Surface analysis of failed implant

실패한 임프란트의 표면 분석

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이 논문을 치의학 석사학위 신청 논문으로 제출함.

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김영종의 석사학위 논문을 인준함.

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실패한 임프란트의 표면 분석

김 영 종
지도교수 : 김 수관
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본 연구의 목적은 실패한 임프란트를 광학 현미경과 주사 전자 현미경으로 표면을 분석함으로써 임프란트의 형태와 표면에 따른 실패 요인을 분석하는 데 있다.

1996년부터 2004년까지 실패한 임프란트 26개가 본 연구에 이용되었으며, 이 중 실린더 형태가 8개, 스크류 형태가 18개였다. 실린더 형태 중 표면처리되지 않은 임프란트가 1개였으며, 표면처리 방법에 따라 수산화인회석 코팅이 3개, 티타늄 plasma spray 표면이 4개였다. 나사 형태 중에서 산부식처리한 면이 11개, 티타늄 plasma spray 표면이 7개였다. 이 임프란트를 동결건조기에 12시간 이상 동결건조하여 주사 전자현미경으로 관찰하였다. 본 연구에서는 실린더 형태가 스크류 형태보다 더 많은 연조직과 세균침착을 나타내는 경우가 많았다. 수산화인회석 코팅과 티타늄 plasma spray 표면이 산부식처리한 면보다 더 많은 연조직과 세균 침착을 나타내는 경우가 많았으며, 일부 나사형에서 milling의 흔적과 fixture의 미세 파절이 관찰되었다. 향후 실패한 임프란트에 대한 임상적인 요소와 함께 조직학적인 평가가 필요하리라 사료된다.

Introduction

Knowledge of the concept of osseointegration has enhanced the success of dental implants owing to improved understanding of the concept of bone stress and bone response. Longitudinal clinical studies report 10-year successrates of 81-85% for implants in the maxilla and 98-99% for implants in the anterior mandible[1]. In 1989, the main causes of implant failure were the selection of inappropriate patients and the accumulation of residue owing to the use of improper prosthetic restoration materials. Various investigators have reported individual points of view and clinical observations concerning implant failure.

At the present time, the rehabilitation of edentulous patients with implants is an accepted, expanding treatment modality owing to the predictable results obtained with commercial pure titanium implants *ad modum* Brnemark. The integration of titanium implants in bone is partly attributable to the biocompatibility of the surface oxide layer[2].

In a 5-year study, Brnemark reported low bone density as a predisposing factor for implant failure and a 44% failure rate for implants in the maxilla[3]. Studies showed that implant failure was affected by bone loss, gingivitis, and inflammation near the implants and that high levels of anaerobic bacteria, especially spirochetes, were related to failure[3]. From a biomechanical perspective, English reported the failure of an implant supporting a fixed local false tooth[4]. The main causes of failure were related to excessive twisting and occlusion force, and poor oral hygiene.

Currently, the most commonly used implants are cylinder-type and screw-type implants, and their surfaces are treated with hydroxyapatite (HA),

titanium plasma spray (TPS), or acid etching.

The purpose of this study was to analyze the causes of implant failure with respect to implant type and surface treatment by using light and scanning electron microscopy to examine the surfaces of failed implants.

Materials and Methods

This study analyzed 26 implants that failed between 1996 and 2004, including 8 cylinder-type and 18 screw-type implants. The cylinder-type implants included one implant with an untreated surface, three HA-coated implants, and four TPS implants. The screw-type implants included nine dual acid etched implants, seven TPS implants, and two synchro-step implants.

The implants were freeze-dried for 12 h using a lyophilizer (FD5510, Ilshin) and analyzed using field emission scanning electron microscopy (FE-SEM, S-4100, Hitachi) with an AC voltage of 20 kV and a resolution of 1.5 mm.

Results

The adhesion of soft tissue was evident on the HA-coated (Fig 1) and TPS implants (Fig 2), while there was less adhesion of soft tissue on the dual acid-etched type (Fig. 3).

Less tissue and bacteria was accumulated on the screw-type implants than on the cylinder-type implants (Fig 4). With the screw-type implants, evidence of milling during manufacture and microfractures in the implant itself were detected (Fig 5).
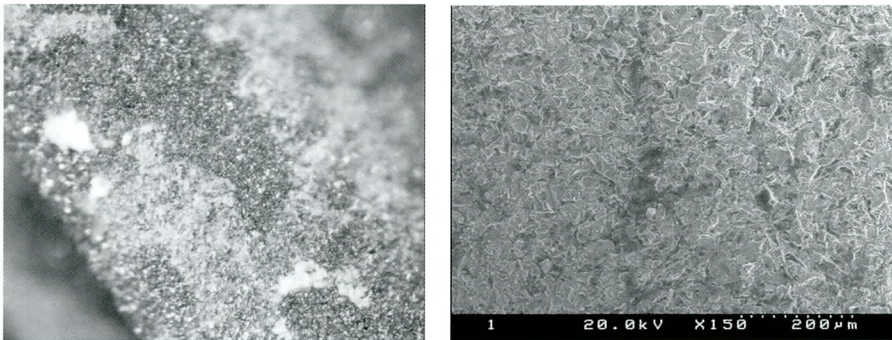

Fig 1. cylinder-type implant, HA-coated surface (3i), white color is indicated the tissue or coating
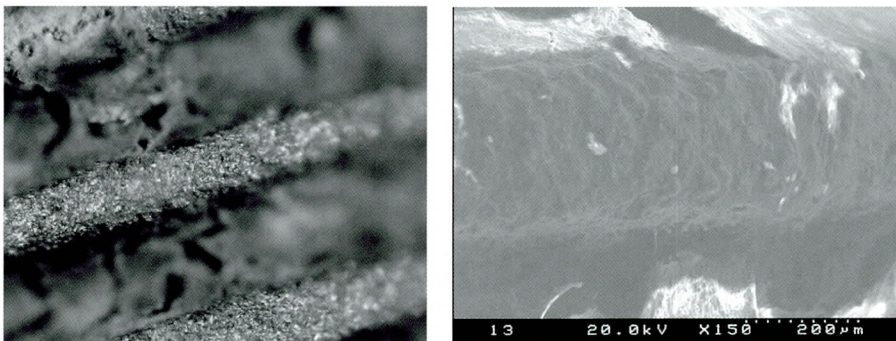

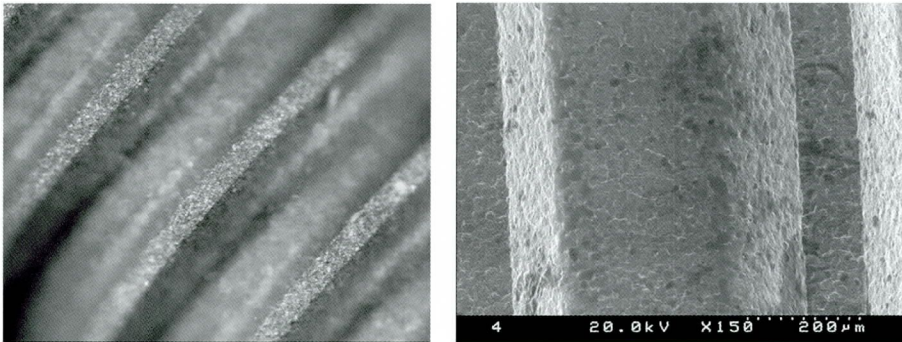Fig 2. screw-type implant, TPS coated surface (bicon), bacteria was eident

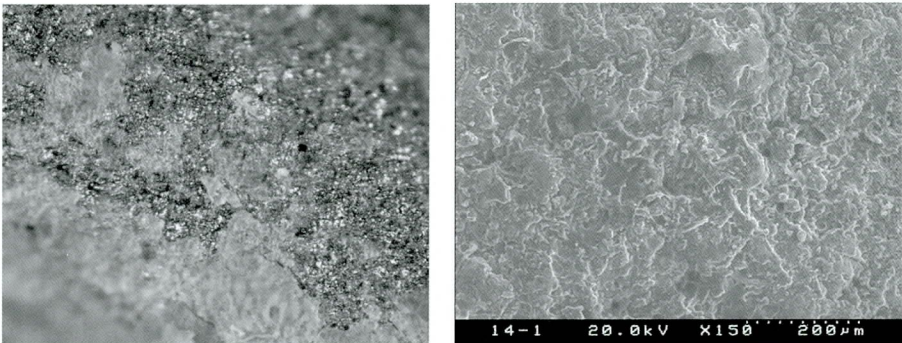Fig 3. screw-type implant, dual acid etching surface (3i), relatively clean surface

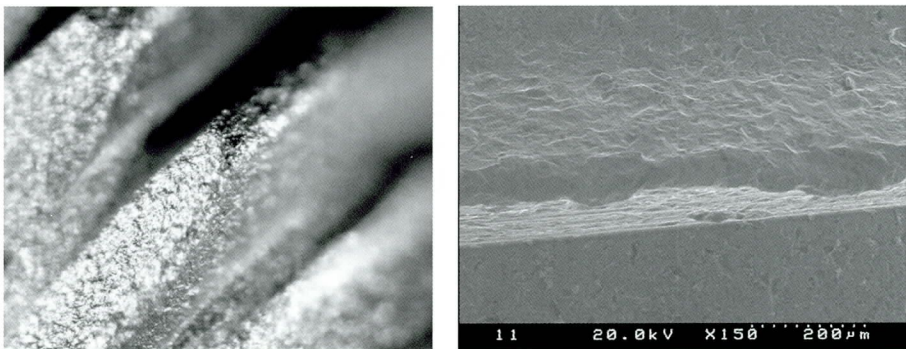Fig 4. cylinder-type implant, TPS coated surface, adhesion of soft tissue was evident

Fig 5. screw-type implant, acid etching surface (Frialit-2), evidence of milling

Discussion

Osseointegration was originally defined as the direct integration between the implant surface and viable bone observable by light microscopy. Recently, osseointegration was re-defined as a "close pattern with well-differentiated, regenerated tissues *in vivo* that can perform normal and clinical functions continuously without rejection between the well-controlled artificial structures, and the structural and functional symbiosis continue by the so-called symbiosis method"[5].

Branemark states that osseointegration occurs only under perfectly stable conditions[6]. Surgical trauma, no matter how minimal, invariably produces a necrotic border zone immediately adjacent to the newly placed implant. Minor movements may inhibit osteogenesis[7]. If bone healing is not allowed to remodel in an environment free of movement and bacterial invasion, then osseointegration will fail, and connective tissue encapsulation will occur[8].

The minimum condition for osseointegration is the absence of irreversible symptoms despite continuous masticatory pressure or movement in the oral cavity; the annual resorption rate of nearby alveolar bone must be less than 0.2 mm; there must be no radiolucent areas near the implants; and more than 80% must be maintained for 10 years[9].

With severely reduced osseointegration and bone loss extending to the apical third of the implant or to the apical vent hole, the possibility of normal recovery is low, and the removal of the implant should be considered[10]. In addition, a mobile implant is referred to clinically as a

failed implant, and removal must be considered[10]. The indication for implant removal owing to a poor outcome is bone loss of more than half the length of the implant that progresses to the vent hole area of the implant or that progresses rapidly within one year of the prosthesis load, and is unresponsive to treatment[10].

If the buccal, lingual, and cortical bone is maintained after implant removal, the bone defect usually heals in a manner similar to the healing of the odontectomy defect. If the defect is large after implant removal, it may be desirable to fill the defect with bone transplant material[10]. Re-implantation may be performed immediately after implant removal or after a treatment period[10].

After implant removal, inflammatory granulation tissues in the defect area and on the surface of the neighboring bone should be removed thoroughly with a curette, and a slightly larger implant may be re-implanted. As it is not feasible to assess the inflammation on the bone surface or the degree of necrosis, the prognosis of immediate re-implantation cannot be predicted. In delayed re-implantation, the soft tissue is allowed to heal completely, and new bone forms in the defect within 1 or 2 months. Then, a slightly larger implant may be used or additional implants may be placed in the nearby healthy bone tissue[10].

To reduce the implant failure rate, it is necessary to select appropriate patients and to use the appropriate treatment plan and surgical procedure, with suitable prosthetic materials and continuous hygienic management[9].

There is little doubt that the biomaterials used in long-term implants should be carefully considered to minimize adverse body reactions both locally and systemically[11].

Surface analysis investigations of failed implants have the advantage of

not causing additional patient discomfort, unlike histological studies, which require the retrieval of an adequate amount of tissue to obtain useful information. In addition, it is easier to examine failed implants surrounded by a soft-tissue capsule than failed implants embedded in plastic or implants successfully integrated in bone, because the analysis is hampered by the plastic or tissue residues[2,12].

The cellular changes are influenced by the physical characteristics and surface pattern of the implant[13]. Predeckie et al.[14] reported that the dynamics of bone healing are dependent on the attachment of cells to the implant surface. Irregularities on the implant surface must form an ideal angle with the surface. Carlsson et al.[15] reported that more force was required to remove an implant with a rough surface 6 months after implantation than to remove an implant with a smooth surface.

Buser et al.[16] investigated the direct bone-implant contact rate using implants with different surfaces, including sandblasted, HA-coated, TPS, and acid-etched surfaces. Of these, the highest rate of bone-implant contact was seen with the sandblasted and acid-etched surfaces.

In experiments using rabbits, Cordioli et al.[17] found that the mean percentage of direct implant-bone contact was higher on acid-etched surfaces (72.42%) than on TPS (56.80%) or smooth surfaces (54.80%). Lazzara et al.[18] obtained similar results in a histomorphological study of humans.

Parr et al.[19] reported that implant failure resulted from tissue damage caused by implant drilling and circulatory impedance. The implantation in an incompletely healed extraction area may cause failure, as the tissue is readily damaged by drilling and impaired circulation occurs readily. The extraction area must be managed carefully during the healing period and

before implantation. Damage during the procedure must be minimized, as excessive damage of the adjacent tissues induces the formation of unwanted tissue fibrosis during healing.

The advantages of an HA-coated implant are faster bone fit, the absence of a fibrosis tissue layer, stronger implant-bone adhesion, higher resistance to surgery, and reduced ion release[20]. A long-term study suggested that an HA coating does not increase long-term bone-implant integration. Therefore, the advantage of a HA coating may be limited to the initial healing period after surgery.

Hayashi et al.[21] reported that bead-coated porous implants had an interface shear strength two- or three-fold that of an HA-coated layer under direct shear loading. Hong et al.[22] found that tensile failure occurred between the HA particles within the bulk, rather than at the HA-bone interface, by 4 weeks after implantation. Although there was enhanced bone ingrowth with HA-coated implants histologically, with little intervening fibrous tissue, it is doubtful that HA-coating alone can ensure the long-term mechanical stability of the implant. Chae et al.[23] suggested that enhanced bone apposition does not imply greater bonding strength.

In mice, Sennerby[24] found that the response of soft tissues injured by a biologically contaminated titanium-covered screw and abutment was comparable to that of clean soft tissuesin a control group. Furthermore, in a experimental study of rabbit tibiae, Ivanoff et al.[25] reported the importance of avoiding the contamination of the implant surface during surgery.

In 1995, Balshi et al.[26] reported microfractures in eight of 4,045 implants, and these were related to excessive bending movements. In the same year, Ranger et al.[27] reported microfractures in 39 of 1,000 implants. In their

experiments, bone resorption near the implants and implant fracture were related to excessive bending movements. In many patients, bone resorption can be detected near the implant before a microfracture of the implant can be detected, especially in molar implants. Pericoronal bone resorption causes more bending stress on the implants owing to the loss of woven bone. Furthermore, such bone resorption extends to the end of the abutment screw and weakens the resistance to bending in this area.

We found that cylinder-type implants resulted in the deposition of more soft tissue and cells than did screw-type implants. HA-coated and TPS-type implants resulted in the deposition of more soft tissue and cells than did acid-etched implants. In some screw-type implants, traces of milling and microfractures were detected.

In the future, the evaluation of clinical factors together with a histological assessment of the failed implants may be required.

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