A literature search with no date limits in PubMed using the keywords "lymphomatoid granulomatosis" and "larynx" found only two reported cases where LG affects the larynx, but in both of them, laryngeal disease was associated with pulmonary involvement.[2,3] The larynx has its own lymphatic structure called larynx-associated lymphoid tissue (LALT). In the subglottis, LALT is replaced throughout life by a diffuse infiltration of strong intensity consisting predominantly of CD3-positive T lymphocytes with scattered CD20-positive B cells,[4] therefore LG could be originated in subglottic tissues.

LG pathogenesis is unclear, but it has been linked to EBV and immunodeficiency. As regards the close to 100% EBV association with LG and the presumed wide expression of EBV latent encoded proteins, it has been strongly inferred that EBV is not just an innocent bystander in the pathogenesis of LG.[5] When dealing with immunodeficiency, it has been proven that most patients with LG have defects in cytotoxic T cell function. That would explain how LG is less rare in many immunodeficiency states.[6] In our case, CD4 count cell was 124 cell/μl due to a good treatment compliance with discordant response.

Many studies have analyzed the connection between LG and AIDS. HIV infection is associated with an increased risk of lymphomas by 60-165 fold even in the combined antiretroviral therapy era. Excellent outcomes with infusion therapy and concurrent rituximab have been reported in the treatment of some of them.[7]

We report that laryngeal LG can mimic Reinke's edema in its early stages. In our own experience, Reinke's edema should not be treated as a casual finding in patients with fever of unknown origin or immunodeficiency.

Our current case was classified as grade III of LG, which is histologically considered as diffuse large B-cell malignant lymphoma,[8] but the first biopsy was reported as polymorph lymphoid proliferation related to a post-transplant lymphoproliferative disorder (PTLD), which can simulate a lower grade of LG. LG and PTLD are associated with immunodeficiency and are driven by EBV. Histopathologically, they present a morphological spectrum spanning polymorphic through monomorphic lymphoid proliferations.[9] The distinctness of LG and PTLD has been emphasized by the difference in the immune response. While LG has a large population of background T-cells, PTLD is recognized by a poor T-cell enviroment.[5]

A better awareness of LG in recent years is at present allowing new therapeutic tools for this disease to be developed. Rituximab, a new monoclonal antibody anti-CD20 has shown promising results in some cases of LG with pulmonary involvement.[10]

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Comparison of Two Serological Tests for the Identification of Recent HIV Infection: Vironostika HIV-1 Microelisa and BED Capture Enzyme Immunoassay

Comparación de dos tests serológicos para la identificación de infecciones recientes por VIH-1: microelisa Vironostika HIV-1 y enzimoinmunoensayo de captura BED

To the Editor:

Identification of recent human immunodeficiency virus (HIV) infection is an important tool for monitoring HIV transmission. During the last few years, several serological assays have been developed for this purpose and used in cross-sectional studies.[1] The serological testing algorithm for recent HIV seroconversion (STARHS) was developed in 1998[2] and used with the Abbott HIVAB 3A11 assay (Abbott Laboratories, Abbott Park, Chicago, Illinois, USA) and with the Vironostika HIV-1 Microelisa System (bioMérieux SA, Marcy l'Etoile, France). The sensitivity of both assays was lowered in order to obtain a negative result in specimens with low antibody titers, such as those from individuals with a recent infection. These assays are no longer available, and laboratories have turned to new methods. The BED assay (Calypte Biomedical Corporation, Portland, Oregon, USA) measures anti-HIV IgG titers[3] and includes a calibrator to ensure comparability of results. Furthermore, the BED assay is included in an external quality program offered by the Centers for Disease Control and Prevention (CDC, Atlanta, Georgia, USA).

In Catalonia, the STARHS was introduced using the Vironostika assay as part of the enhanced HIV/STI surveillance program in 2003. As the Vironostika assay has no longer been available since 2007, our laboratory changed to the BED assay in 2008. Our aim was to assess whether the results obtained by both techniques were comparable.

Table 1
Performance of both assays according to clinical information.

Group	Total no. of patients	No. of recent infections according to assay		
		Vironostika	BED	Both assays
Known recent HIV infection	15	14	14	13
AIDS	45	12	9	7
Long-standing HIV infection	3	0	0	0
Unclassified[a]	38	9	11	9
Total	101	35	35	29

[a] Patients with no clinical criteria for AIDS, without evidence of recent infection, neither long-standing infection.

A total of 101 serum specimens from HIV-1–positive individuals were selected from a previous study.[4] The selection criteria were sufficient sample volume and minimum available clinical and laboratory information ($CD4^+$ T-cell count, HIV viral load, HIV infection stage, previous antiretroviral treatment). Patients were classified into three groups. The first group included 15 recently infected individuals (sera were drawn no more than 6 months after seroconversion in nine patients, and the rest had a diagnosis of acute HIV infection). The second group included three patients with long-standing infection (subjects infected for >12 months) and 45 patients with a diagnosis of AIDS (clinical criteria or $CD4^+$ T-cell count under 200 cells/μl). Finally, 38 specimens were unable to be classified as either recent infections, long-standing infections, or AIDS.

The agreement between Vironostika and BED assays was good (κ=0.738, P<.005), which is consistent with the results of two published studies.[5,6] Sensitivity to detect recent infection was 93.3% (95% CI: 68.1 – 99.8) for both the Vironostika and the BED assays. Specificity for detecting long-term infections was 75.0% (95% CI: 60.4 – 86.4) using the Vironostika, and 81.3% (95% CI: 67.4 – 91.1) using the BED. Positive predictive values were 60.9% (95% CI: 38.5 – 80.3) using the BED, and 53.8% (95% CI: 33.4 – 73.4) using the Vironostika. Negative predictive values were 97.5% (95% CI: 86.8 – 99.9) using the BED, and 97.3% (95% CI: 85.8 – 99.9) using the Vironostika. Table 1 shows the samples identified as recent infections by both BED and Vironostika according to their clinical characteristics and laboratory information.

The BED assay correctly classified a greater proportion of recent infections and patients with AIDS than Vironostika. These results are similar to those of a previous study,[6] in which the Vironostika kit also tended to misclassify more individuals with long-standing infections or AIDS as recently infected in a comparison with the avidity index method.[7] The misclassification of patients with AIDS or $CD4^+$ T-cell counts $\leq$200 cells/μl is explained by the low anti-HIV IgG titers. Hence the importance of excluding those samples belonging to patients fulfilling these criteria from STARHS testing when this information is available.

The BED assay offers several advantages over the Vironostika: i) it has been reported to have better reproducibility, since it is based on the HIV IgG/non-HIV IgG ratio and uses a simple 1:100 dilution;[8] ii) the BED assay can also be automated providing more precise results than the Vironostika assay, which is performed manually; and iii) the window period of the Vironostika assay differs for B and non-B HIV-1 subtypes, whereas these differences are less pronounced with the BED.[9] For all those reasons, the BED assay offers a good alternative to the discontinued Vironostika assay.

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Appendix.

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Enfermedad meningocócica invasiva por *Neisseria meningitidis* del serogrupo Y

Invasive meningococcal disease due to Neisseria meningitidis *serogroup Y*

Sr. Editor:

Los aislados de *Neisseria meningitidis* del serogrupo Y son muy poco frecuentes en nuestro medio. Se presenta un caso de bacteriemia y neumonía causada por este microorganismo.

Varón de 17 años, natural de Bolivia con residencia en España desde hace 5 años, que acude a urgencias en Marzo de 2010. Al ingreso presentó cefalea, congestión nasal, rinorrea verdosa y fiebre de hasta 38,8 °C de tres días de evolución. En la exploración física el paciente estaba bien orientado, sin signos meníngeos ni lesiones cutáneas. La presión arterial era de 100/66 mmHg, no presentaba afectación cardiocirculatoria y la radiografía de torax inicial no mostró nódulos ni consolidaciones. El hemograma de urgencia mostraba 16800 leucocitos/mm^3; el 90,3% neutrófilos, 3,5% linfocitos, 0, 2% eosinófilos y 323000 plaquetas/mm^3. La PCR era de 3.27 mg/L. Como antecedentes personales destacaba haber sido sometido a una amigdalectomia un mes antes del ingreso.

Se realizó una punción lumbar que dio salida a un líquido claro y acelular, con tinción de Gram en la que no se observaban ni células ni bacterias, cuyo cultivo finalmente resultó negativo.

A su llegada a planta, unas horas más tarde, el paciente se encuentra estable, y se procede a la extracción de hemocultivos. El paciente presentaba tos productiva y esputo herrumbroso, objetivándose en una nueva radiografía de tórax una afectación parenquimatosa de tipo alveolar en el lóbulo inferior izquierdo. Por lo que se inició antibioterapia con levofloxacino.

A las 28 horas de incubación, en los hemocultivos se aisló *N. meningitidis* serogrupo Y, genosubtipo VR1:5-1; VR2: 10-4, sensible a cefotaxima (concentración inhibitoria mínima 0,003 mg/l), meropenem, ciprofloxacino, levofloxacino, cloranfenicol y rifampicina.

Tras interrogar a la familia se confirma que el paciente había completado el calendario vacunal de la Comunidad Autónoma de Madrid, incluida la vacuna frente a meningococo C.