is the most frequent, while other subtypes, such as angioimmunoblastic and anaplastic large cell lymphoma (ALCL) are less common. Less than 40 cases of ALCL have been reported in HIV-infected patients.[6,7]

ALCL was first characterised by Stein, who described a new type of lymphoma consisting of large anaplastic lymphoid cells with a strong expression of CD30, and a tendency to grow cohesively and invade lymph node sinuses.[8] As in our case the common type is characterised by sheets of large lymphoid cells with horseshoe-shaped nuclei containing multiple nucleoli. Tumour cells have an abundant cytoplasm with vacuoles and an increased Golgi region. Most cases of ALCL express T-cell markers. The CD3 complex (TCR) is one of the most commonly expressed T-cell antigens, whereas unlike that of our patient CD4 or CD8 expression is less common.[9] Some ALCLs are associated with a 2;5 chromosomal translocation encoding the tyrosine kinase anaplastic lymphoma kinase (ALK).[10] It is assumed that both CD30 and ALK are involved in the growth and replication of the tumour cells.[7]

ALCL in HIV-infected patients has a distinct course, being much more aggressive than in immunocompetent patients. Although it is usually associated with extranodal involvement and systemic symptoms,[5] presentation with rapidly appearing painful subcutaneous nodes, as in our patient, is very rare. Two clinical forms of ALCL have been described, systemic and cutaneous.[7] Although the skin may be involved in both forms, in systemic cases the hypodermis is affected, but characteristically the dermis is preserved. This was the pattern in the case reported here. As in this case, ALCL tends to affect patients with severe immunodepression, and contrary to cases in non-infected population, rarely expresses ALK, which is associated with better responses to chemotherapy.[4,9] Immune reconstitution by HAART is crucial. Anti-neoplastic regimens are frequently considered, but the prognosis is poor, with a median survival of 5 months.[5,10]

Although uncommon, clinicians caring for patients with HIV infection should be aware of this tumour, especially in patients presenting with swollen lymph nodes, or subcutaneous nodes. Since it affects patients with severe immunodeficiency, and has an ominous prognosis, good control of HIV infection and subsequent immunodepression is the best preventive strategy.

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Are HIV-infected patients a high-risk population for hepatitis E virus infection in Spain?

¿Son los pacientes VIH positivos un grupo de riesgo para la infección por virus de la hepatitis E en España?

To the Editors,

We read the article by Rodriguez-Frías et al. [1] with interest. The authors reported a seroprevalence of anti-HEV antibodies (IgG anti-HEV) between 2.2% and 7% in Spain. However, the prevalence of anti-HEV antibodies varies according to the population included in the study, and is even much higher in HIV infected patients. Data on the frequency of anti-HEV antibodies in these patients in Spain are scarce, and it is a controversial issue in other countries, such as England where Feane et al.[2] reported a similar seroprevalence in controls and patients with HIV infection.

Therefore, we tested 178 plasma samples from 178 HIV-infected patients who attended our Infectious Disease Department for monitoring of HAART therapy between December 2011 and January 2012. Among them, 140 (78.65%) were males with a mean age of 46 years (range: 20–78). IgG anti-HEV antibodies were detected in serum by a commercial enzyme immunoassay (EIA) kit (HEV Ab, DiaPro Diagnostic Bioprobes, Milan, Italy) following the manufacturer's instructions. All positive samples were studied further for the presence of IgM anti-HEV antibodies (HEV IgM, DiaPro Diagnostic Bioprobes, Milan, Italy). A result was considered positive by both tests when the ratio of the sample optical density and the cut-off value was higher than 2. Positive results by EIA were confirmed by Western blot analysis (RecomBlot HEV IgG/IgM; Mikrogen, Martinsried, Germany). In addition, HEV RNA was amplified by reverse transcriptase (RT)-nested PCR[3] in all serum samples with IgG or IgM anti-HEV. All the patients included in this study were living in urban or the surrounding areas of Madrid. IgG anti-HEV antibodies were found in 18 out of 178 (10.11%). IgM anti-HEV antibodies were detected in 1 out of 18 IgG anti-HEV positive samples, suggesting acute or recent infection. HEV RNA was positive in a IgG anti-HEV positive patient. None of them presented clinical symptoms related to viral acute hepatitis currently or in recent years. ALT and AST were normal in all the patients who had IgG, IgM anti-HEV and/or HEV RNA in serum.

The seroprevalence of HEV infection has been studied in other probable risk groups, such as immigrants in Madrid[4] (Table 1). Our results showed similar frequencies of detection of IgG anti-HEV

Table 1
Prevalence of IgG anti-HEV among different groups of population in Spain according to Refs. 3–6.

	Number of samples	Prevalence
Blood donors	863	2.9%
Haemodialysis patients	63	6.3%
HCV post transfusionally infected children	42	0%
Sub Saharan immigrants	90	5.5%
Pregnant women	1040	3.6%
Pig handlers	113	18.6%
HIV-infected patients	178	10.11%

antibodies in all the population tested, with the exception of pig handlers[5] (18.6%) and HIV-infected patients (10.11%). In blood donors and pregnant women, the prevalence is 2.8% and 3.6%, respectively,[6,7] significantly lower than in the HIV infected population, 10.11% ($p < .01$).

Our data are similar to those reported by Jardi et al. in Catalonia,[8] Spain. They found a prevalence of 9% among 238 HIV-infected patients. However, it is interesting that in healthy adults the prevalence is much higher in Catalonia[9] than in Madrid,[4] 7% and 2.8%, respectively. This difference may be due to epidemiological characteristics of the patients studied (rural or urban areas, gender, age, profession), or methodology. The geographical situation of Madrid inside the country may also have a role, since a very high prevalence has been reported in southwest France.[10]

In summary, our results show a high seroprevalence of HEV infection in HIV positive patients different to that observed in blood donors, pregnant women, and other considered high risk groups (except pig handlers, as hepatitis E is a zoonosis and its reservoir is swine). The normal values of ALT and absence of clinical symptoms recorded in all the patients suggests that asymptomatic infection could have taken place frequently in the past in these patients, and they can be considered as a risk group for HEV infection.

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Influenza A (H1N1) complicated by invasive aspergillosis in non-severely immunocompromised patients

Influenza A (H1N1) complicada en pacientes sin inmunocompromiso severo: coinfección con Aspergilosis pulmonar

Introduction

Pulmonary aspergillosis (PA) is a disease which usually occurs in immunocompromised hosts with an overall mortality around 60%.[1] There have been some reports in the literature of invasive PA after pandemic influenza A (H1N1) infection.[2,3] We present two cases of invasive aspergillosis after influenza A (H1N1) infection in patients without classical predisposing factors.

Case report

The first patient is a 43-year old diabetic male, who was admitted to the intensive care unit (ICU) due to diabetic ketoacidosis, pneumococcal pneumonia and influenza A (H1N1). He received levofloxacin, ceftriaxone, oseltamivir and methylprednisolone (80 mg/day/1wk). On day 12, fever and respiratory failure reappeared, invasive mechanical ventilation was required. The chest computed tomography (CT) scan showed a cavity of 6 cm × 5 cm. *Aspergillus fumigatus* grew in a sputum sample and the galactomannan antigen (AGA) tested positive in BAL fluid. Intravenous voriconazole was started as well as corticosteroids due to respiratory distress syndrome. He was discharged after three months of hospitalization. The second case is a 56-year old diabetic man with alcoholic liver cirrhosis admitted to ICU due to alcoholic hepatitis, a methicillin sensitive *Staphylococcus aureus* bacteraemic pneumonia and influenza A (H1N1). He was started with oseltamivir, cloxacillin, meropenem and methylprednisolone (60 mg/day/1 wk). On day 14, the patient developed right hemiparesis. Magnetic resonance imaging showed various parenchymatous lesions suggestive of brain abscesses. A chest CT had bilateral cavitated nodules. *A. fumigatus* grew in BAL fluid. Treatment was switched to voriconazole, anidulafungin and cloxacillin. The patient died on hospitalization day 21.

Discussion

We present two cases of influenza A (H1N1) complicated by invasive aspergillosis. These patients had none of the risk factors