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# Bone cement, thermal injury and the radiolucent zone

Six patients, with giant cell tumors of bone treated by curettage and bone cementing, were followed radiographically for 4 (2–6) years postoperatively.

The radiolucent zones between cortical bone and cement increased in width up to 0.5 mm during the first 6 months and then remained stationary. During the same period the zone adjacent to cancellous bone increased up to 2.5 mm and was surrounded by a sclerotic rim. In two cases the zone adjacent to cancellous bone diminished by 1–1.5 mm during the first 2 years after surgery. There was a positive correlation between the maximal width of the zone adjacent to cancellous bone and the volume of cement fillings.

Our findings indicate that the radiolucent zone surrounding bone cement is caused by thermal necrosis.

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A radiolucent zone frequently appears at the bone-cement interface following arthroplasties (Salvati et al. 1976, Ahlberg & Lindén 1977, Beckenbaugh & Ilstrup 1978, Jónsson 1981). Several explanations for this have been suggested: vascular injury (Slooff 1971, Rhinelander et al. 1979, Sund & Rosenqvist 1983), thermal or cytotoxic injury (Willert et al. 1974, Feith 1975), infection (Bergström et al. 1974), reaction to wear products (Brinkmann & Heilmann 1974), metal sensitivity (Evans et al. 1974), micromovements (Salvati et al. 1976), and stimulation of macrophages by cement (Freeman et al. 1982).

To study the development of the zone in a model where several of these causes are not applicable, we have analysed its appearance in cases of giant cell tumors of bone (GCT) treated by curettage and cementing.

## Material and methods

During the period 1972–1982, 14 patients with primary or locally recurrent GCT were treated by curettage and bone cementing (Persson et al. 1984). Patients with local recurrence, fracture, inefficient packing of bone cement, migration of the cement filling through a large cortical opening, and bone chips remaining from a previous operation were excluded, leaving six patients suitable for analysis. There were five women and one man, with a mean age of 31 (15–40) years. The tumors were located in the distal radius (1), distal femur (3), and proximal tibia (2). All operations were performed in a bloodless field. There were no postoperative infections.

The mean follow-up time was 3.7 (2–6) years. During this period the patients had 6–12 conventional radiographic examinations (frontal and lateral exposures) each, giving a total of 50 examinations. All radiographs were reviewed with regard to the volume of cement fillings, the width of the radiolucent zone, and the development of a sclerotic rim. The width of the zone, measured with a scale loupe having a magnification of 7, was measured at 5 mm intervals around the cement fillings in the frontal and lateral projections.

The cement fillings were divided into 1 cm thick discs perpendicular to the long axis of the bone (Figure 1). These discs were measured in the frontal and lateral projections and approximated to elliptical cylinders. The volume of a filling was calculated as the sum of the volumes of the corresponding discs.

## Results

A radiolucent zone between cement and cortical bone developed in all cases. It increased in width to a maximum of 0.5 mm during the first 6 months and then remained stationary. During the same period a zone between cement and cancellous bone developed and increased in width up to a maximum of 0.5–2.5 mm (Figure 2). In two cases the zone diminished by

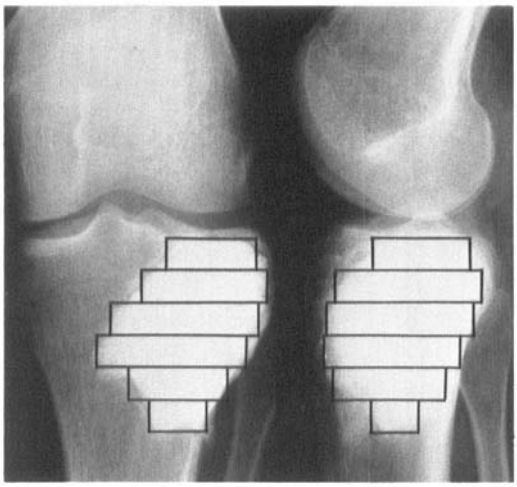

Figure 1. A cement filling divided into 1-cm-thick discs for calculation of the volume.

1–1.5 mm during the first 2 years after surgery (Figures 2 and 3).

In three locations in two cases where the cement filling adjacent to cancellous bone was concave, a considerably wider zone developed (Figure 3).

In the cancellous bone a distinct sclerotic rim surrounding the radiolucent zone developed within 3–7 months in all cases (Figure 2). This sclerotic rim migrated toward the cement filling in the two cases whose radiolucent zones diminished (Figures 2 and 3).

There was a significant positive correlation between the maximal mean width of the zone adjacent to the cancellous bone, and the volume of the cement filling (Figure 4).

## Discussion

An increasing radiolucent zone is caused by resorption of a layer of necrotic bone near the cement (Willert et al. 1974). This necrosis has been attributed to peroperative trauma: local vascular injury at reaming (Slooff 1971, Rhinelander et al. 1979, Sund & Rosenqvist 1983), thermal injury at polymerization (Willert et al.

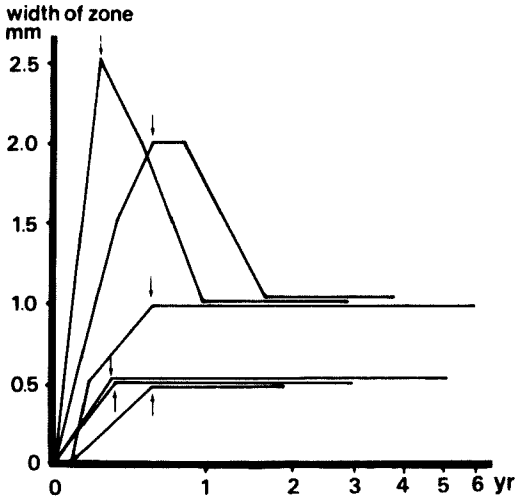


Figure 2. The development of the radiolucent zone (maximal width in 0.5 mm intervals) adjacent to the cancellous bone. Arrows indicate appearance of the sclerotic rim.

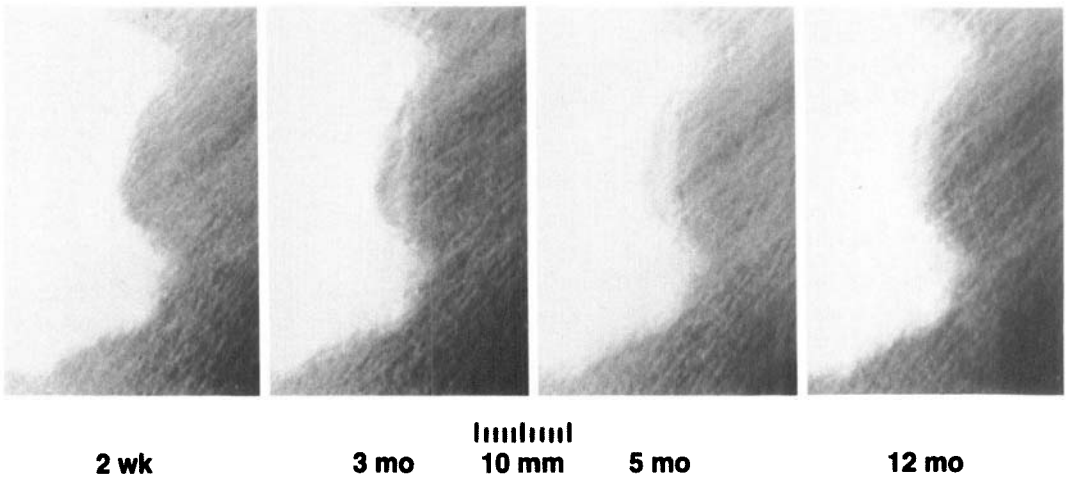


Figure 3. The development of the radiolucent zone adjacent to a concavity in a cement filling 2 weeks, 3 months, 5 months and 12 months after surgery.

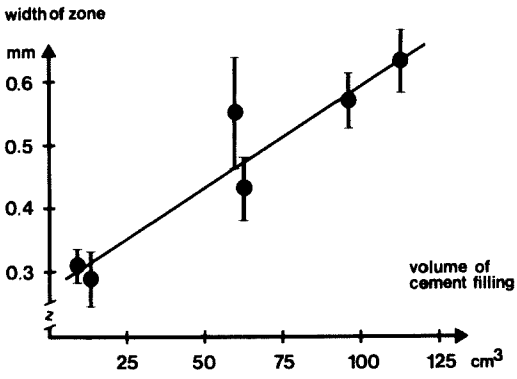


Figure 4. Width (maximal mean) of the radiolucent zones adjacent to the cancellous bone related to the volume of the fillings. $r = 0.95$, $p < 0.004$. Vertical bars indicate s.e.m.

1974, Feith 1975) and cytotoxicity by the methylmethacrylate monomer (Willert et al. 1974, Feith 1975). Rhinelander et al. (1979) demonstrated that surgical trauma caused vascular disturbance in the diaphysis, but not in the cancellous bone of the metaphysis. Furthermore, Feith (1975) showed that thermal injury at polymerization was more important. The threshold temperature for cell injury is time-dependent; epidermal cell necrosis occurs according to Moritz & Henriques (1947) after 5 s at 60°C, after 30 s at 55°C and after 5 min at 50°C. Based on these findings, Huiskes (1980) calculated a 'necrosis map' for the hip (Figure 5) and concluded that a larger layer of bone became necrotic in areas adjacent to a concave cement surface. Methylmethacrylate

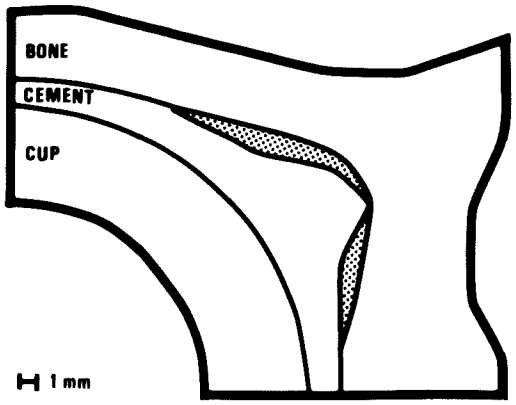


Figure 5. Necrosis map, simplified after Huiskes (1980). ⁘ = thermal necrosis of bone.

monomer is highly cytotoxic (Feith 1975, Linder 1976), but the local effect of the leaking monomer is considered insignificant (Linder 1977). Also, the monomer leakage is proportional to the surface area, but independent of the thickness of the cement (Linder et al. 1976).

Several other causes for the radiolucent zone have been proposed: infection (Bergström et al. 1974), a bone-resorbing granulomatous reaction to wear products (Brinkmann & Heilmann 1974), metal sensitivity (Evans et al. 1974), micromovements (Salvati et al. 1976) and bone resorption due to stimulation of macrophages attracted by the cement (Freeman et al. 1982). In our material some of these causes are not applicable; there were no infections and no products of wear from polyethylene or metal.

The correlation found between the width of the zone adjacent to cancellous bone and the volume of the cement filling (Figure 4) supports the idea that the radiolucent zone is caused by thermal necrosis. This is further supported by the observation that the zone was wider in concave areas and narrower in the adjacent convex areas (Figure 3), in close accordance with the theoretical prediction of Huiskes (1980).

The remaining theories, i.e. that the zone is caused by local vascular injury at reaming, by micromovements or by macrophages attracted to the bone cement, can explain neither the correlation between the width of the zone and the volume of the filling nor the wider zone in the concavities of the cement. Also, all tumors were metaphyseal, where vascular injury due to surgical trauma is minimal (Rhinelander et al. 1979).

We conclude that the radiolucent zone in our model can only be explained by thermal injury. The sclerotic rim, found in all our cases, is a result of bone regeneration secondary to stress concentration (Sweet & Madewell 1981). The regenerative capacity is, however, also severely impaired by thermal injury (Lundskog 1972, Eriksson & Albrektsson 1984); we found no complete healing of the radiolucent zone (Figure 2).

The thermal injury can be reduced by diminishing the amount of cement (Meyer et al. 1973, Linder 1977), and by heat abductors, i.e.

metal implants (Reckling & Dillon 1977, Huiskes 1980). The more frequent appearance of a zone around the polyethylene acetabular and tibial components compared to the metallic femoral components (Salvati et al. 1976, Ahlberg & Lindén 1977, Beckenbaugh & Ilstrup 1978, Charnley 1979, Jónsson 1981), may thus be explained by heat abduction. The reduced rate of loosening of metal-backed acetabular components compared to non-metal-backed (Harris & White 1982) may also be a consequence of heat abduction, rather than of lowered stress in the bone.

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