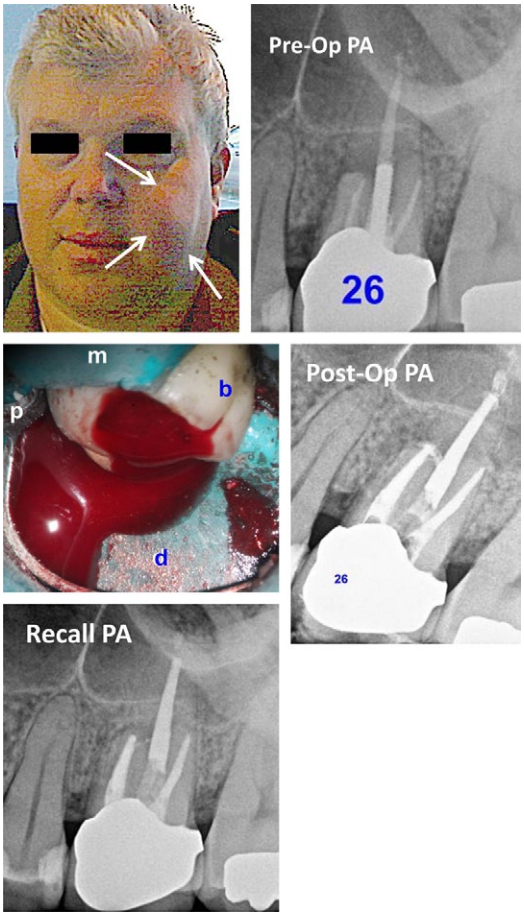
RICHMOND
ENDODONTIC CENTRE

FROM AAA TO AAH

JUN 2017



Acute Apical Abscess (AAA) is painful and arises quickly; it is a true endodontic emergency. It consists of a severe inflammatory reaction to pulp necrosis and an infective process in the canal system. There will be pus formation and swelling of neighbouring tissues. Fascial space involvement, malaise, fever, and lymph-adenopathy may be obvious or imminent. The unanticipated symptoms develop startlingly quickly, causing both alarm and fear to emanate in the affected person.

Patients will report a continuous ache and possible throbbing sensation with random bouts of sharp pain. The tooth and surrounding tissue are remarkably tender to even slight palpation. On occasion, insufferable misery is elicited with even the slightest of touch, such as the tongue inadvertently brushing against the tooth. Understandably, these poor souls may demand pain relief in lieu of examination. Although empathetic to the hair-trigger sensitivity and debilitating response, methodical examination and tests with a genuine diagnosis cannot be precluded. Diagnosis will be a challenge; proceed gently and skillfully to the utmost of your ability. Protocol forbids the hopeful prescription of oral antibiotics to enable examination another day or to anaesthetise the area and treat the 'most likely' candidate.

Unfortunately, there may be no radiographic signs of destruction on pre-op PA's or even CBCT images. Conversely, it is pure conjecture to rely on intuition and radiographic evidence of apical destruction to 'select' which tooth to treat.

The images above are of an AAA case I treated. The middle-aged gent had come straight from the hospital where overnight he had received IV antibiotics and opioids. The extent of the fascial space involvement is apparent on the clinical photo. Mild tenderness and pain was first noticed in the early afternoon the day before. By late evening his family brought to him the hospital as the swelling had started to impinge on his left eye, he had a fever, and the pain was all-consuming.

The clinical exam, pulp/periradicular tests, and x-rays confirmed the diagnosis of previously treated with AAA for 26. All other Q2 posterior teeth were mildly tender to percussion and palpation unlike the hypersensitive 26. Pulp tests confirmed the vitality of the neighbouring teeth and the pre-op PA's evidenced a widened PDL at the apex of the MB root of 26. There was severe swelling of the vestibule buccal to 26 with no discernable sinus tract.

The MB2 was not previously treated. However, upon gaining patency on the palatal canal a tsunami of blood extruded from the orifice. The active haeme exudate eventually subsided and I was able to fully instrument and irrigate the canals prior to medicating with Diapex. The buccal swelling was incised and drained; the patient was encouraged to 'milk' the swelling in the cheek. Post-operatively there was an immediate reduction in the size of the intra-oral swelling and fascial space involvement. The treatment was completed 30 days later without incident. Evidence of healing is apparent on the six-month recall PA and the 26 remains to be asymptomatic and functional.

AAA will surprise both the patient and the clinician. Such cases present many clinical and technical challenges to reliably attaining immediate pain relief and cessation of the infective process. If diagnosed correctly and treated astutely one can turn AAA to aah (i.e. relief) both quickly and predictably.

Regards,

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