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CASE REPORTS

Endo-Periodontal Lesion in a Diabetic Patient: A Case Report

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ABSTRACT
Endodontic infection in Diabetic patients gets an enhanced expression in periradicular tissues. Many histological and microbial studies report this. Nevertheless, clinical aspects of this relationship remain few documented. The present case report illustrates an acute endo-periodontal lesion with a purulent exudate in a 39 -year -old female type I Diabetic patient and highlights successful management of endodontic and periodontal infection in an uncontrolled diabetic patient.
KEYWORDS: Diabetes Mellitus, Endo-periodontal lesion

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INTRODUCTION
Diabetes Mellitus (DM) is a group of disorders affecting the metabolism of carbohydrates, lipids, and proteins, which is mainly characterized by hyperglycemia. Type I Diabetes results from autoimmune destruction of pancreatic –cells with complete loss of insulin secretion; Type 2 Diabetes results from insulin resistance without compensation through additional insulin production [1]. Glycated hemoglobin (HbA1c) is used as a gold standard for mean glycemia and provides an overview of the risk for the development of DM complications. HbA1c levels ≤ 6.5% are considered a goal for optimal glycemic control in diabetes patients [2]. Endodontic infection occurs due to colonization of the tooth pulp space with microorganisms and pulp necrosis. Hence, periradicular tissues undergo inflammatory processes in response to microbial infection of the root canal system to limit the spread of infectious elements [3]. Apical periodontitis is the most studied sequel to endodontic infections, which is viewed as a dynamic encounter between microbial factors and host defenses at this level resulting in local inflammation, resorption of hard tissues, and destruction of periapical structures. Disease management eradicates microbial infection or substantially reduces the microbial load from the root canal and prevents reinfection through endodontic treatment.
Altered tissue metabolism and microangiopathy related to Diabetes Mellitus may lead to ischemia of local tissue, reduced resistance to microbial infection, and reduced tissue repair capacity. The dental pulp of diabetic patients may have limited dental collateral circulation, impaired immune response, and an increased risk of infection or pulp necrosis. [4]
Periodontal disease was recognized as the sixth complication of Diabetes Mellitus by the World Health Organization. Current evidence showed that hyperglycemia leads to an exaggerated immuno-inflammatory response to the periodontal pathogenic bacterial challenge resulting in more rapid and severe periodontal tissue destruction [5]. Nevertheless, the literature on the prognosis, progression, and healing of endodontic infection in diabetic patients are scarce. Some reports stated that DM is associated with a larger size of periapical lesions and a slower healing rate in periradicular tissues after root canal treatment [6], others revealed that diabetics harbor more virulent root canal microbial profiles, which makes diabetic patients more susceptible to more serious endodontic infections, inter-appointment flare-ups are also higher in Diabetic host [7]. From the previous findings, we can assume that DM may be a modulating factor of endodontic infections and that it may compromise the healing process of periradicular tissues. Endo-periodontal lesions are clinical conditions involving

the pulp and periodontal tissues related to the migration of microorganisms and inflammatory mediators between the root canal and the periodontium. The endo-periodontal lesion in a periodontitis patient is characterized by the presence of deep periodontal pockets, altered response pulp vitality tests, purulent exudate, and tooth mobility [8]. Numerous reports supported-out the adverse effect of periodontal infection on glycemic control in diabetic patients [9]. It was stated that chronic inflammation in periradicular tissues could promote an increase in over insulin resistance, altering the metabolic control in diabetic patients [10]. Nevertheless, the interaction between endodontic infection and diabetic state was few documented. Cintra et al. [11] investigated the effect of apical periodontitis on blood glucose concentrations and HbA1c levels, concluding that oral infection affects glycemic conditions in diabetic rats. Shultze et al. [12] reported improved insulin sensitivity after endodontic treatment in teeth having a combined endodontic-periodontal lesion.

CASE REPORT

A 39 year- the old female patient was referred to the department of conservative dentistry and endodontics with a palatal swelling in relation to tooth #16 nonresponsive to periodontal treatment as a main chief complaint (Figure1 a). The patient's medical history revealed a type I Diabetes Mellitus (DM) under insulin therapy, with four times insulin intakes in the day as prescribed by the referring Diabetologist. The patient's latest glycated hemoglobin levels showed poor glycemic control (Hb1Ac=9%) despite the regular medical check-ups and the patient's compliance with treatment of DM. In the same patient, tooth #14 was with pulp necrosis.

Full-mouth examination revealed that teeth (17, 16, 15, 14, 26, 27, 35, 44, 47) were the remaining teeth and were with periodontitis.

- Clinical findings in tooth#16

- Inspection of tooth #16 and the surrounding tissues showed the presence of a periodontal defect on the buccal and palatal aspect of the tooth (Figure1 b) and a suppurating ulcerative lesion expanding along the palatal side of the tooth.
- Periodontal attachment loss was recorded around tooth # 16 throughout periodontal probing. 10 mm pocket depth was recorded in the palatal side of the tooth.
- Thermal pulp sensitivity testing was with a negative pulp response.

- Clinical findings in tooth #14

- Tooth#14 showed a deep carious lesion in the cervical tooth area with a radicular extension (Figure 2a)
- A periodontal attachment loss was recorded around tooth #14 on periodontal probing.
- The tooth was non-responsive to thermal sensitivity tests
- Axial percussion revealed a different response in tooth #14 comparatively to adjacent teeth.

- Radiographic findings in tooth #16

Radiological examination revealed a generalized marginal alveolar bone loss, thus indicating a diseased periodontal context modified by Diabetes according to the American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP) 2017 consensus report. [13]

A coronal approximal restoration deeply extending to the Mesial pulp horn was identified radiographically. Periapical radiographs examination revealed the presence of horizontal alveolar bone lysis extending to the root middle third and a periapical radiolucency with respect to tooth #16 (Figure 1 c).

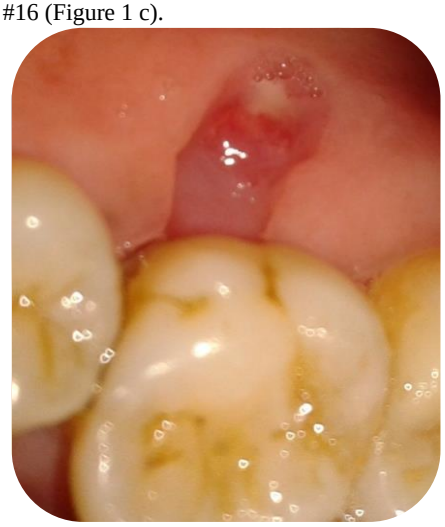

Figure 1a: Suppurative lesion with unusual shape in the palatal side of tooth #16

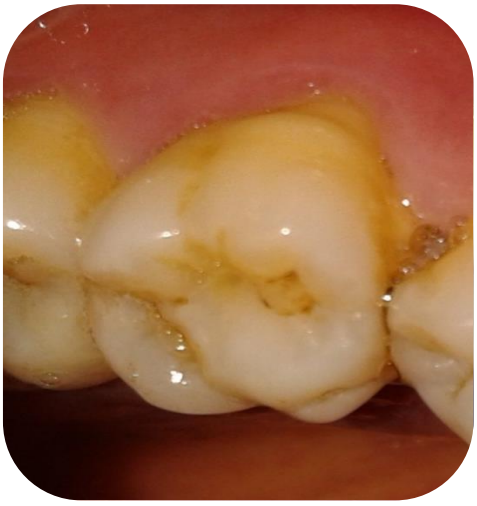

Figure 1b:Periodontal attachment loss in tooth #16

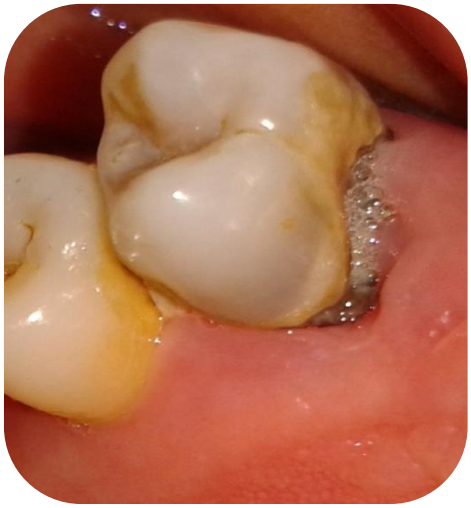

Figure 2a: Periodontal attachment loss and deep carious lesion in the mesial aspect of tooth #14

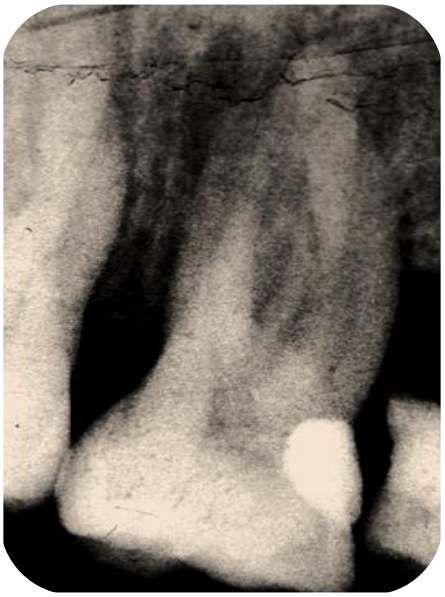

Figure 1c: Pre-operative radiograph of tooth#16

- Radiographic findings in tooth#14

Periapical radiograph revealed that the carious lesion was penetrating the pulp cavity of tooth #14. A Periodontal ligament space widening was recorded in the periapical area of the tooth. (Figure 2b)

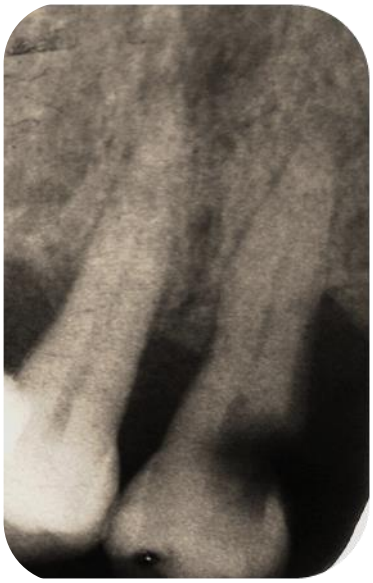

Figure 2b: Pre-operative radiograph of tooth #14

- Diagnosis

From previous data, the suppurating lesion in relation to tooth #16 was diagnosed as a primary endodontic lesion with secondary periodontal involvement occurring in a periodontally diseased context as per Simon, Glick, and Frank's classification [14]. And according to Herrera et al. [8], tooth #16 represents a Grade 3 endo-periodontal lesion occurring in a periodontitis patient.

Tooth #14 was diagnosed with chronic apical periodontitis.

-Treatment Approach

The treatment planning approach involved management of the endodontic infection, combined with periodontal therapy, including scaling and root planning, hygiene instruction, and establishment of regular periodontal maintenance.

Management of endodontic infection

Endodontic treatment was performed in tooth #16 and tooth # 14 under antibioprophylaxis with Amoxicillin 2g provided 1 hour before the endodontic procedure, in accordance with the guidelines of the European society of endodontology in its position statement (2018) [15].

Pulp necrosis was confirmed throughout endodontic access cavity preparation, which supports the hypothesis of root canal infection contribution to the suppurative lesion in relation to tooth #16 (Figure 1d).

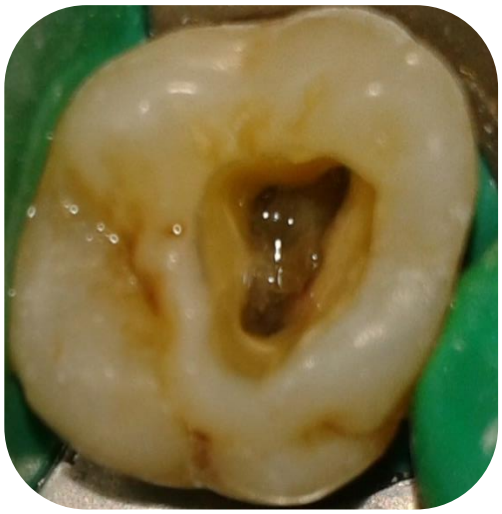

Figure 1d: Pulp necrosis confirmed in tooth #16 during endodontic access cavity preparation

The root canal system was shaped using Protaper® rotary file system (Dentsply-Maillefer, Baillagues, Switzerland). The crown-down technique helps to improve the delivery of irrigating solutions into the apical root canal third and achieve a better root canal disinfection.

During root canals instrumentation, Glyde™File Prep (Dentsply Maillefer, Ballaigues, Switzerland) was used to optimize the dissolution of dentinal root canal walls inorganic components that may harbor residual infection.

Copious irrigation with 2.5% sodium hypochlorite was used during root canal instrumentation to prevent packing of debris in the root canal apical portion, extrusion of microbial infection into periapical tissues, and enhance disinfection and debris removal.

A powder/ liquid mixture of calcium hydroxide was prepared using a local anesthetic to get a tick paste used as intracanal medication and temporarily left for ten days in the root canals due to persistence of inflammatory exudate after chemo-mechanical debridement. Calcium hydroxide is recognized for the inactivation of bacterial endotoxins leading to inflammatory reactions in periapical tissues. Thus it was used for its antimicrobial and anti-inflammatory properties [16]. Root canal filling was achieved in a second appointment (Figure 1e and Figure 2c).

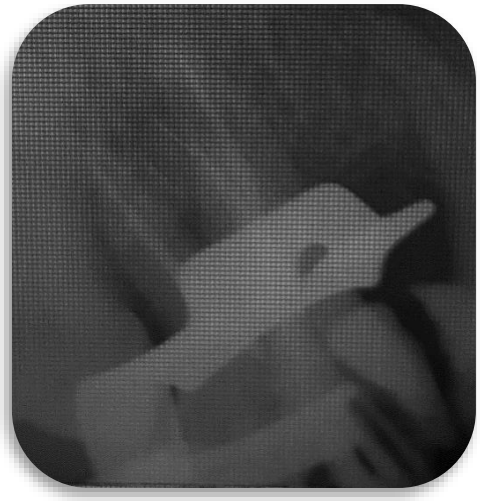

Figure 1e: Endodontic treatment performed in tooth #16

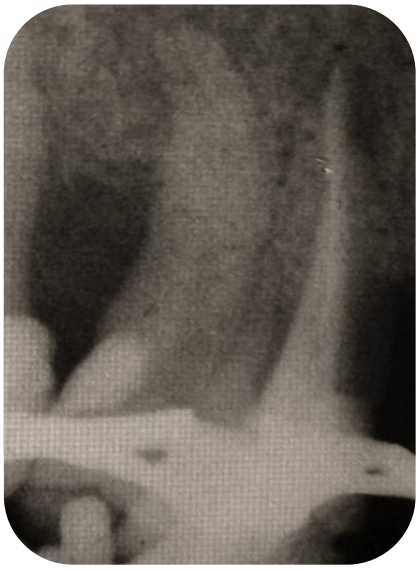

Figure 2c: Endodontic treatment performed in tooth #14

Periodontal debridement

Scaling and root planning had been achieved in tooth #16. Periodontal debridement was also carried out in the patient's remaining teeth with periodontitis.

Follow-up

Regular follow-Ups were scheduled to evaluate the administered therapies outcomes and monitor the endo-periodontal lesion evolution in tooth# 16. The patient had been reevaluated three weeks later, then 1month, 6months, and 12 months.

Response to treatment

- 3-Week Follow-Up

Resolution of the purulent exudate with respect to the palatal lesion in tooth # 16. (Figure 1f).

- Six-Month Follow-Up

A Decrease in the Pocket depth values was recorded in both tooth # 16 and tooth # 14 after periodontal debridement and endodontic treatment. In the palatal side of tooth # 16, the pocket depth dropped from 10mm to 6mm.

Periapical bone reorganization lesion has been recorded on periapical radiographic control in tooth #16. (Figure 1g)

- Twelve-Month and Twenty-four-Month Follow-Up

No recurrence of the palatal lesion in tooth #16 has been recorded. Periapical lesion healing in the same tooth was achieved. (Figure 1h)

Regular follow-ups of the patient also revealed an improvement in HbA1c levels that dropped from 9% to 7,5%. (Figure 3)

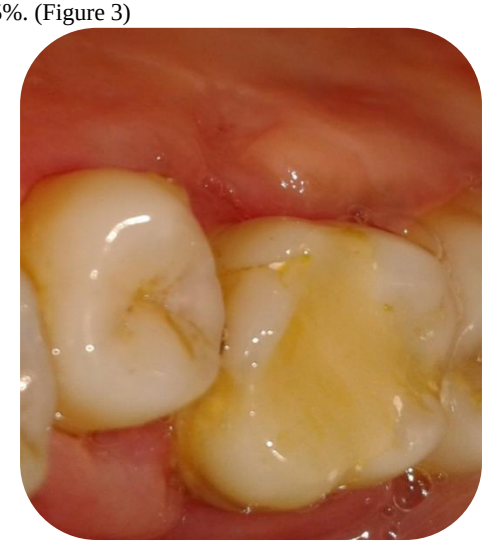

Figure 1f: disappearance of the purulent exudate with respect to the palatal lesion in tooth#16 3weeks after endodontic treatment achievement

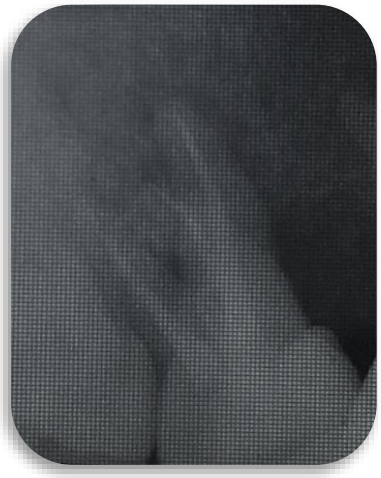

Figure 1g : Six-Month radiographic control

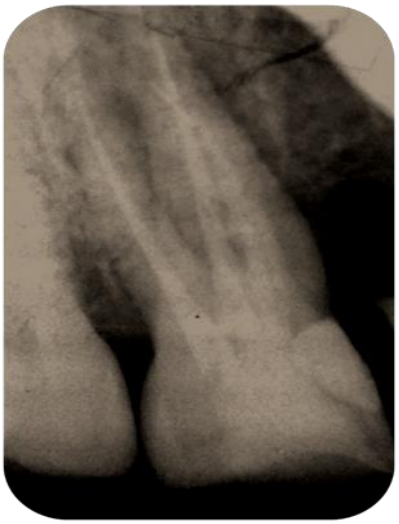

Figure 1h : twenty four Month radiographic control

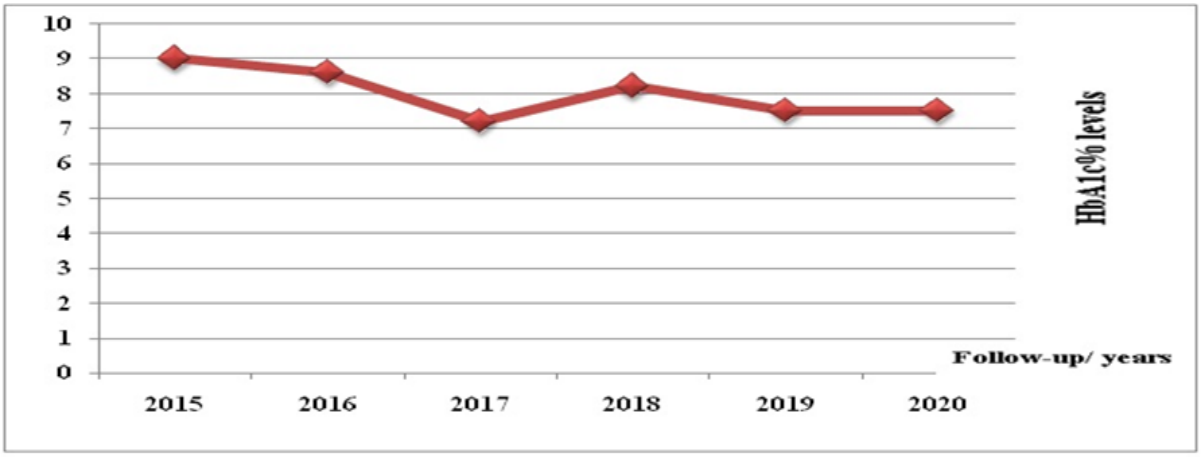

Figure 3: Regular follow-ups revealed HbA1c levels decrease following management of root canal infection in tooth #16 and tooth #14

DISCUSSION

The present case report highlights the successful management of an endo-periodontal lesion in an uncontrolled type I Diabetic patient.

Current evidence supports uncontrolled diabetes leading to periodontal disease aggravation. However, literature on the pathogenesis, progression, and healing of endodontic infection complications in periradicular tissues in diabetic patients is remarkably scarce. In fact, the Diabetic host may be more susceptible to significant infections because of different root canal microbial profiles in teeth with pulp necrosis, and infected root canals in diabetics harbor a more virulent microbial profile [17]. Furthermore, hyperglycemia related to an uncontrolled diabetic state is associated with the subsequent formation of advanced glycation end products (AGEs), which correspond to glycation and oxidation of proteins and lipids. Accumulation of AGEs in the plasma and tissues and interaction with their receptors in tissues may lead to an exacerbated inflammatory response with the increase of the production of cytokines and chemokines, immune cells including monocytes and macrophages, a higher bone resorptive and reduced bone formation activities, reduced resistance to microbial infection and decreased repair capacity [18]. Diabetes has been reported to be related to growth factor decrease, apoptosis of leucocytes, and arrest of polymorphonuclear leucocytes recruitment. Thereby, persistent hyperglycemia enhances bacterial multiplication and leads to an exaggerated immuno-inflammatory response to pathogenic bacterial challenges. [19]

Periodontal disease and endodontic infection share important similitudes. Both are polymicrobial infections with a common microbiota and predominance of Gram-negative anaerobic bacteria. On the basis of all previous data, we can assume that expression of endodontic infection in periradicular tissues within uncontrolled Diabetic hosts is highly enhanced.

In diabetic hosts, other factors can occur; increased glucose content in saliva may increase the adhesion of fungi in epithelial cells, and the normal oral flora becomes altered in response to endocrine disorders, which may explain the higher incidence of opportunist infections [20]. Candida Albicans is considered an opportunist microorganism that was isolated more frequently in diabetic patients, Candida species are found not only in periodontal pockets but also in teeth with pulp necrosis and periradicular tissues disease due to endodontic infection, and they possess multiple virulence factors that help the invasion of the host tissues and evasion of host defense mechanisms[21]. The ulcerative Periradicular purulent lesion in tooth#16 in this case report can be attributed to the coexistence of periodontal and endodontic infection in an uncontrolled diabetic host, which could have contributed to an exacerbated inflammatory response in periradicular tissues, dealing with both infection origins allowed adequate wound healing of injured tissues around the tooth.

Further evidence is needed to establish that endodontic infection has an adverse effect on glycemic control and that eradicating endodontic infection can contribute to glycemic control management. In the present case report, we can suggest HbA1c levels improvement following endodontic infection management in both of tooth #16 and tooth # 14, and after periodontal debridement carried out in the same teeth and the other remaining teeth with periodontitis, bearing in mind that the patient was under regular medical check-ups and complying with Diabetologist hygiene and dietary rules and insulin intakes. Many microbiological findings can support this fact. Cintra et al. [11] observed that glycemic control was affected in diabetic rats by endodontic infection and increased blood glucose and HbA1c levels. Inflammatory mediators released in periodontal inflammation have been reported to antagonize insulin action and activate inflammatory responses, increase the production of cytokines and chemokines, and activation of immune cells, cause together local insulin resistance.

CONCLUSION

In uncontrolled Diabetic patients, the endodontic infection may take severe expressions in periradicular tissues, especially if combined with periodontitis. Adequate management of this infection can lead to favorable outcomes regarding periradicular lesions healing and Diabetes mellitus control, even in patients with poor glycemic control.

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None.

AUTHORS' CONTRIBUTIONS

The participation of each author corresponds to the criteria of authorship and contributorship emphasized in the Recommendations for the Conduct, Reporting, Editing, and Publication of Scholarly work in Medical Journals of the International Committee of Medical Journal Editors. Indeed, all the authors have actively participated in the redaction, the revision of the manuscript, and provided approval for this final revised version.

COMPETING INTERESTS

The authors declare no competing interests with this case.

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None.

PATIENT CONSENT

Written informed consent was obtained from the patient for the publication of this case report.

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