greatly reduced after adjustment for the confounders listed above: the adjusted odds ratios were 0.9 (95% CI 0.3–2.7) for use for 1–2 years; 1.6 (95% CI 0.5–4.9) for use for 3–9 years; and 1.2 (95% CI 0.4–3.1) for use for more than 10 years.

Based on repeat interviews with 63 randomly selected subjects, the reliability of reported aluminium pot use was only fair: kappas were 0.53 (age 20 years); 0.35 (age 50 years); and 0.47 (current age). The rank correlation coefficients for frequency of eating stewed fruit at age 20 years, age 50 years, and at current age were 0.66, 0.54, and 0.60, respectively.

Discussion

This is the first epidemiological study of the hypothesis that aluminium in antacids and cooking pots increases the risk of hip fracture. In support of the hypothesis, we found that use of aluminium pots at age 20 years was associated with an increased risk of hip fracture. This association persisted after adjusting for numerous confounders. The findings in relation to use of aluminium-containing antacids and eating fruit stewed in aluminium pots were more equivocal.

The finding of a harmful effect of aluminium pot use only for age 20 years needs explanation. Most of the subjects in this study were aged 20 in the 1920s and 1930s: perhaps older style aluminium pots were more likely to have the aluminium leached out of them than more recently manufactured pots. Alternatively, aluminium might have an effect on bone that is still increasing in density (as it is in a 20 year old) but have no effect on the rate at which bone is lost in older people. This would be consistent with the observation that children with renal failure appear to be at greater risk of aluminium-related bone disease than adults with renal failure [11].

Leaching of aluminium from cooking pots is most

likely if acidic fruits are cooked in aluminium pots for several hours [8]. In the 1930s, Beal and colleagues found that stewing tomatoes, rhubarb, or apples in aluminium pots increased the aluminium content of these foods as much as 100-fold [12]. If aluminium is related to hip fractures, it would be expected that subjects who used aluminium pots and who were frequent consumers of stewed fruit would be at greater risk of hip fracture than aluminium pot users who rarely ate stewed fruit. There was some weak evidence for this in our study.

It has been established that patients with renal failure can accumulate aluminium in their bones, owing to a combination of high levels of exposure to aluminium and impaired excretion [11]. However, it is unclear whether orally ingested aluminium is deposited in the bones of people without renal disease. Recker *et al.* reported increased bone aluminium content in a 49-year-old man with peptic ulcer disease and normal renal function who had used antacids for 25 years [13]. The recent accident at Camelford in Cornwall, when large amounts of aluminium sulphate were inadvertently put into local drinking water, also provides support for the possibility that oral aluminium can be deposited in the bones of normal people [14].

It is now recognized that aluminium hydroxide in antacids binds with dietary phosphate in the gut, which can lead to hypophosphataemia and subsequent osteomalacia [15]. This is another mechanism by which oral aluminium could increase the risk of hip fracture in people with normal renal function.

This is the first epidemiological study of the relationship between exposure to aluminium and hip fracture. Wood *et al.* investigated the association between aluminium in drinking water and bone density [16]. They examined bone density in the femoral neck in 386 hip-fracture patients, some of whom lived in an area with high aluminium water content and some of whom lived in an area with low levels of aluminium in the water [16]. There was no difference in bone density between the two groups of patients.

The main limitation of our study is the validity of the measurement of aluminium exposure—do people know what their cooking pots are made of? Although the reliability of reported aluminium pot use was not high in this study (kappas ranged from 0.35 to 0.53), it was no worse than for many other variables studied by epidemiologists. A systematic over-reporting of aluminium pot use by case subjects compared to controls could explain the observed increased risk of hip fracture associated with use of aluminium pots at age 20 years. This is possible but seems unlikely.

Could use of aluminium-containing products explain the increase in age-adjusted hip fracture incidence rates that have been observed in many parts of the world [1] (including Australia [17])? Although aluminium was first extracted from bauxite in 1827, it was not until 1886 that an electrolytic process was discovered that made it available cheaply enough for everyday use [18]. Hence, only people born in the twentieth century have had the opportunity to be exposed to aluminium for most of their lives.

While far from conclusive, the findings of the study reported in this paper suggest that epidemiological studies are needed that are specifically designed to investigate the possibility that there is a hip fracture–aluminium association. These studies should assess aluminium exposure from a range of sources that include the diet, drinking water, cooking pots, deodorants, and medications.

References

1. Melton LJ, O'Fallon WM, Riggs BL. Secular trends in incidence of hip fracture. *Calcif Tissue Int* 1987;**41**:57–64.
2. Obrant KJ, Bengner U, Johnell O, Nilsson BE, Sernbo I. Increasing age-adjusted risk of fragility fractures: a sign of increasing osteoporosis in successive generations? *Calcif Tissue Int* 1989;**44**:157–67.
3. Mjoberg B. Aluminium may cause senile dementia and bone fragility. *Calcif Tissue Int* 1989;**47**:259–60.
4. Platts MM, Goode GC, Hislop JS. Composition of the domestic water supply and the incidence of fractures and encephalopathy in patients on home dialysis. *Br Med J* 1977;**2**:657–60.
5. Ellis HA, McCarthy JH, Herrington J. Bone aluminium in haemodialysed patients and in rats injected with aluminium chloride: relationship to impaired bone mineralisation. *J Clin Pathol* 1979;**32**:832–44.
6. Robertson JA, Felsenfeld AJ, Haygood CC, Wilson P. Clarke C, Llach F. Animal model of aluminium-induced osteomalacia: role of chronic renal failure. *Kidney Int* 1983;**23**:327–35.
7. Cumming RG, Klineberg RJ. Case–control study of risk factors for hip fractures in the elderly. *Am J Epidemiol* 1994;**139**:493–503.
8. Pennington JAT. Aluminium content of foods and diets. *Food Addit Contam* 1987;**5**:161–232.
9. Cumming RG, Klineberg RJ. A study of the reproducibility of long-term recall in the elderly. *Epidemiology* 1994;**5**:116–19.
10. Rothman KJ. *Modern epidemiology*. Boston: Little, Brown & Co., 1986.
11. Sherard DJ, Andress DL. Aluminium-related osteodystrophy. *Adv Intern Med* 1989;**34**:307–24.
12. Beal GD, Unangst RB, Wigman HB, Cox GJ. Aluminium content of foodstuffs cooked in glass and in aluminium. *Ind Eng Chem* 1932;**24**:407–9.
13. Recker RR, Blotcky AJ, Leffler JA, Rack EP. Evidence for aluminium absorption from the gastrointestinal tract and bone deposition by aluminium carbonate ingestion with normal renal function. *J Lab Clin Med* 1977;**90**:810–15.
14. Eastwood JB, Levin GE, Pazanias M, Taylor AP, Denton J, Freemont AJ. Aluminium deposition in bone after contamination of drinking water supply. *Lancet* 1990;**336**:462–4.
15. Chines A, Pacifici R. Antacid and sucralfate-induced hypophosphatemic osteomalacia: a case report and review of the literature. *Calfic Tissue Int* 1990;**47**:291–5.
16. Wood DJ, Cooper C, Stevens J, Edwardson J. Bone mass and dementia in hip fracture patients from areas with different aluminium concentrations in water supplies. *Age Ageing* 1988;**17**:415–19.

17. Lau EMC. Admission rates for hip fracture in Australia in the last decade. *Med J Aust* 1993;**158**:604–6.
18. Prescott A. What's the harm in aluminium? *New Sci* Jan 1989;**21**:58–62.

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