

Some clinicians claim that cysts do not heal following root canal treatment. Cholesterol crystals occur in the cyst lumen, lining epithelium, and/or fibrous connective tissue capsule. Based on morphological observations, some authors speculated that true cysts are refractory to treatment because they may behave like a self-sustaining pathologic entity, independent of the influence of endodontic infection. Thus, pocket cysts would have more plausible reasons to become recalcitrant than true cysts, because bacteria in the cyst lumen may provide continuous stimuli for growth and survival of epithelial cells. Actually, the medical literature provides evidence that activated macrophages can deal with cholesterol crystals.⁶⁶ Similar to true cysts, there is no evidence showing that cholesterol crystals can prevent periradicular wound healing after nonsurgical root canal therapy. Inflammatory cells are always seen in the epithelium lining and/or fibrous connective tissue capsule of apical true cysts. There is no evidence that epithelial cells, initially stimulated to grow by inflammatory stimuli in apical true cysts, become immortal and self-sustaining. A study in guinea pigs evaluated the subcutaneous tissue response to implantation of pure cholesterol crystals and reported that macrophages and multinucleated giant. It was extrapolated that accumulation of cholesterol crystals in periradicular lesions may prevent healing following endodontic treatment, and given the extraradicular location of the crystals, even retreatment would not succeed. Because the prevalence of cholesterol crystals in periradicular lesions has been shown to range from 18% to 44% some might infer that the same proportion of lesions would not heal following root canal treatment, if the cholesterol crystals had the ability to sustain inflammation by themselves. So far, no clinical or imaging diagnostic method can reliably differentiate granulomas from cysts, let alone true cysts from pocket cysts. Neoplasm is related to genetic damage of cells, including mutation of proto-oncogenes or loss of tumor suppressor genes to restrain cell growth and proliferation. Neoplastic cells undergo spontaneous division, usually supported by autocrine secretion of growth factors, and are self-sustaining and immortal. Exceptions include cases of chronic bacterial infections contributing to the development of malignancies, such as *Helicobacter pylori* infection and gastric cancer. It must be pointed out that the examined cases may not have necessarily represented cystic lesions, since the study design does not permit histologic examination to confirm the diagnosis. Nonhealing lesions, irrespective of their histologic diagnosis, are virtually always a result of persistent intraradicular infection or extraradicular infection. In some cases with persistent drainage of the cyst fluid into the canal, application of a calcium hydroxide dressing or an apical plug of calcium hydroxide may help achieve a dry canal. Cholesterol crystals can be formed locally in areas of tissue damage or necrosis caused by release of cholesterol from disintegrated cell membranes. It is also highly unlikely that the epithelial cells of true cysts, but not pocket cysts, can suddenly change the patterns of gene expression and start behaving like self-sustaining neoplastic cells. Common examples are tobacco chewing and oral cancer, and smoking and lung cancer. For more details.