CORRESPONDENCE

JAW INVOLVEMENT IN BURKITT'S LYMPHOMA

To the Editor:

The August 1988 issue contained a report by Barriga et al of a 27-year-old HIV-positive bisexual man who, in a period of 39 months, developed two clonally discrete Burkitt's lymphomas.1 The initial lymphoma presented as a pelvic mass accompanied by ascites and pleural effusions. A dozen cycles of chemotherapy led to complete remission and the treatment was terminated 1 year after diagnosis. Twenty-seven months later, a left mandibular swelling occurred that enlarged to a 3.5 × 7 cm mass within 2 months. Excisional biopsy revealed Burkitt's lymphoma. No other tumor was found. Six cycles of the previous regimen resulted in no detectable disease for at least 11 months.

As the authors indicated, the clonal distinctions are compatible with the second tumor being an entirely separate entity. It also suggests that the jaw involvement was favored by conditions other than those that induced the initial pelvic localization. It is worth pondering how possible pathways for jaw tumor development might differ from those operative elsewhere in the body in this type of lymphoma that is so closely linked to an infectious agent.

I hypothesized that dental structures predispose to the development of Burkitt jaw tumors because they provide pathways permitting Epstein-Barr Virus (EBV) to reach the lymphocytes in the jaw marrow.2 For example, in children, the physiologic shedding of deciduous teeth or deep caries with pulp necrosis in the deciduous and permanent teeth could provide access of EBV to their actively hematopoietic jaw marrow. This may explain why endemic Burkitt's lymphoma with its frequent jaw involvement is typically a disease of children.3 But what could the mechanism be in adults, such as the 27-year-old man mentioned previously? After childhood, Burkitt jaw tumors are rare because the jaw marrow has become yellow and so the lymphocytic substrate is absent. However, adults who have severe tooth decay and consequent pulpal necrosis may develop chronic periapical infection and accumulate lymphocytes in the inflammatory response, and could be at risk for jaw tumor formation should EBV be present in their oropharyngeal secretions. Furthermore, like bacteria, these smaller microorganisms could traverse the necrotic dental tissues to the tips of the dental roots. There, these viral particles would contact the lymphocyte, plasma cell, and immunoglobulin-rich periapical granulomas4 that had localized in the adjacent alveolar bone to combat the primary infection of bacterial etiology, dental caries. This juxtaposition of virus and B lymphocytes, as elsewhere in the body, would favor lymphocyte transformation at the site.

This mechanism requires that EBV be present in the oropharyngeal secretions. EBV is also present in another disease, infectious mononucleosis.5 Therefore, I would like to inquire whether this patient had a history or clinical or serologic evidence of infectious mononucleosis about 9 months to 2 years before the development of his jaw mass. Those intervals may represent the range of the incubation period of Burkitt's lymphoma. In the earlier paper describing this patient,6 his throat cultures were said to be EBV-positive. The interval from his initial tumor to the subsequent jaw mass somewhat exceeds the assumed range for the incubation period. It would be of interest to learn over how long a period his throat cultures were positive. If they became negative when remission was achieved in treating his initial tumor, then what intervened to reintroduce the virus to the oropharyngeal secretions? Among the possibilities in this bisexual adult are that he participated in oral sex from an EBV-positive individual so that the virus again entered his oropharyngeal region. Yet, once there how did it reach his jaw marrow? I believe this was accomplished through dental pathways.

It is unfortunate that the dental status of patients with Burkitt's lymphoma is rarely recorded. However, support for the importance of dental pathways is afforded by reports that two children having carious teeth developed tumors in the adjacent jaw.7,8 I see no reason why this could not also occur in adults if the carious process is sufficiently chronic to result in periapical granuloma formation. In this particular case, I wonder whether evidence for such predisposing factors was sought. Did physical examination of the oral region detect deep caries? Did dental radiographs, not merely jaw films, demonstrate periapical radiolucencies in one or more teeth in the left posterior mandible where the recent tumor developed? Additionally, is it known whether the patient had extractions of any premolars or molars in that area within 2 years or so before the jaw mass developed? Extractions would permit salivary EBV to enter the exposed tooth sockets to their depths more directly and contact any lymphoid tissue that was not removed with the infected teeth.

Speaking in general terms, it is possible that the achievement and maintenance of proper oral hygiene, the filling of carious teeth, and treatment of inflamed periodontium that would be of value in all individuals might be unusually important not only before chemotherapy to minimize mucositis but thereafter. In the ever-increasing population of individuals successfully treated by chemotherapy, continuous proper dental care might minimize the risk of Burkitt's jaw tumor development should EBV be present or subsequently introduced into their oropharyngeal regions.

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REFERENCES


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