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lesson of the month (1)

The ill, jaundiced patient: a triple whammy and the importance of waiting for all results

There are many causes of raised liver function tests in HIV infection. This lesson discusses a case where autoimmune hepatitis, acute hepatitis B and acute toxoplasmosis were diagnosed in a lady presenting with abdominal pain and jaundice. Oral steroids for autoimmune hepatitis may have worsened the clinical picture as her hepatitis serology was not available at the time. This lesson highlights the importance of waiting for all serology results to return in an ill jaundiced patient before deciding on active management and treatment

Lesson

A 43-year-old Somalian housewife was initially seen in the gastroenterology outpatients department in December 2006. She had moved to the UK from Denmark in 2005 where she was diagnosed as having autoimmune hepatitis. Her liver function tests (LFTs) were raised having an aspartase aminotransferase (AST) 71 IU/l, gamma glucoronyl transferase (γGT) 355 IU/l, alkaline phosphatase (ALP) 599 IU/l, albumin (Alb) 34 IU/l and bilirubin (Bil) of 11 IU/l. Smooth muscle antibody (SMA) serology was positive with a negative antimitochondrial antibody (AMA). The rest of the autoimmune screen was negative. An ultrasound suggested a slightly enlarged liver with a biopsy showing focal mild interface hepatitis, bile duct paucity, lobular chronic inflammation and mild fibrosis suggestive of primary biliary cirrhosis (PBC) and autoimmune hepatitis (AIH) 'overlap' syndrome. Ursodeoxycholic acid (UDCA) treatment was started at this time. Unfortunately, the patient did not attend follow-up and re-presented in September 2007 feeling tired but having stable LFTs. At this time, her GP had requested an HIV-1 antibody test which was positive, although a delayed referral, and attendance was made at the HIV clinic.

In January 2008, she was admitted to the acute medical ward with a two-week history of right upper quadrant pain, anorexia, nausea and jaundice. She had marked hepatomegaly but no other stigmata of chronic liver disease. Her LFTs were grossly elevated: AST 2,844 IU/l, ALP 507 IU/l, Bil 88 IU/l and Alb 25 IU/l. Her full blood count, and urea and electrolytes were normal. Viral hepatitis serology and various HIV markers were still pending and in view of her worsening clinical state and LFTs, thought by the admitting team to be due to a possible flare of her autoimmune hepatitis, oral steroids were commenced (40 mg prednisolone) along with co-trimoxazole. Her clotting, alpha fetoprotein, caeruloplasmin and alpha-1-antitrypsin levels were normal and her AST improved quickly from 3,466 to 966 IU/l. Her hepatitis B (HBV) serology came back as surface antigen positive, e antigen negative and e antibody positive. She was negative for hepatitis A, C and D (delta). Her CD4 cell count was 270 cells/μl, RNA HIV viral load was 12,623 copies/ml and her HBV DNA viral load was >300,000 copies/ml. Her steroid, co-trimoxazole and UDCA were stopped immediately and she was sent home after her clinical function improved and LFTs stabilised further.

In the HIV clinic a few days later she was commenced on tenofovir (TDF), emtricitabine (FTC) and efavirenz (EFV). Further results showed positive toxoplasmosis immunoglobulin M (IgM) with a normal computed tomography brain scan. All her other baseline HIV markers were normal. The patient continues to do well with her LFTs now completely normalised and her HIV and HBV viral loads being undetectable. Her antiretroviral regimen was switched to atripla (TDF, FTC, EFV combination) which she continues to tolerate well.

Discussion

It was interesting to see the diagnoses of a possible acute HIV seroconversion illness, acute HBV infection, PBC/AIH 'overlap' syndrome and acute toxoplasmosis infection presenting with jaundice and raised LFTs. The rapid resolution of the clinical picture with oral steroids suggests a non-infective hepatitis or possibly the natural course of acute HBV infection, the latter of which may have been worsened by steroid therapy. The LFTs only normalised for the first time when the patient was commenced on antiretroviral therapy which also acted against HBV and the relationship therefore between the prior documented PBC/AIH syndrome and latter HIV infection is somewhat blurred.

Toxoplasmosis and HIV seroconversion illness does not tend to affect the liver and AIH is rarely seen in HIV infection.[1] AIH can be seen as part of immune reconstitution in HIV infection[2] where the response is thought to be against pathogens or autoantigens.[3] Viral hepatitis co-infected with HIV can cause autoimmune manifestations and extrahepatic manifestations of hepatitis B are well documented.[4] The pathogenic role of the replicating virus possibly via immune complexes is thought to be the responsible mechanism and does not tend to respond to traditional immunosuppressive and steroid therapy.[5]

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References

1 Puius YA, Dove LM, Brust DG, Shah DP, Lefkowitch JH. Three cases of autoimmune hepatitis in HIV-infected patients. *J Clin Gastroenterol* 2008;42:425–9.
2 O'Leary JG, Zachary K, Misdraji J, Chung RT. De novo autoimmune hepatitis during immune reconstitution in an HIV-infected patient receiving highly active antiretroviral therapy. *Clin Infect Dis* 2008;46:e12–e14.
3 German V, Vassiloyanakopoulos A, Sampaziotis D, Giannakos G. Autoimmune hepatitis in an HIV infected patient that responded to antiretroviral therapy. *Scand J Infect Dis* 2005;37:148–51.
4 Woitas RP, Stoschus B, Terjung B *et al.* Hepatitis C-associated autoimmunity in patients coinfected with HIV. *Liver Int* 2005;25:1114–21.
5 Trepo C, Guillevin L. Polyarteritis nodosa and extrahepatic manifestations of HBV infection: the case against autoimmune intervention in pathogenesis. *Autoimmun* 2001;16:269–74.

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LESSON OF THE MONTH

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lesson of the month (2)

Delivery of safe and effective care out of hours: the impact of the shared clinical record on a patient's out-of-hours contact with specialist palliative care

Access to adequate clinical information is essential for out-of-hours palliative care teams and general practitioners, specific examples to illustrate and justify this need are surprisingly rare in the medical literature. Without access to the full clinical background the patient in this lesson may have been inappropriately admitted to a palliative care unit and delayed investigations would have misguided the admitting doctor's assessment, planned investigations and management.

Lesson

At 20.00 on a Saturday evening the on-call palliative medicine registrar received a telephone call from a GP requesting admission for a patient to a palliative care inpatient unit. The GP was not the patient's usual physician and therefore only had limited background clinical information available. He reported that the patient was a 52-year-old woman known to have breast cancer who was being managed 'palliatively'. He had no further information about the extent of her disease. The patient had, however, informed him that she recently had undergone chemotherapy and was known to the local oncology centre.

The current problem was nausea and vomiting (which was not controlled on oral metoclopramide), poor appetite and dehydration. The family had telephoned the oncology centre advice line and were told that the nausea and vomiting were unlikely to be related to the recent chemotherapy.

The patient had expressed a preference to be admitted to hospital rather than managed at home and the family had asked the GP to arrange admission specifically to the palliative care unit. The GP was therefore requesting admission for symptom management and evaluation of any reversible cause of the vomiting, such as hypercalcaemia.

The registrar discussed the case with the on-call consultant and agreed to admit the patient. In the interim the consultant remotely accessed the shared electronic oncology and palliative care record to see if any further background information was accessible.

On accessing the Cancer Network Information Cymru (CANISC) record, key further clinical information became apparent. Firstly, the patient did not have advanced disease but had been staged as T2N1MO after a mastectomy and axillary

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