A pain in the neck

A 47-year-old male, born in India, with genotype 3a chronic hepatitis C virus infection, a responder-relapser to 24 weeks of pegylated interferon and ribavirin therapy, successfully underwent elective liver transplantation (LT) for hepatocellular carcinoma (two lesions; 50 mm, Segment VIII, and 26 mm, Segment V) and synthetic dysfunction (MELD 8). He received a heart beating donor graft; his immunosuppression regimen consisted of tacrolimus and a reducing dose of oral corticosteroids. Microscopy of the explanted liver is shown in figure 1A. The patient underwent a 12-month protocol liver biopsy to assess effects of the hepatitis C virus infection (figure 1B). Two months following the liver biopsy, the patient presented with fever and progressive dyspnoea. Imaging studies revealed a large left pleural effusion with left lower lobe consolidation. Pre-tracheal, pre-carinal, and bilateral anterior small sub-diaphragmatic lymph nodes were also enlarged. Ten days after presentation, a large (8 cm) left sub-mandibular mass developed (figure 2).

**QUESTION**
What do the liver biopsies show and what is the unifying diagnosis?

See page below for answer

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**Figure 1** (A) Native-hepatectomy specimen (H&E staining, original magnification 400×), and (B) allograft liver biopsy specimen at 12 months (diastase treatment/periodic acid-Schiff reagent, with haematoxylin counter-stain, original magnification 200×).

**Figure 2** Left-sided sub-mandibular mass.
ANSWER

From question on see above

Microscopy of the explanted liver demonstrates granulomata without demonstrable microorganisms. Subsequent microscopy of a fine-needle aspiration specimen from the submandibular mass demonstrated acid fast bacilli. The unifying diagnosis therefore, is activation of latent mycobacterium tuberculosis (MTB) infection post LT.

Reactivation of MTB infection remains the commonest cause of clinically manifested TB after solid organ transplantation. Management of reactivated TB after LT is difficult because anti-TB drugs can be both hepatotoxic and able to interact with immunosuppression. There is no gold standard for diagnosing latent TB. The tuberculin skin test is commonly used in screening but lacks sensitivity, especially in the immunosuppressed. It also lacks specificity because of cross-reactivity with environmental mycobacteria and BCG. Newer interferon-γ release assays have been used in LT recipients and work by detecting the proliferative response of peripheral lymphocytes to specific MTB antigens. These assays’ greater specificity for MTB infection predict better development of TB than do tuberculin skin test results, with no loss of sensitivity. As ‘positive’ interferon-γ releases assays, test results do not distinguish between latent and active MTB infection for which chemotherapy regimens differ; a ‘positive’ result mandates assessment for active MTB. Approximately half the TB cases seen in the UK are extra-pulmonary, so knowledge of the features of extra-pulmonary TB is important.

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REFERENCES

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