1:16 IgA antibody (62% of case patients vs. 50% of control patients; P = .17) and IgM antibody (10% vs. 0%; P = .02). However, the relationship between AAA and the presence of IgA antibodies was attenuated after adjustment (adjusted OR, 1.17).

We found that serological evidence of past infection with *C. pneumoniae* is positively associated with AAA. However, *C. pneumoniae* infection is uncertain. Nevertheless, these findings may lead to important insights into the etiology and pathophysiology of AAA. Infection of aortic tissue by other infectious agents, chiefly Treponema pallidum, has long been recognized as a cause of AAA. In vitro and animal model studies have shown that the arterial system is a potential target tissue for *C. pneumoniae* infection [8, 9], and at least 2 studies have demonstrated that AAA tissue often harbors evidence of infection [5, 6]. In addition, a recent study discovered *C. pneumoniae*–specific T lymphocytes among in vivo activated cells from AAA tissue, suggesting that *C. pneumoniae* might be more than an “innocent bystander” [10]. Further research into the possible etiologic role of *C. pneumoniae* in aortic aneurysm is warranted.

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Possible Malignant Transformation of Benign Lymphoepithelial Parotid Lesions in Human Immunodeficiency Virus–Infected Patients: Report of Three Cases

Benign lymphoepithelial parotid lesions (BLL) are intraparotid pathological changes that are commonly thought to be an early manifestation of human immunodeficiency virus (HIV) infection. It is not well known whether BLL may undergo malignant transformation into B cell lymphoma and may therefore be a sort of pre-cancerous lesion. We report 3 cases of possible malignant transformation of BLL in HIV-infected patients.

Benign lymphoepithelial parotid lesions (BLL) are frequently seen in patients infected with human immunodeficiency virus (HIV) and are easily diagnosed by means of ultrasonography, given the typical presence of glandular cysts with thick internal septa [1]. It is possible that these lesions represent a favorable prognostic factor as regards HIV-disease progression, both in children and adults [2, 3]. Furthermore, they may act as a viral reservoir, similar to peripheral lymph nodes [4]. It is not clear, however, whether BLL may undergo malignant transformation into non-Hodgkin’s lymphoma (NHL) and therefore represent a sort of premalignant lesion. Here we report 3 cases of HIV-infected patients in whom ascertained or presumable BLL preceded the occurrence of NHL.

A 61-year-old man was treated as an outpatient at our clinic in January 1996 because of indolent swelling of the left parotid gland with xerostomia. His CD4+ cell count was 191 cells/μL and his CD8+ cell count was 1464 cells/μL. An ultrasonographic study showed the presence of cysts compatible with benign lymphoepithelial parotid lesion (BLL; figure 1A), and a fine-needle aspiration under sonographic guidance (advanced to the

References
edge of the solid component) demonstrated BLL (figure 1B). In December 1996, he was admitted to our department because of further parotid enlargement with evident physical modification of the gland, which appeared tender and adherent to the subcutaneous tissue. An ultrasonographic examination demonstrated a solid parotid mass (figure 1C). A surgical biopsy showed high-grade non-Hodgkin’s lymphoma (NHL; figure 1D). The patient was given 4 courses of antileukemic therapy with cyclophosphamide, vincristine, and prednisone. However, despite an abatement of the parotid enlargement, his general condition worsened, and he died in June 1997.

A 35-year-old woman with known HIV seropositivity since 1987 attended our outpatient clinic in November 1996 because of an indolent swelling of the right parotid gland and xerostomia. Ultrasonography showed cystic lesions typical of BLL. No definite diagnosis could be made because the patient refused to undergo a biopsy. At that time, her CD4+ cell count was 191 cells/μL, and her CD8+ cell count was 1064 cells/μL.

Six months later, because of the worsening of the parotid lesion, she underwent fine-needle aspiration under sonographic guidance, which demonstrated high-grade NHL. She was given 5 courses of chemotherapy with cyclophosphamide, vincristine, and prednisone, with subsequent improvement of clinical conditions. As of September 1999, she was well, with no evidence of NHL recurrence.

A 26-year-old woman with known HIV seropositivity since 1985 presented with indolent swelling of the right parotid gland and xerostomia. She underwent surgical biopsy that showed...
BLL. At that time, her CD4+ cell count was 195 cells/µL, and her CD8+ cell count was 1635 cells/µL. Six months later, because of the enlargement of her left parotid gland, she underwent percutaneous biopsy, the results of which showed evidence of high-grade B-cell lymphoma. Consequently, she was given 4 courses of antitumor therapy with cyclophosphamide, vincristine, and prednisone, with good clinical response. The patient died 4 years later with a presumptive diagnosis of primary cerebral lymphoma. No autopsy was performed.

In 1990, Itescu et al. [5] first described a clinical syndrome called diffuse infiltrative lymphocytosis syndrome (DILS), which was characterized by parotid enlargement, persistent circulating CD8 lymphocytosis, and the expression of certain antigens of the major histocompatibility complex (MHC).

All 3 of our cases had CD8 lymphocytosis and clinical findings consistent with DILS, such as parotid enlargement and xerostomia. Our report raises an important question: can BLL be considered a premalignant lesion in HIV-infected patients? In the first case, 2 histologic examinations were performed almost 1 year apart; the first was negative for malignant cells, whereas the second demonstrated high-grade B-cell lymphoma. The malignant transformation was accompanied by a change in both clinical and sonographic appearance. In the second case, we cannot rule out the presence of NHL since the first observation. However, there are 2 factors to be considered: (1) physical characteristics of the enlarged gland changed over time, and (2) at first examination, sonography showed a textural pattern consistent with BLL. The third case is questionable, since there was clinical and histologic evidence of BLL, and then, 6 months later, a diagnosis of high-grade NHL was made following a needle biopsy of the opposite parotid gland. Obviously, we cannot exclude the presence of 2 independent clinical events that are simply temporally related. In our opinion, HIV patients with BLL should undergo ultrasonography and, possibly, histologic examination in comitance with any physical modification of the parotid lesions. Improved survival in HIV-infected patients—due to highly effective antiretroviral therapy—can conceivably increase the incidence of tumoral diseases. Therefore, further studies are needed in order to better define the exact role played by BLL in favoring the occurrence of B cell lymphomas in such patients.

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Peripherally Inserted Central Catheters in Patients with AIDS Are Associated with a Low Infection Rate

We reviewed the medical records of all human immunodeficiency virus (HIV)-infected patients who had a peripherally inserted central catheter (PICC) placed during a 1-year period. Ninety-seven PICCs were inserted in 66 patients for 8337 catheter-days. Eighty of 97 catheters were used primarily to treat cytomegalovirus disease. The mean time to any complication was 150 days. The total infection rate was 6.1 per 1000 catheter-days. The total infection rate was 1.3 per 1000 catheter-days, and the serious infection rate was 0.8 per 1000 catheter-days. The mean time to a serious infection was 310 days. The non-infectious complication rate was 4.6 per 1000 catheter-days. PICCs were associated with a low infection rate and a moderate mechanical complication rate, which compare favorably with historical rates seen in AIDS patients with other types of central venous access devices. PICCs are a reasonable alternative to other central venous access devices in patients with HIV or AIDS.

Patients with AIDS require long-term central venous access devices for iv therapy. Several types of central venous access devices are available, including percutaneously placed, nontunneled nonimplantable central venous catheters (NT-CVCs), peripherally inserted central catheters (PICCs), tunneled cuffed catheters (i.e., Hickman or Groshong type), and fully implantable devices (i.e., Mediport or Port-a-cath type). However, these devices are associated with substantial morbidity when used in...