INITIATION AND PROPAGATION OF FATIGUE MICROCRACKS FROM A DEFECT IN A CEMENTED TOTAL HIP ARTHROPLASTY

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INTRODUCTION

Voids/defects in cement mantle or at interfaces were reported as a major source of fatigue microcracks (MCs) that occurred in cemented total hip arthroplasty (THA) [1,2]. Presently, it is difficult to monitor the progression of MC activities in real time. In this work, we presented a case study to reveal the initiation and accumulation of MCs from a proximal defect in a cemented THA, using acoustic emission microcrack graphs (AEMG) technique [3].

METHODS

A series of standard hand mixed cemented THA specimens were prepared using Spectron stem, Palacos R cement and Sawbone femurs. In X-ray examination, one specimen with a proximal defect of 6.5 × 2.5 × 14.0 mm (in anterior-posterior, medial-lateral and distal-proximal direction, respectively) was identified (Fig. 1a) and selected to study the influence of the defect on MCs activities. The specimen was subjected to 5 million cycles of fatigue loading (267/2670 N at 2 Hz) on a MTS machine. MC signals were detected by an AE system and their locations were calculated. Six virtual sections (A1 to A6, 5 mm thick each) were selected from the defect area (Fig.1b). MCs that occurred in each section were counted and plotted onto the anterolateral quadrant (Fig.1c) to visualize the initiation and accumulation of MCs (a process called AEMG [3]). After the experiment, the specimen was sectioned at seven locations (A-G in Fig.1a) for SEM inspections.

RESULTS AND DISCUSSION

The AEMG and the number of MCs of virtual sections in the first three hours (Fig.2) indicated that the fatigue damage initiated from the lower lateral end of the defect (section A4) 4 seconds after test started. The damage spread to the surrounding area of the defect (A2-A5) immediately and the number of MCs increased dramatically. Damage propagation (evaluated by number of MCs) decreased as the test proceeded, but with three recover periods which started from the 12th, 18th and 22nd days, respectively (Fig. 3c). Most MCs accumulated around the middle-lower defect (A3-A5). SEM observed a continuous debonding at cement-stem interface next to the defect at physical section A (Fig.3a). A large amount of cement debris could be identified in the debonding (Fig.3b). This indicated the MCs in the middle and final fatigue process was caused mainly by the grinding between damaged interfaces instead of the damage propagation. No MCs located along cement-bone interface and on either side of the defect. Few insignificant MCs were detected in other physical sections.

CONCLUSIONS

AEMG analysis indicated that defect was indeed the key reason of fatigue MCs. These MCs initiated at the lower lateral end of the defect and propagated upward to the surrounding area. Damage propagation was slow down then and interface grinding became the major source of MCs.

REFERENCES

[1] Powers et al., J Arthroplasty, 1998; [2] Topoleski et al., J Biomed Mater Res. 1992; [3] Qi et al., ibid, 2004.

ACKNOWLEDGEMENTS

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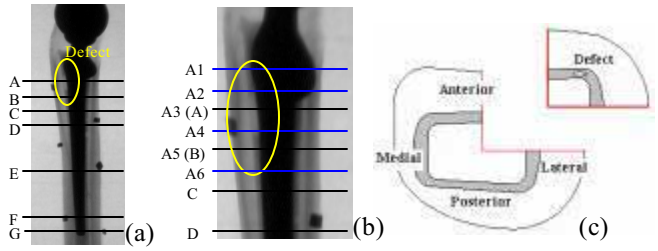

Fig. 1. (a) X-ray of the specimen, sectioned at A to G. (b) Six virtual sections (A1 to A6). (c) Anterolateral quadrant of A3.

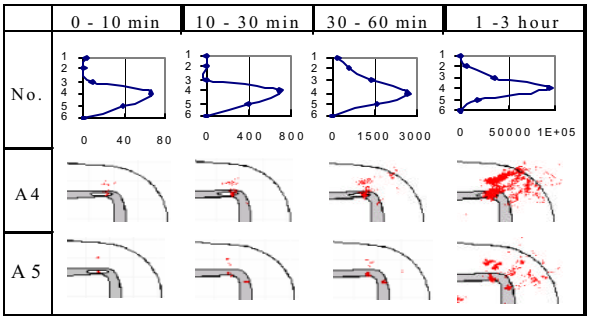

Fig. 2. Number of MC cumulated in A1 to A6, (represented by 1 to 6) and their distribution (A4 to A5) in the first 3 hours.

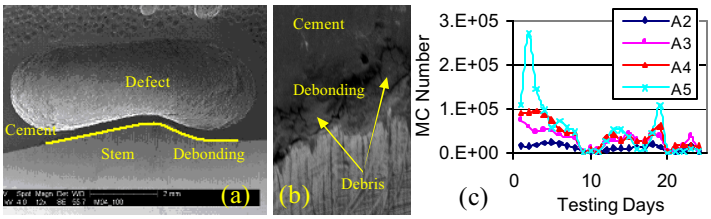

Fig. 3. (a) ESEM image of the defect with debonding marked by yellow curve (12×). (b) Details of the debonding (333×). (c) Number of MCs occurred in A2 to A5.