Splenic calcification in systemic lupus erythematosus

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DESCRIPTION

A 39-year-old woman with known systemic lupus erythematosus (SLE) nephropathy, antiphospholipid syndrome and chronic renal failure was evaluated for renal transplant. She was asymptomatic; nevertheless, the abdominal ultrasonogram showed splenic calcification (figure 1). The chest radiograph showed faint lesions suggestive of (L) hypochondrial calcification (figure 2). The tuberculin skin test result revealed induration of 3 mm. She had no history of treatment for tuberculosis or brucellosis. The angiotensin-converting enzyme was normal at 19 U/L (normal range 29–112 U/L). A CT scan of the abdomen and pelvis showed a bulky spleen that contained numerous small and differently sized smooth calcific foci probably related to granulomatous disease. No calcification was seen in the liver (figure 3A, B). There were no definite or suspicious pulmonary nodules seen on a whole body fluorodeoxyglucose positron-emission tomography (PET) scan. Similarly, numerous tiny splenic calcifications with no abnormal metabolic activity were detected (figure 4). The haemoglobin level was 11.5 g/dL, white blood count was 7.1×10^9/L, platelet count was 244×10^9/L and erythrocyte sedimentation rate was 22 mm/hour. Results of the sickling test, HIV, brucella serology and hydatid and schistosoma serology tests were all negative. She was given a pneumococcal vaccine and is awaiting a deceased donor kidney transplant.

Although splenomegaly is detected in 9%–18% of patients with SLE, splenic calcification has been rarely described in such patients.1 Splenic calcification may precede autosplenectomy and hyposplenism, emphasising the importance of

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Figure 1 Ultrasound abdomen showing multiple small hypechoic shadowing throughout the splenic parenchyma.

Figure 2 Chest X-ray with faint (L) hypochondrial calcification.

Figure 3 CT scan showing spleen that contains numerous small different size smooth calcific foci.

Figure 4 Positron emission tomography scan showing splenic calcification without abnormal metabolic activity.
pneumococcal vaccination in such patients. The pattern of calcification is so distinctive that it could hint at the diagnosis of SLE. There are usually widely distributed small, rounded, calcific lesions scattered throughout the spleen. Conversely, the antiphospholipid syndrome detected in our patient can predispose patients to segmental splenic infarction rather than diffuse calcification. The exact explanation why this calcification occurred only in the spleen is not clear. There are suggestions it could signify calcification in the ‘onion-skin nodules’ which is regarded as a pathognomonic histopathological feature of SLE.2 Brucellosis, tuberculosis and hydatid disease are endemic to this area; although unique numerous, thin, target-like calcifications in the spleen have been described in brucellosis, calcifications in tuberculosis are more widespread and involve the liver and lymph nodes.3

Contributors FEE diagnosed the patient condition and followed her up in the clinic, written the manuscript and approved the final version and was fully responsible for the accuracy and integrity of the article. SFE did the literature search and contributed to the writing and revising the manuscript, final approval of the version for publication and agreed to be accountable for the accuracy and integrity of the article. RA, the infectious diseases fellow, was following the patient in the clinic in addition to preparation of the images. He revised and approved the final version of the manuscript and agreed to be accountable for the accuracy and integrity of the article.

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