Table I. Continued

Author/ year	Age	Gender	Race	Jaw	Location	Radiograph
	21	F	?	Mandible	Distal third molar	Unilocular
	22	F	?	Mandible	Premolar/molar	?
	39	F	?	Mandible	Premolar/molar	?
	43	F	?	Mandible	?	?
	50	F	?	Mandible	Premolar	Unilocular
Taylor, 1999	17	F	?	Mandible	Canine/premolar	Multilocular
De Lima, 2008	24	F	W	Mandible	Right to left first molar	?
Younis, 2008	57	F	W	Mandible	Premolar/molar	Unilocular
Tosios,	18	M	?	Mandible	Premolar/molar	?
2008	20	F	?	Mandible	Premolar/molar	?
	50	M	?	Mandible	Premolar/molar	?
	73	M	?	Mandible	Premolar/molar	?
	15	M	?	Mandible	Premolar/molar	?
	59	M	?	Mandible	Premolar/molar	?
	25	M	?	Mandible	Premolar/molar	?
Damm, 2013	75	F	?	Mandible	Incisor	Unilocular
This case	22	F	W	Mandible	Premolar/molar	Multilocular

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CLINICAL PATHOLOGIC CONFERENCE CASE 2: PALATAL PERFORATION

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Clinical Presentation: A 41-year-old man presented with a 1-week history of a "hole in the roof of his mouth" with associated discomfort (Figure 1). He reported burning the roof of his mouth 4 weeks previously while eating a hot slice of pizza. Initially, there was a pin-sized hole on the palate that continued to enlarge over 1 week. He had since been unable to eat or drink anything comfortably, as whatever he ate or drank entered his nasal passage. At his initial visit, he presented with an "obturator" that he fashioned out of chewing gum that allowed him to sip on soup and Ensure. The patient's medical history was significant for back problems and chronic sinus infections managed with over-the-counter medications. The patient reportedly smoked 1 pack of cigarettes a day for many years and did not consume alcohol. As a warehouse custodian, he reported working in a chalk-filled environment. He lived with his wife, 6-year-old son, 3 dogs, and 2 cats.

Oral examination revealed a uniformly round perforation, approximately 8 mm in diameter, in the left anterior hard palate region, just off the midline and posterior to the palatal rughae (Figure 1). The defect did not have a base, and oral–nasal

communication was evident. The surrounding mucosa demonstrated diffuse erythema, which extended posteriorly and across the palatal midline.

Differential Diagnosis: The acute presentation of a uniformly round perforation on the palate suggested several possibilities. Physical trauma was among the first considerations, especially in light of the patient's own report of having burnt his palate. However, it was highly unlikely that a pizza burn would cause destruction of underlying hard tissue. The round defect also suggested a physical injury with a foreign object (pencil, tool, etc.), but there was no history of such trauma. Nasotracheal intubation during general anesthesia administration can result in palatal perforation, especially if the patient has diabetes or is immunocompromised.[1] Our patient reported being generally healthy, with no history of recent or past surgery.

Drug related chemical injury was considered highly likely given the clinical presentation and lack of other systemic or localized symptoms. Drugs such as cocaine when insufflated (snorted) can produce the type of defects presented due to localized vasculitis and ischemia.[1-5] The patient did not report his previous history of cocaine abuse at this initial visit and admitted to it only after initial diagnostic radiographs were obtained.

A number of infectious diseases are known to cause palatal perforation in some cases. Tertiary syphilis can cause large areas of granulomatous inflammation and necrosis with perforation of the palatal bone resulting in an oronasal fistula.[1,6] Other infections associated with palatal perforation, necrosis, and destruction include zygomycosis, aspergillosis, sinonasal blastomycosis, histoplasmosis, coccidiodimycosis, tuberculosis, leprosy, rhinoscleroderma, toxoplasmosis, and leishmaniasis.[7-9] At the initial visit, at least on the surface, there appeared to be no granulomatous inflammation with necrosis, ulceration, or both. Our patient was generally healthy, with no history of constitutional signs or symptoms. His routine physical and bloodwork results were reportedly within normal limits. Furthermore, no necrotic or ulcerative tissue was present, requiring biopsy and histopathologic examination.

Next, an immune-mediated etiology was considered. Diseases, such as systemic lupus erythematosus, granulomatous polyangitis (GPA, or Wegener disease), sarcoidosis, and other vasculitides,[1,7] can potentially present with palatal perforation but usually present with evident necrosis and soft tissue destruction. Also, patients with these diseases present with generalized multisystem involvement with accompanying constitutional signs and symptoms. Our patient did not present with any of the above findings making these diagnoses unlikely.

Finally, a malignant neoplasm extending from the maxillary sinus or nasal cavity capable of causing the palatal perforation was considered. These malignancies may include natural killer–cell or T-cell lymphomas and other destructive midline malignancies, including olfactory neuroblastoma (esthesioneuroblastoma) or sinonasal undifferentiated carcinoma.[10-12] Palatal malignant neoplasms that may also cause extensive tissue destruction include malignant salivary gland tumors (mucoepidermoid carcinoma, adenoid cystic carcinoma, and other malignancies of minor salivary gland origin), malignant nerve sheath tumors, and metastasis.[13] These processes are generally associated with a fungating mass, with evident ulceration, as well as necrosis with or without constitutional signs or symptoms. Our patient was asymptomatic, and the presenting lesion was inconsistent with a neoplastic process.

Given the initial clinical presentation, a diagnosis of either physical trauma or drug-induced (cocaine) nasal floor perforation was considered.

Diagnosis and Management: The patient's initial interview was conducted in the presence of his 6-year-old son. Additional clinical information revealed that there was no history of pain, paresthesia, exudative discharge, foul odor, or swelling. The patient was healthy and reportedly received a full physical with bloodwork 2 months ago, and the findings had been within normal limits. Extraoral examination was unremarkable, with no evidence of swelling or lymphadenopathy. Not surprisingly, following removal of the "obturator," pronounced rhinolalia was noted upon enunciation. Oral examination revealed the perforation described above. The further oral examination revealed a moderately restored, partially edentulous dentition. There was no evidence of ulceration, discharge, or swelling; the area in question was nontender to palpation.

A panoramic radiograph showed partially edentulous adult dentition with several restorations (Figure 2). The palatal perforation could not be properly evaluated. Occlusal radiographic examination confirmed the presence of a large oral–nasal–antral perforation (Figure 3). A large, partially defined radiolucency measuring approximately 4 to 5 cm anteroposteriorly across the palatal midline and 1 to 3 cm in width. This large radiographic defect, in the absence of obvious swelling, necrosis, and discharge triggered an otolaryngologic referral for nasal endoscopy and laryngoscopy. Diagnostic computed tomography of the head was requested ahead of the patient's scheduled otolaryngology appointment.

The history, the presentation, and the notable findings noted on plain radiography suggested the possibility of substance abuse–associated palatal perforation. The patient was contacted the day after his initial visit. He was asked again about any previous history of substance abuse. He admitted to a distant history of cocaine insufflation (>10 years) and reported being addiction free for over 10 years. He had been understandably reluctant to discuss this the previous day because his son had been present in the examination room.

Diagnostic computed tomography revealed perforation and destruction of the nasal floor, palatal vault, and nasal septum and diffuse lytic change involving the lateral nasal walls and turbinates (Figures 4A and B). Soft tissue sinus membrane thickening was noted in keeping with the patient's history of sinusitis. The features were characteristic of midline destructive disease (MDL), which can be caused by a range of conditions, including cocaine abuse.

Following nasal endoscopy, laryngoscopy and a thorough otolaryngologic evaluation the above findings of MDL were confirmed. However, there was no evidence of necrotic tissue or mass on examination. The perforation was surrounded by mildly inflamed soft tissue but was described as being generally "clean." A biopsy of the area was not indicated; further serologic tests were not performed. Further audiometry revealed left-sided hearing loss.

The clinical history, presentation, and radiographic findings provided sufficient evidence to arrive at a final diagnosis of palatal perforation associated with so-called cocaine-induced MDL (CIMDL).[14,15] The patient's previous history of cocaine abuse was responsible for the notable osseous destruction of the midface. The palatal burn caused by eating a hot pizza slice was incidental and was the inciting agent that caused the mucosal defect. The mucosa overlying this large submucosal osseous defect was likely the only layer of integument separating the oral cavity from the floor of the nose. An acrylic obturator was fabricated to improve function in the patient. The patient was presented with the option of undergoing surgical reconstruction with a palatal flap, especially given that he had been drug free for more than 10 years. At his 6-month and 1-year follow-up visits,

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