

The "Point of Care" section answers everyday clinical questions by providing practical information that aims to be useful at the point of patient care. The responses reflect the opinions of the contributors and do not purport to set forth standards of care or clinical practice guidelines. Readers are encouraged to do more reading on the topics covered. If you would like to contribute to this section, please contact editor-in-chief Dr. John O'Keefe at [jokeefe@cda-adc.ca](mailto:jokeefe@cda-adc.ca).

## QUESTION 1

### What is the role of the dentist in the early diagnosis of temporal arteritis?

Temporal arteritis is an inflammatory disease of the large and medium arteries. It is characterized histologically by the presence of giant cells at the level of the arterial wall.<sup>1,2</sup> Biopsy is required for histological confirmation of the disease,<sup>3</sup> but the rate of false negatives on biopsy exceeds 60% the rate of true positives (Fig. 1).<sup>4</sup> This high rate of false negatives is due in part to the segmental nature of the damage to the temporal artery, which does not necessarily occur along the entire length of the artery.<sup>3</sup>

At least 3 different names are used to refer to this clinical entity: temporal arteritis, Horton's disease (not to be confused with Horton's cephalalgia or cluster headache) and giant-cell arteritis. Although the condition is characterized by arteritis (among other things), it is not necessarily limited to the temporal artery; similarly, giant cells are not always present and may also be found in other forms of arteritis.

The annual incidence of the disease among people 50 years of age or older in northern Europe and the northern United States is 5 to 30 per 100,000.<sup>2,5,6</sup> Its occurrence is associated with certain known risk factors (Table 1).

Temporal arteritis is a serious disease, and one of its most dreaded complications is blindness.<sup>7</sup> However, if diagnosed early, it is relatively easy to

**Table 1** Risk factors for temporal arteritis<sup>2,5</sup>

Variable	Group at risk
Age	> 50 years
Sex	2 to 4 times more frequent among women than men
Ethnic origin	White
Genetic predisposition	Family history

treat.<sup>8</sup> Given that one of the many clinical signs is jaw claudication<sup>9</sup> (pain on chewing), patients with temporal arteritis may decide to consult a dentist, who may thus be a key player in early diagnosis.

#### Manifestations and Consequences

The symptoms of temporal arteritis are highly variable. The occurrence of frequent (e.g., daily) temporal or occipital headaches that are resistant to regular analgesics is the most common symptom, found in two-thirds of patients.<sup>6</sup> Fever, present in 50% of cases, is usually moderate.<sup>6</sup> Anorexia with weight loss and asthenia is observed in one-third of patients.<sup>6</sup> In 40% to 50% of cases, temporal arteritis is associated with polymyalgia rheumatica, which is characterized by sudden inflammatory pain in the shoulders and hips.<sup>2</sup>

Local manifestations are rarer and sometimes more subtle. Swelling of the temporal artery with induration is a pathognomonic sign of the disease (Fig. 2). The artery is sensitive to palpation, and arteriosclerotic occlusion may make it more difficult to find the pulse. Jaw claudication, occurring in one-third of subjects,<sup>6</sup> is also characteristic of the disease.<sup>9</sup> It is important, however, not to confuse jaw claudication with temporomandibular joint disorders or other abnormalities of the buccofacial sphere (Box 1). In cases of temporal arteritis, the pain is caused by partial occlusion and ischemia of the local arteries. The patient typically reports pain of the mandible,



**Fig. 1:** Patient who has undergone biopsy of the temporal artery.



**Fig. 2:** Dilatation of the temporal artery in a patient with temporal arteritis.

**Box 1** Differential diagnosis of mandibular pain on chewing

Migraine headache  
 Vascular pain  
 Trigeminal neuralgia  
 Dental problems  
 Temporal arteritis or Horton's disease

which is exacerbated by brief, intense periods of chewing (e.g., while chewing hard foods). The pain disappears at rest<sup>6,9,10</sup> and is not provoked by sporadic chewing or by opening the jaws wide.

The vascular manifestations are highly variable and, in rare cases, include partial or total coagulative necrosis of the hemitongue, which may be unilateral or bilateral.

The most dreaded ocular complication, which may be the first complication to arise, is sudden, possibly irreversible loss of vision.<sup>6,11,12</sup> In many cases, the appearance of prodromal signs (such as amaurosis fugax, ptosis or diplopia) allows an appropriate response to prevent these complications.<sup>11,12</sup>

In rare cases, this condition, if left untreated, may lead to death, usually because of ischemic stroke, myocardial infarction or aortic rupture. Digestive ischemia and gangrene of the extremities are other major complications.<sup>5</sup>

**The Role of the Dentist**

Patients who experience pain on chewing typically consult a dentist, believing the problem to be of dental origin. Rapid diagnosis of temporal arteritis can make a real difference in terms of the patient's prognosis, particularly with regard to visual complications. The longer it takes to diagnose the disease, the greater the risk that an ischemic complication will develop.<sup>6</sup> Dentists should therefore be alert to the possibility of this diagnosis in elderly patients, particularly women, who present with mandibular pain on chewing, especially if there is no relationship between the reported pain and a specific dental problem. The patient should be referred urgently to an ophthalmologist or internist for diagnosis and appropriate treatment with corticosteroids.<sup>11,12</sup> ➤

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## QUESTION 2

## Why do implants fracture, and how can I prevent this from happening?

In most cases, implants are extremely efficacious, but unexpected negative outcomes are occasionally encountered. If an implant is lost, not only has the primary treatment proven unsuccessful, but additional treatment may be required to achieve a positive outcome. Prevention of complications should be one of the primary goals of implant therapy.

Three aspects of implant fracture make this complication worthy of attention. First, although implant fractures are rare, they represent a notable proportion of late implant failures. Second, this complication is usually preventable. Third, management of implant fracture is usually more complex than management of other types of implant failure, because the presence of the apical fractured fragment complicates treatment: the remaining implant segment prevents ready insertion of another implant, while trephination and removal of the segment may create a large defect.

The reported incidence of implant fracture is very low — 0.08% to 0.74%<sup>1,2</sup> — representing 20% or less of all late implant failures. Given recent improvements in the design and composition of dental implants and a better understanding of the restorative interface, the current incidence of implant fracture is probably even lower than the reported figures. Most fractures are horizontal and occur either just below the prosthetic platform or at the second or third external thread (the point at which the abutment screw usually ends). Implant fractures are fatigue fractures, meaning that they occur over numerous cycles of occlusal loading. Consequently, they take time to develop, with most fractures occurring after 5 years of clinical function.<sup>3</sup> Most fractures are preceded by multiple episodes of loosening or fracture of the prosthetic or abutment screw (Fig. 1); as such, occlusally mediated fatigue fracture is probably the primary cause of implant fracture.<sup>3</sup> Although it is tempting to suspect that manufacturing defects may be the cause of implant fractures, the available research does not support this hypothesis.<sup>4</sup>

A rare but equally unpleasant cause of implant fracture is connected with the indiscriminate application of torque-controlled motors or torque drivers to narrow-platform internal connection implants. The dimensional limitations imposed by the design features of these implants diminish

the built-in safety margin, and excessive torque values (i.e., beyond the manufacturer's recommendations) may damage the connection between fixture and abutment. Such a fracture may occur at the insertion of the fixture or, more likely, at the insertion of the prosthesis. The incidence of this complication is unknown.

### Diagnosis

The ease of diagnosis of implant fracture depends largely on the extent of the fracture and the number of implants supporting the prosthesis. If an implant supporting a single crown has completely fractured, the patient will typically present to the office with the crown in hand. Alternatively, if the implant has fractured close to the prosthetic platform, but the abutment screw has not yet fractured, the clinical presentation may be a loose crown that can be rotated on its axis.

If an implant has developed a fracture line but has not yet completely broken into 2 pieces, the diagnosis can be challenging. A lengthy period of time may transpire from the moment the fracture begins to develop until it reaches its inevitable conclusion. It is unknown whether the bone changes that are often associated with implant fractures precede and contribute to the fracture or are merely a consequence of the fracture in progress.

Fracture of an implant supporting a multi-unit restoration may not be as obvious clinically, since the support provided by the remaining implants may preclude any movement of the prosthesis.



**Figure 1:** An implant that fractured after several years of function. This fracture was preceded by several episodes of loosening of the multi-unit prosthesis.

Hence, the patient may be asymptomatic and completely unaware of the problem. Over time, however, the decline in support to the prosthesis will likely result in further complications. Secondary inflammatory soft-tissue changes may also be observed.

### Prevention

Several basic principles may help to reduce the chances of implant fracture. In current general practice, implant fracture is overwhelmingly a phenomenon of posterior single-implant restorations. The load-sharing feature of well-fitting multi-unit restorations usually shields any single implant from undue occlusal forces (unless excessive cantilevers are present). Single implants in the posterior region are particularly vulnerable to occlusal overload because of greater occlusal forces in the posterior region, the wider occlusal tables of posterior restorations, the possibility of significant bending moments and the generation of laterally directed forces because of cusp orientation. Rangert and others<sup>5</sup> found that 90% of implant fractures occurred in the posterior region. Prevention is easily accomplished:

- Use wider-diameter implants to decrease bending moments in the posterior region and to provide greater bulk of metal to resist deformation under off-axis loading.
- Use multiple implants to share the load.
- Decrease the width of the occlusal table.
- Decrease offsets and cantilevers.
- For single implants, ensure that there are no occlusal or excursive contacts (must be able to slide shim stock through).
- Be particularly cautious in planning treatment for a single terminal posterior implant. These implants are not shielded by the presence of a tooth posterior to them, and their occlusal dimensions are subject to excessive contouring by laboratory personnel.
- Perform yearly follow-up to ensure that the opposing tooth has not overerupted to contact a restoration supported by a single implant.
- If multiple implants are used, ensure passive fit of the framework.
- Encourage patients with a history of potentially destructive parafunctional behaviour to wear a nightguard appliance.
- Follow the manufacturer's recommendations with respect to the use of implant components, paying particular attention to recommended

torque values. Exceeding these values may cause irreparable damage to the components.

- Repeated fracture (of acrylic, porcelain or screws) or loosening of the prosthesis is a clue to poor distribution of the patient's occlusal forces to the components of the masticatory apparatus that are least able to withstand them. These early complications are easy to resolve and can call attention to other, more serious complications to follow.

### Conclusion

Implant fracture is a rare, usually preventable complication of dental implant therapy. Careful planning and execution of restorations according to documented protective principles should help to prevent most cases of implant fracture. ♦

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*In the next "Point of Care" article, the authors discuss management of fractured implants.*

## QUESTION 3

## What is the best strategy for managing a fractured implant?

In the previous “Point of Care” article, we reviewed the causes of implant fracture and suggested ways to reduce the chances of this problem occurring. However, if an implant does fracture, management will depend on the location of the fracture (between teeth or in a terminal position), the level of the fracture (superficial or deep), the space remaining between the fixture and the adjacent teeth, the condition of adjacent teeth or implants, and the patient’s esthetic and functional needs. A superficially fractured implant that is accessible can (under certain conditions) be restored by cementing a post into the screw channel. Although such a restoration will not be retrievable, this approach may allow other, more invasive alternatives to be postponed or avoided. An implant that is very close to the adjacent teeth (as can occur with a single implant replacing a mandibular incisor) may be impossible to remove by trephination without damage to the adjacent teeth. Such an implant is best left alone after fracture.

### Management

If prosthodontic replacement of a fractured implant is desired, attention should first focus on the status of adjacent teeth, implants or edentulous spaces. The dentist should determine whether adjacent teeth or implants can be modified to provide a bridge (or a cantilevered bridge) and whether adjacent (usually distal) edentulous spaces can be used to anchor additional implants, so that the final restoration can be cantilevered forward over the site of the fractured fixture. Surgical removal of the fixture should be undertaken only after other options have been considered and deemed unsuitable.

Clinical management of a fractured implant must be driven by the desired restorative outcome and the risk of further deterioration of the remaining implant segment. Practitioners must resist the temptation to remove a fractured implant simply because the radiographic appearance is unattractive or because leaving the implant in place will allow future clinicians to see that the fracture has occurred. Unattractive radiographic appearance is not a disease. “Sleeper” implants (implants that have been left submerged or that have been resubmerged for a variety of reasons) have been known to remain in the jaw indefinitely without

complications. Such implants can always be removed at a later date, should the need arise.

If removal of the implant by trephination is deemed appropriate, the procedure must be executed with due care and precision. It must be remembered that after trephination the osteotomy site will be larger than the original diameter of the fixture. Because management of the resulting bony defect depends largely on the prosthodontic plan, it is imperative that this plan be formulated before the surgical procedure. For example, if the site will not be used for any fixed or removable prosthodontic purpose now or in the future, grafting of the site is unnecessary.

Before the implant is removed, the surgical colleague needs to know the following:

- the brand, diameter and length of the implant (so that a trephine of appropriate diameter can be prepared; **Fig. 1**)
- the final prosthodontic treatment plan (Is the goal to simply remove the implant, or will a site be created to host a future implant?)
- the location of adjacent anatomic boundaries and structures that may affect surgical access.

### Removing the Fixture

If the fixture is to be trephined, the following steps are usually taken:

- 1 Estimate the length of the remaining fractured segment. This is important for determining the proper depth of trephination.
- 2 Elevate a full-thickness flap for access and visibility (**Fig. 2**). If buccal or lingual depressions are present, the flap elevation should involve the entire



**Figure 1:** A trephine of an appropriate diameter should be selected.



**Figure 2:** A full-thickness flap is reflected to visualize the fractured fixture and adjacent surgical anatomy.



**Figure 3:** The trephine is carefully aligned and centered with the long axis of the fixture and is used to create a controlled circumferential trough around the fractured fixture.

expected length of the remaining fixture, to avoid the unpleasant possibility of the cutting edge perforating through the bone laterally and creating a soft-tissue injury. At all times, the speed and the dimensions of the cutting edges of the trephine must be borne in mind.

- 3 Estimate the angulation of the fixture by inserting a long impression pin into the central channel of the fixture. Knowledge of the angulation is important in ensuring proper alignment of the trephine with the long axis of the fixture.
- 4 Use the trephine to create a circumferential trough around the fixture (Fig. 3). The selected trephine should be only barely wider than the diameter of the fixture.
- 5 Place a tall healing abutment, an impression coping or impression pin into the internal channel of the fixture to create a simple means of lifting the segment out of the bone.
- 6 A narrow elevator is usually needed to gently fracture the remaining few bony trabeculations that are invariably attached to the apical portion of the fixture.
- 7 The site can now be grafted as required. Sharp bony projections may need to be smoothed out to avoid puncturing the flap following closure and to create a smooth bony contour that is not bothersome to the patient.

8 If the site is grafted, an implant can be placed after an appropriate period of healing. The healing period depends on the quality and quantity of native bone that remains after removal of the implant.

9 Following healing of the implant, the restorative phase should focus on avoiding the factors that led to the fracture and need for secondary intervention.

### Submerging the Fixture

Scenarios have already been pointed out where it may not be necessary, desirable or possible to remove the fixture. In these situations, the decision can be made to submerge the fixture (rather than remove it, as described above). The following steps can be undertaken:

- 1 Smooth out any sharp edges at the fracture site with a high-speed surgical handpiece and copious irrigation.
- 2 Ensure that the most coronal aspect of the fixture is sufficiently deep to permit uneventful soft-tissue coverage.
- 3 Rigorously irrigate the internal aspect of the fixture.
- 4 Long-term follow-up is required to ensure continued health of adjacent tissues.

### Conclusion

Several strategies are available to deal with a fractured implant. These aim to preserve the health of the remaining structures and take into account the overall rehabilitative plan for the patient. The risks associated with removing a fractured implant must always be balanced against the risks of future disease should the fractured implant be left in place. ♦

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