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Case Report

Selective Explantation in Advanced Peri-Implantitis: Clinical Healing, Exploratory Inflammatory Profiling, and SEM/EDS Analysis of One Retrieved Implant—A Case Report

Marzena Dominiak [1], Jacek Matys [1,*], Paweł Kubasiewicz-Ross [1], Barbara Sterczała [1], Artur Pitułaj [1], Alicja Baranowska [1], Eduard Valmaseda Castellón [2], Ricardo Castro Alves [3] and Victor Geller [1]

[1] Dental Surgery Department, Wrocław Medical University, Krakowska 26, 50-425 Wrocław, Poland; marzena.dominiak@umw.edu.pl (M.D.); pawel.kubasiewicz-ross@umw.edu.pl (P.K.-R.); barbara.sterczala@umw.edu.pl (B.S.); artur.pitulaj@umw.edu.pl (A.P.); research@victorgeller.com (V.G.)
[2] Department of Dentistry, Faculty of Medicine and Health Sciences, University of Barcelona, 08907 Barcelona, Spain; eduardvalmaseda@ub.edu
[3] Clinical Research Unit (CRU), CiiEM, Egas Moniz—Cooperativa de Ensino Superior, Caparica, 2829-511 Almada, Portugal; ralves@egasmoniz.edu.pt
* Correspondence: jacek.matys@umw.edu.pl

Abstract

Background: This case report documents selective explantation in advanced peri-implantitis, early clinical healing, and exploratory blood and retrieval observations. **Methods:** A 71-year-old woman with hypertension had radiographic peri-implant bone defects around six mandibular implants. Implants 35 and 42 had evidence of suppuration, probing depths up to 8 mm, clinical mobility, and bone loss exceeding 66% at one or more surfaces, including full-length mesial defects, and were removed. Treatment combined professional hygiene and mechanical debridement, explantation, curettage, adjunctive Er:YAG irradiation of the post-explantation defect walls, chlorhexidine, systemic amoxicillin, and supportive care. The first blood sample was collected on the day of surgery, 2 weeks after the initial hygiene consultation. A second sample was obtained at 6 weeks, while four implants with radiographic defects remained in situ. **Results:** Both explantation sites showed uncomplicated soft-tissue healing over 2 months; no osseous healing outcome was assessed. At 4 months, the retained implants showed no bleeding on probing, suppuration, or clinically detectable mobility, without establishing long-term disease control. The blood findings were descriptive observations from one patient and showed no consistent downward inflammatory pattern. Ex vivo scanning electron microscopy with energy-dispersive X-ray spectroscopy (SEM/EDS) was performed on implant 35. **Conclusions:** SEM/EDS analysis of the retrieved implant revealed surface alterations consistent with mechanical wear and localized variations in elemental composition, providing additional insight into the structural and chemical characteristics of an implant affected by advanced peri-implantitis. Blood-derived inflammatory markers may offer complementary information about the patient's general systemic inflammatory status and contribute to a more comprehensive clinical assessment. However, their specific diagnostic and prognostic value in peri-implantitis remains to be established through larger prospective clinical studies.

Keywords: advanced peri-implantitis; case report; implant explantation; SEM/EDS; titanium surface; systemic inflammatory markers; short-term healing

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1. Introduction

Peri-implantitis is a plaque-associated pathological condition affecting tissues around dental implants and is characterized by inflammation of the peri-implant mucosa and progressive loss of supporting bone beyond expected physiological remodeling [1]. Diagnosis combines bleeding and/or suppuration on probing, increased peri-implant probing depths, and radiographic bone loss [1–3]. Local clinical and radiographic measurements remain the basis for treatment planning and follow-up.

Therapy involves biofilm control, mechanical instrumentation, re-evaluation, and, when indicated, surgical access or reconstructive treatment [3–6]. In advanced cases, extensive loss of support combined with true implant mobility may make implant-preserving surgery clinically inappropriate; resective or regenerative treatment cannot be assumed to restore predictable support to a mobile implant [3,7–9]. Explantation decisions must account for residual osseointegration, defect morphology, anatomical risk, and prosthetic requirements rather than a single bone-loss percentage [3,7–10]. Available removal methods include reverse-torque devices, trephine burs, surgical drills, piezosurgery, and forceps removal when severe loss of osseointegration and mobility permit an atraumatic approach [7,9–11].

Evidence gaps remain regarding adjunctive management of post-explantation defects and the interpretation of retrieved implant surfaces. Er:YAG irradiation has been investigated for tissue ablation and peri-implant debridement, but heterogeneous protocols do not establish an independent treatment benefit or validate a specific post-explantation regimen [4,5,12–20]. Systemic inflammatory profiling is exploratory: CBC variables and cytokines are nonspecific, associations with peri-implantitis remain incompletely understood, and CBC-derived indices are not validated diagnostic or treatment-response measures for this disease [21–24].

The primary aim was to document clinical decision-making for selective explantation of two severely affected implants and short-term soft-tissue healing within a multimodal treatment pathway. Secondary aims were qualitative ex vivo SEM/EDS characterization of implant 35 and descriptive blood profiling before surgery and at 6 weeks. The clinical, systemic, and material-level findings were considered complementary observations without inferring a causal chain or an independent effect of Er:YAG irradiation.

2. Case Presentation

2.1. Patient Information

A 71-year-old female patient with advanced active peri-implantitis was treated within the PERI-EDU project (Integrating Peri-Implantitis Research into Higher Education Curriculum, No. 2024-1-PL01-KA220-HED-000251208). The patient was managed within the broader PERI-EDU project framework but was not included in the primary analytical cohort. The clinical procedures and data collection were performed in accordance with the study protocol approved by the Bioethics Committee at Wroclaw Medical University, Poland (approval No. KB 601/2024, dated 20 December 2024). The case report complies with the CARE guideline.

The patient reported hypertension and denied other diagnosed systemic diseases, allergies, smoking or other addictions, and dietary supplement use. Her current medications included Bibloc (bisoprolol fumarate) at 2.5 mg once daily and Betaserc (betahistine dihydrochloride) at 24 mg once daily. She presented with complete dentition in the maxilla and a cemented implant-supported prosthetic restoration in the mandible; according to the patient's history, the mandibular teeth had been lost due to caries. The six mandibular implants were located at sites 37, 35, 32, 42, 44, and 46. Radiographic peri-implant bone defects were present around all six implants, with the most advanced loss at sites 35 and

42. At these sites, mesial bone loss extended along the full implant length and bone loss exceeded 66% at one or more surfaces. Both implants showed suppuration and clinical mobility (grade 1 at site 35 and grade 2 at site 42). The designation "hopeless prognosis" reflects the authors' integrated clinical judgment based on the concurrent presence of advanced bone loss, suppuration, increased probing depth, and mobility; it is not presented as a universally standardized peri-implantitis category. Both implants were reported to be 4.2 mm in diameter and 8.0 mm in length. The manufacturer and original surface characteristics could not be verified because an implant card was unavailable (Figure 1).

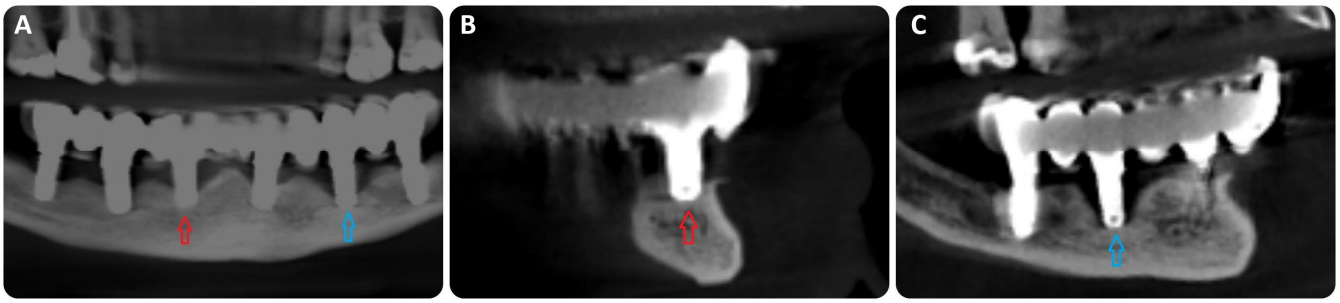


Figure 1. Baseline radiographic assessment on the day of treatment. (**A**) Panoramic radiograph showing the mandibular implants; implant 42 is marked with a red arrow and implant 35 is marked with a blue arrow. (**B**) Baseline cross-sectional cone-beam computed tomography (CBCT) image of implant 42 showing severe peri-implant bone loss. (**C**) Baseline cross-sectional CBCT image of implant 35 showing advanced peri-implant bone loss. The images were used for diagnosis and treatment planning; no quantitative regeneration outcome was derived.

2.2. Main Concerns and Clinical Findings

Advanced active peri-implantitis at sites 35 and 42 was diagnosed from the combined clinical and radiographic findings. The patient reported pain at the two affected implant sites. Both sites showed peri-implant mucosal swelling, bleeding and suppuration on probing, increased probing depths, and implant mobility. Probing depths were measured with a UNC-15 periodontal probe (Deppeler SA, Rolle, Switzerland). At implant 42, probing depths were 5 mm lingually, 6 mm buccally, 8 mm mesially, and 6 mm distally. At implant 35, they were 5 mm lingually, 6 mm buccally, 8 mm mesially, and 5 mm distally. Mobility of the implant-supported prosthetic restoration also suggested a prosthetic or biomechanical contribution.

At baseline, the oral hygiene index (OHI) was 2, and the approximal plaque index (API) was 15%. The peri-implant soft-tissue phenotype differed between the two sites. At implant 42, both the height of keratinized tissue (HKT) and width of keratinized tissue (WKT) were 5 mm. In contrast, at implant 35, HKT and WKT were both 0 mm after Lugol's staining, and the mucosal phenotype was classified as thin. Palpation confirmed suppuration at both implants 35 and 42.

2.3. Exploratory Blood-Based Inflammatory Profiling

Peripheral venous blood was collected on the day of surgery and 6 weeks after explantation. The first collection is termed the pre-surgical/post-initial-debridement sample because professional oral hygiene treatment and mechanical debridement were performed 2 weeks earlier; it is not an untreated baseline. The 6-week follow-up and blood-sampling visit was specified in the study protocol. This assessment describes an early postoperative observation beyond initial soft-tissue wound closure [25], not a validated interval for systemic biomarker response or an assessment of acute postoperative inflammation. Four implants with radiographic defects remained in situ throughout the blood-sampling period. The comparison therefore represents partially treated peri-implant disease before

surgery versus an early observation after selective explantation within ongoing multimodal care, not disease elimination. Absolute CBC values, hs-CRP, and measured cytokines are emphasized; the six derived indices are supplementary exploratory summaries. Blood samples were collected in the morning after fasting, and laboratory analyses were performed through Diagnostyka S.A. (Wrocław, Poland). For serum analysis, blood was allowed to clot for at least 30 min and centrifuged at 1800× *g* for 10 min at 15–25 °C. The separated serum was stored frozen for up to 4 months. Serum samples from both visits were analyzed in the same analytical batch, using the same assay kit type for each analyte. However, reagent lot numbers were unavailable. Complete blood count (CBC) analysis was performed on whole blood and did not involve the serum preparation or freezing procedures. The exact collection and processing intervals, cytokine sample storage temperature, number of freeze–thaw cycles, and specific cytokine assay platform, detection and quantification limits, and analytical precision could not be verified.

The selected CBC-derived indices were NLR, PLR, MLR, SII, SIRI, and AISI/PIV. They were calculated from routine absolute blood-cell counts and retained as descriptive cellular summaries in Supplementary Table S1. They share the same underlying neutrophil, lymphocyte, monocyte, and platelet counts and are mathematically interdependent. No index was treated as a validated peri-implantitis biomarker, and no diagnostic threshold or percentage-change criterion was applied.

Absolute concentrations of measured serum cytokines were reported together with the laboratory reference intervals in Supplementary Table S2. The laboratory source identified one analyte only as "Interleukin 1"; because the assay isoform could not be verified, it is reported as IL-1 without assigning a beta isoform. The laboratory reported IL-12p70 as 1.32 pg/mL in the first sample and <1.9 pg/mL at 6 weeks. The second result was treated as left-censored below the laboratory reporting threshold, without assigning an exact concentration. Because the first reported value lies below the second visit's reporting threshold, reliable quantification of that lower value and analytical comparability between visits could not be verified. Both results were retained as reported for transparency, but neither a directional comparison nor a quantitative change estimate was made.

NLR, PLR, MLR, SII, SIRI, and AISI/PIV were calculated as follows: NLR = neutrophil count/lymphocyte count; PLR = platelet count/lymphocyte count; MLR = monocyte count/lymphocyte count; SII = platelet count × neutrophil count/lymphocyte count; SIRI = neutrophil count × monocyte count/lymphocyte count; and AISI/PIV = neutrophil count × monocyte count × platelet count/lymphocyte count. Because this was a single-case descriptive analysis, no inferential statistical testing was performed.

2.4. Therapeutic Intervention

Two weeks before the surgical appointment, the patient underwent professional oral hygiene procedures. Accessible implant and prosthetic surfaces were mechanically debrided with a scaler fitted with a composite tip. On the day of surgery, after removal of the mandibular implant-supported restoration, the oral cavity was disinfected with 0.2% chlorhexidine digluconate (EludrilPerio 0.2%, Pierre Fabre Médicament Production, Gien, France). Local infiltration anesthesia was administered using 4% articaine with epinephrine 1:200,000 (Septanest, Septodont, Saint-Maur-des-Fossés, France), total volume 5.1 mL, and envelope flaps were elevated at sites 35 and 42. Implants 35 and 42 were selected for explantation because of >66% bone loss at one or more surfaces, a full-length mesial defect, bleeding and suppuration on probing, increased probing depths, and mobility. The concurrent mobility and extensive loss of support made predictable retention after resective or regenerative surgery unlikely in the treating clinicians' assessment [3,7–9]. The remaining four implants had no clinically detectable mobility (grade 0), less extensive

bone loss, and generally shallower probing depths, although implant 44 had a 7-mm mesial pocket. They were retained to preserve prosthetic support, with professional oral hygiene scheduled every 4 months and close monitoring; their baseline documentation is summarized in Table 1. Inflamed soft tissue was removed, and implants 35 and 42 were extracted with dental forceps because their mobility permitted an atraumatic approach.

Table 1. Available baseline clinical and radiographic findings at the six mandibular implants.

Implant Site	PD L/B/M/D (mm)	BOP/Suppuration	Mobility	Radiographic Bone Loss	Management
35	5/6/8/5	Present/present	Grade 1	>66% at ≥1 surface; full-length mesial defect	Explantation
42	5/6/8/6	Present/present	Grade 2	>66% at ≥1 surface; full-length mesial defect	Explantation
37	4/3/3/3	Present/absent	Grade 0	10% at deepest measured point	Retained; professional oral hygiene every 4 months
32	3/3/3/3	Absent/absent	Grade 0	30% at deepest measured point	Retained; professional oral hygiene every 4 months
44	2/6/7/4	Present/absent	Grade 0	35% at deepest measured point	Retained; professional oral hygiene every 4 months
46	2/2/2/2	Absent/absent	Grade 0	30% at deepest measured point	Retained; professional oral hygiene every 4 months

PD, probing depth; L/B/M/D, lingual/buccal/mesial/distal; BOP, bleeding on probing; grade 0, no clinically detectable mobility. BOP was present at retained implants 37 and 44 and absent at implants 32 and 46; suppuration was absent at all four retained implants at baseline. Bone-loss percentages for the retained implants describe the greatest loss at the deepest measured point. The four retained implants had no recorded BOP, suppuration, or clinically detectable mobility at 4 months; probing depths and site-specific plaque findings at that visit were not available.

Flap surgery, explantation, curettage, and laser application were limited to sites 35 and 42. At implants 37, 32, 44, and 46, the only treatment before explantation was the professional oral hygiene and mechanical cleaning procedures performed 2 weeks earlier. No additional site-specific instrumentation, surgery, or laser treatment was performed at these four implants during the explantation appointment. They subsequently remained under supportive peri-implant care.

Residual granulation tissue was mechanically curetted. The entire accessible bony wall of each post-explantation defect was then irradiated with an Er:YAG laser (LightTouch, Light Instruments, Yokneam, Israel) at 150 mJ per pulse, 20 Hz, and 3.0 W, using a 1-mm tip in non-contact mode at an approximate 1-mm working distance. Irradiation was applied for 60 s per defect with continuous water cooling at 40 mL/min, while the tip was moved continuously over the accessible defect walls. No implant surface was irradiated during this post-explantation stage; accordingly, the procedure is described as adjunctive defect debridement rather than implant-surface decontamination. The nominal fluence at the tip aperture was approximately 19.1 J/cm^2 per pulse. The Er-YAG laser was used after implant removal to clean the surgical site and remove residual tissue from the exposed bony defect walls under water cooling. Although previous studies support the potential use of Er lasers in peri-implant procedures [13–16], the specific protocol applied in this case (150 mJ, 20 Hz, 60 s) has not yet been validated for post-explantation defects. The EFP S3 guideline does not suggest Er-YAG laser treatment for implant-surface decontamination during peri-implantitis surgery [3]. However, in the present case, the laser was applied to the bony defect walls after explantation, rather than to the implant surfaces.

After final inspection and irrigation, the defects were protected with an absorbable hemostatic sponge and closed with 5-0 resorbable sutures (Novosyn®, B. Braun Surgical

S.A., Rubí, Spain) and 5-0 monofilament nylon sutures (Dafilon®, B. Braun Surgical S.A., Rubí, Spain). Postoperative care included amoxicillin 1.0 g every 12 h for 7 days, 0.2% chlorhexidine digluconate three times daily for 2 weeks, oral-hygiene instructions, and nimesulide 100 mg every 12 h as needed for pain for 1–2 days. Chlorhexidine was used as temporary adjunctive plaque control during early wound healing, and nimesulide was prescribed for short-term symptom relief. The decision to prescribe amoxicillin was based on the treating clinician's judgment and experience in the context of painful, suppurating sites and same-session flap elevation and explantation of two implants (Table 2, Figure 2).

Table 2. CARE-style timeline of the principal interventions and assessments.

Time Point	Clinical/Intervention Event	Laboratory/Imaging Event
−2 weeks	Professional hygiene and mechanical debridement	—
Day 0	Explantation of implants 35 and 42; curettage; Er:YAG irradiation; wound closure	Pre-surgical/post-initial-debridement blood sample
10 days	Suture removal; early soft-tissue review	—
6 weeks	—	Second blood sample
2 months	Healing review of explantation sites	Clinical photographs
4 months	Assessment of four retained implants; supportive-care plan	—

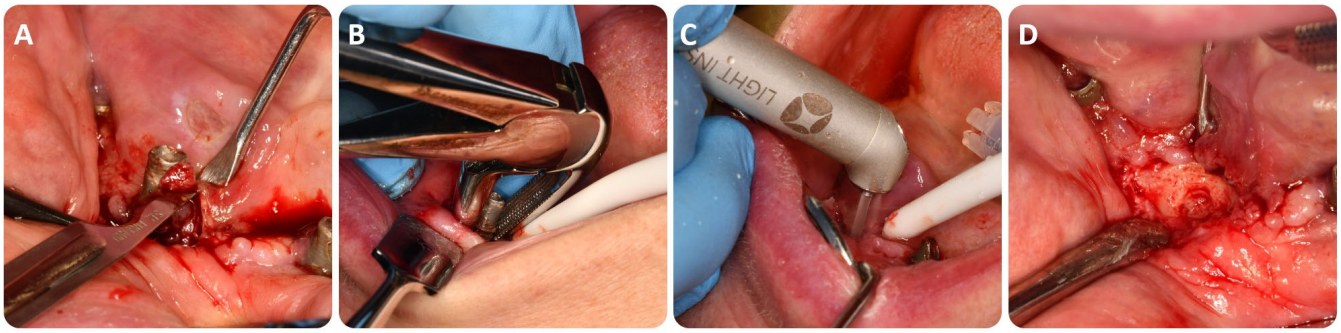


Figure 2. Intraoperative stages. (**A**) Removal of inflamed peri-implant soft tissue; (**B**) forceps removal of an affected implant; (**C**) adjunctive Er:YAG irradiation of the accessible bony walls after explantation and mechanical curettage; and (**D**) immediate postoperative appearance.

2.5. Ex Vivo SEM/EDS Assessment of Implant 35

Only implant 35 underwent SEM/EDS; implant 42 was also removed at the same surgical appointment but was not analyzed. After explantation, implant 35 was placed in a plastic vial containing 5% formaldehyde solution, stored at room temperature (approximately 22 °C), and sent for SEM/EDS analysis. Examination was performed within 2 weeks of explantation. The retrieved implant was assigned laboratory code WR10. No additional cleaning or mechanical surface modification was performed after explantation and before SEM/EDS examination.

Surface morphology was evaluated using a Phenom Pro X scanning electron microscope equipped with an energy-dispersive X-ray spectroscopy detector. Five surface regions were purposively selected by an experienced SEM/EDS analyst based on morphological features considered most suggestive of surface deterioration, including possible corrosion, in the context of peri-implantitis. Areas showing obvious mechanical impressions or scratches considered compatible with forceps contact were excluded. The selected regions were examined at four magnifications. The exact correspondence between the analyzed regions and implant surfaces exposed in vivo before explantation could not be verified

from the available documentation. Representative SEM micrographs, point spectra, and elemental maps were assessed qualitatively; no quantitative elemental mass fractions or control implant were available.

2.6. Clinical Follow-Up

At 10 days, the treated explantation sites showed satisfactory soft-tissue healing without pain, dehiscence, suppuration, excessive bleeding, or another site-specific complication. At 2 months (22 May 2026), both sites remained clinically stable without recurrent local infection or delayed healing. A separate prosthesis-related mucosal ulcer near the orifice of the left sublingual gland was attributed clinically to mechanical irritation and was not located at either explantation site. At 4 months, the four retained implants were evaluated separately: no bleeding on probing, suppuration, or clinically detectable mobility was recorded. They remained under supportive peri-implant care, with professional cleaning scheduled every 4 months during the first year and subsequent intervals adjusted to risk and clinical findings. Site-specific probing depths and plaque findings for the retained implants at 4 months were not available for this report. After explantation, the patient was referred for further prosthetic treatment, with a planned mandibular overdenture on the four retained implants (37, 32, 44, and 46). The supportive-care interval follows the EFP recommendation for visits every 3–4 months during the first year, with subsequent visits tailored to risk [3] (Figure 3).

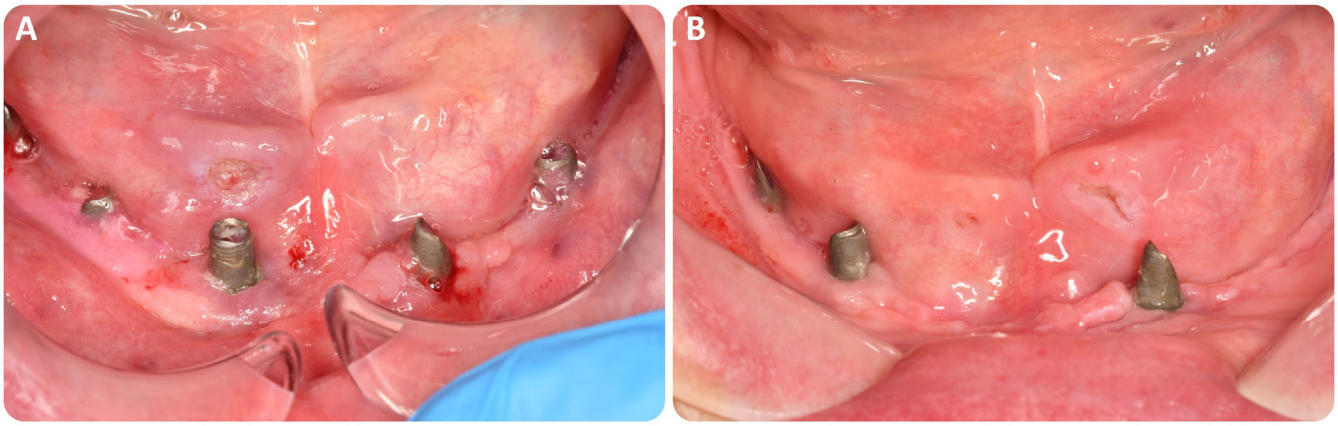


Figure 3. Clinical appearance at the predefined time points. (**A**) Baseline view on the day of surgery, showing inflamed peri-implant tissues around implants 35 and 42 before explantation, and (**B**) 8-week view, showing satisfactory soft-tissue healing at the two explantation sites. The separate prosthesis-related ulcer was outside the field shown.

2.7. SEM/EDS Findings

SEM showed numerous microcracks, linear surface markings, and adherent deposits on the WR10 surface. EDS spectra and elemental maps identified a titanium-dominant substrate with oxygen and localized carbon- and silicon-rich regions; residual bone-like deposits were also described in the laboratory report. These features were compatible with surface deterioration, including possible wear or corrosion, but did not establish either mechanism. Their timing and origin could not be determined, and contributions from explantation or specimen handling could not be excluded. The localized carbon and silicon signals were nonspecific and did not identify a particular abrasive, manufacturing process, or previous maintenance procedure (Figure 4).

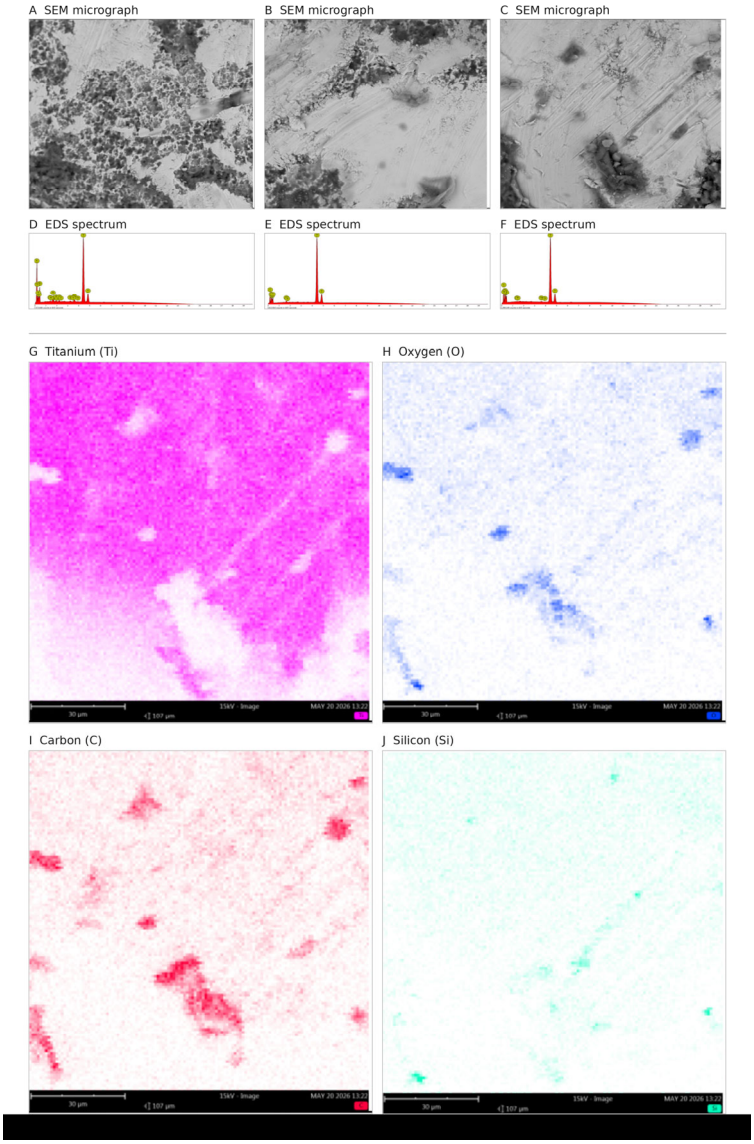


Figure 4. Ex vivo SEM/EDS assessment of explanted implant 35 (laboratory code WR10). Representative SEM micrographs and EDX spectra are shown above elemental maps for titanium, oxygen, carbon, and silicon. The surface displayed microcracks, linear surface markings, and localized carbon- and silicon-rich areas. Scale bars and acquisition metadata are embedded in the original laboratory images. The green dots represent the measured EDS spectral data points, while the red line indicates the fitted spectrum.

2.8. Exploratory Circulating Inflammatory Findings

Absolute CBC measurements at the pre-surgical/post-initial-debridement and 6-week visits included: white blood cells, 6.98 and 7.43 $\times$ 10^3/µL; neutrophils, 3.41 and 4.04 $\times$ 10^3/µL; lymphocytes, 2.71 and 2.55 $\times$ 10^3/µL; monocytes, 0.57 and 0.65 $\times$ 10^3/µL; and platelets, 214 and 221 $\times$ 10^3/µL. hs-CRP was 1.11 and 1.39 mg/L. These values remained within the corresponding laboratory reference intervals (Supplementary Table S2). All six derived indices increased numerically (Supplementary Table S1). Absolute cytokine concentrations also changed: TNF-α 1.16 to 2.05 pg/mL, IL-6 1.85 to 3.49 pg/mL, IL-8 2.73 to 5.49 pg/mL, IL-1 1.49 to 1.83 pg/mL, and IL-10 1.47 to 2.30 pg/mL. TNF-α, IL-6, IL-8, and IL-10 remained within the laboratory reference intervals; IL-1 was above the reported interval at both time points. IL-12p70 was 1.32 pg/mL at the first visit and <1.9 pg/mL at 6 weeks. The latter censored result does not establish the direction or magnitude of change.

No consistent downward pattern was observed across the selected circulating inflammatory measures at 6 weeks. This laboratory outcome is reported separately from the uncomplicated healing of the two explantation sites and from the 4-month clinical status of the four retained implants. Both blood samples were obtained within a partially treated disease course: the first followed initial debridement, and four implants with radiographic defects remained in situ at the second sampling period. The numerical differences cannot be attributed specifically to selective explantation or interpreted as evidence of biological improvement or deterioration.

Supplementary Table S1 reports the six mathematically related CBC-derived indices, formulas, and calculation units. Supplementary Table S2 provides raw laboratory values, units, and reference intervals. Its metabolic, lipid, thyroid, nutritional, and other ancillary measurements are explicitly labeled as contextual data and were not analyzed as peri-implantitis treatment outcomes. Percentage changes and fasting-dependent composite metabolic or endocrine indices were not calculated.

3. Discussion

The principal contribution of this case report is the clinical reasoning behind selective explantation of implants 35 and 42, supported by early follow-up and qualitative retrieval observations. Both removed implants showed extensive bone loss, suppuration, deep probing depths, and mobility; these findings made predictable retention clinically unlikely [3,7–9]. The four other implants had grade 0 mobility and less extensive radiographic bone loss (10–35% at the deepest measured point), supporting retention for prosthetic rehabilitation under surveillance [26]. Baseline probing depths were 2–4 mm at implants 37, 32, and 46, whereas implant 44 had buccal and mesial depths of 6 and 7 mm, respectively (Table 1). Baseline BOP was present at implants 37 and 44 and absent at implants 32 and 46, with no suppuration at any of the four retained implants. The deeper pocket and BOP at implant 44 warrant continued site-specific monitoring.

Both explantation sites showed uncomplicated early soft-tissue healing. Implant removal, flap surgery, removal of inflamed tissue, mechanical curettage, adjunctive Er:YAG irradiation of the post-explantation defect walls, wound closure, chlorhexidine, systemic amoxicillin, plaque control, and natural wound repair occurred within the same pathway. No Er:YAG-specific contribution can be isolated from this case. The absence of bleeding, suppuration, and clinically detectable mobility at the retained implants at 4 months is encouraging but does not establish disease stability without serial site-specific probing and radiographic data.

The SEM/EDS examination documented surface alterations on retrieved implant 35. Retrieval analyses provide context for assessing surface deposits and composition [27]. Tribological and corrosion-related processes are possible, unconfirmed mechanisms; SEM/EDS alone cannot establish tribocorrosion, fretting, galvanic corrosion, or crevice corrosion. Although areas with obvious marks considered compatible with forceps contact were excluded, visual selection cannot eliminate all retrieval-related artifacts or establish that the remaining changes precede explantation. The findings therefore cannot be attributed specifically to peri-implant inflammation. The unknown manufacturer, original surface treatment, and implant/abutment/restoration alloy composition prevent reliable separation of manufacturing topography, service-related wear, and damage introduced during retrieval or preparation. Carbon may originate from biological residues, environmental contamination, or sample preparation including formaldehyde fixation, while silicon enrichment cannot be used to identify a specific abrasive or clinical procedure. The literature on titanium particles and an umbrella review of metal-associated lesions describe potential interactions among material release and peri-implant inflammatory, reactive, or hypersensi-

tivity lesions [28,29]. Here, no particle release, tissue deposition, allergy, or metal-associated lesion was demonstrated, and no such mechanism can be assigned to this patient.

Blood-derived inflammatory markers may provide useful complementary information about the patient's general inflammatory status alongside clinical and radiographic assessment of peri-implantitis. Routine complete blood counts and derived inflammatory indices are readily available, while cytokine measurements may offer additional insight into systemic inflammatory activity. In the present case, no consistent reduction in inflammatory markers was observed 6 weeks after explantation, despite uncomplicated local wound healing. This may reflect the complexity of systemic inflammatory responses, particularly as four implants with radiographic bone defects remained in place. Although CBC-derived indices share the same underlying blood cell counts [21–24], their potential value in monitoring inflammatory changes deserves further investigation. Importantly, combining blood-based inflammatory profiling with SEM/EDS analysis may provide a broader perspective on peri-implantitis. Observed implant surface alterations and differences in elemental composition could be investigated alongside local inflammation and circulating inflammatory markers to better understand their possible relationships [30]. Future studies integrating these approaches may help identify whether specific patterns of implant surface damage and peri-implant inflammation are associated with measurable systemic inflammatory responses.

The findings of this case should be considered in light of several limitations. The study involved a single patient, two blood sampling time points, and a 4-month clinical follow-up, without an untreated or non-laser comparison group. The first blood sample was collected after initial mechanical debridement, and four implants with radiographic defects remained in place, potentially influencing circulating inflammatory markers. Age, hypertension, medication use, and natural biological variation may also have affected the laboratory findings. Although blood collection and serum preparation conditions were documented, some cytokine assay characteristics remain unverified, limiting the interpretation of small differences between measurements. The SEM/EDS analysis was limited to one retrieved implant, with no matched control, and focused on selected surface regions. The exact correspondence of the analyzed fields with surfaces exposed in vivo before removal could not be verified, and potential effects of forceps extraction, subsequent handling, and formaldehyde fixation could not be excluded. Consequently, the origin and timing of the observed surface alterations could not be determined. In addition, incomplete longitudinal clinical measurements and a lack of documented outcomes for the planned prosthetic rehabilitation limit assessment of longer-term treatment results. Future prospective studies involving larger patient groups, standardized blood sampling, repeated inflammatory measurements, detailed clinical monitoring, and comparative implant surface analysis are needed to clarify the potential relationships between implant surface alterations, local peri-implant inflammation, and systemic inflammatory responses.

4. Conclusions

This case demonstrates that selective explantation of severely affected implants, combined with multimodal treatment, can achieve uncomplicated early soft-tissue healing. SEM/EDS analysis revealed surface alterations consistent with mechanical wear and localized differences in elemental composition, providing valuable insight into the surface characteristics of an implant affected by advanced peri-implantitis. Blood-derived inflammatory markers, including CBC-based indices and cytokines, may complement clinical and radiographic findings by providing additional information about the patient's systemic inflammatory status. Although their diagnostic and prognostic value in peri-implantitis remains uncertain, integrating laboratory inflammatory profiling with clinical assessment

and implant surface analysis represents a promising direction for future research. Larger prospective studies are needed to clarify the relationships between implant surface alterations, local peri-implant inflammation, and systemic inflammatory responses, and to determine the potential clinical applications of these combined approaches.

Supplementary Materials: The following supporting information can be downloaded at https://www.mdpi.com/article/10.3390/prosthesis8100112/s1. Table S1. Selected CBC-derived inflammatory indices at the pre-surgical/post-initial-debridement sample and 6 weeks; Table S2. Raw laboratory values at the same time points, with units and laboratory reference ranges; ancillary measurements are contextual data, not treatment outcomes.

Author Contributions: Conceptualization, J.M. and P.K.-R.; methodology, J.M. and P.K.-R.; software, J.M.; validation, M.D., J.M. and P.K.-R.; formal analysis, M.D.; investigation, P.K.-R. and A.P.; resources, V.G.; data curation, V.G.; writing—original draft preparation, J.M., A.P., B.S., A.B. and V.G.; writing—review and editing, P.K.-R., E.V.C. and R.C.A.; visualization, J.M.; supervision, M.D.; project administration, J.M.; funding acquisition, M.D. All authors have read and agreed to the published version of the manuscript.

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Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki and approved by the Bioethics Committee at Wroclaw Medical University, Poland (approval No. KB 601/2024, dated 20 December 2024).

Informed Consent Statement: Written informed consent was obtained from the patient for participation in the study and for publication of this case report and any accompanying clinical images.

Data Availability Statement: The pseudonymized source data supporting this case report are available from the corresponding author upon reasonable request subject to the applicable ethical and privacy restrictions. They are not publicly available because they contain individual-level clinical information.

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