Thus anakinra therapy is warranted in patients with inflammatory disorders associated with mutation in *CIAS1*, but physicians must be aware of the increased risk of infection associated with this drug.

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[^{18}F]FDG-PET of giant-cell aortitis

Sir, A 74-yr-old woman was admitted to hospital with a 6-month history of weakness, decreasing appetite, vomiting, abdominal pain and weight loss of 14 kg. Laboratory studies showed that the white

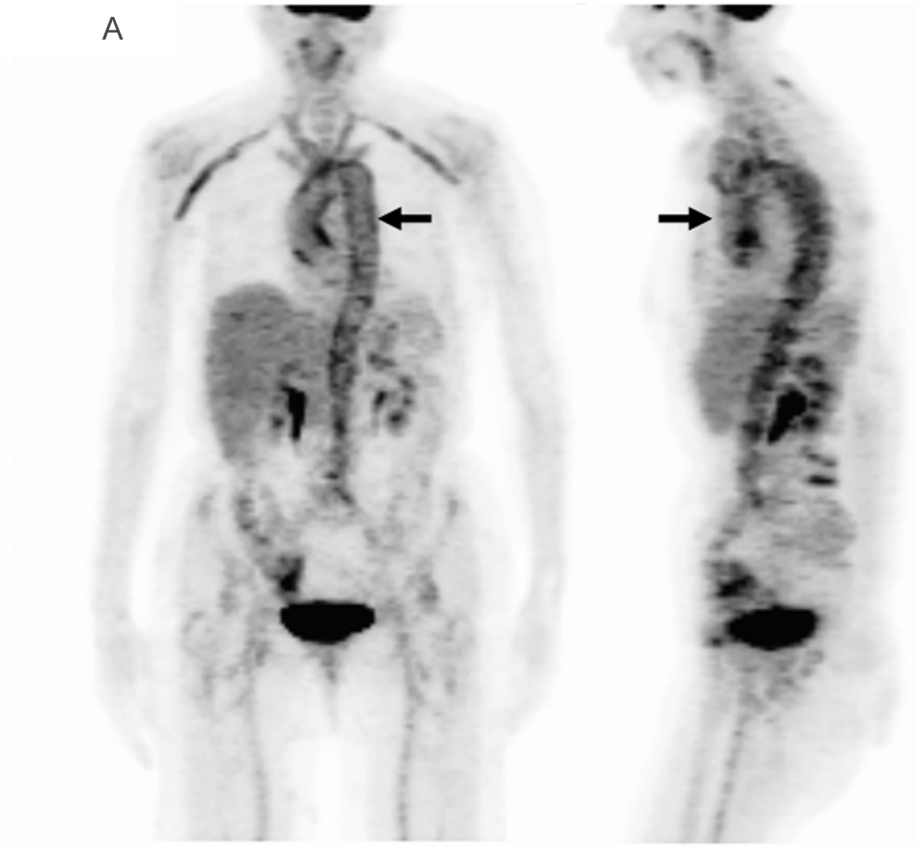

Fig. 1. FDG-PET of a giant cell arteritis patient, which clearly demonstrates pathologically elevated glucose uptake within the vessel wall of the whole aorta (arrow).

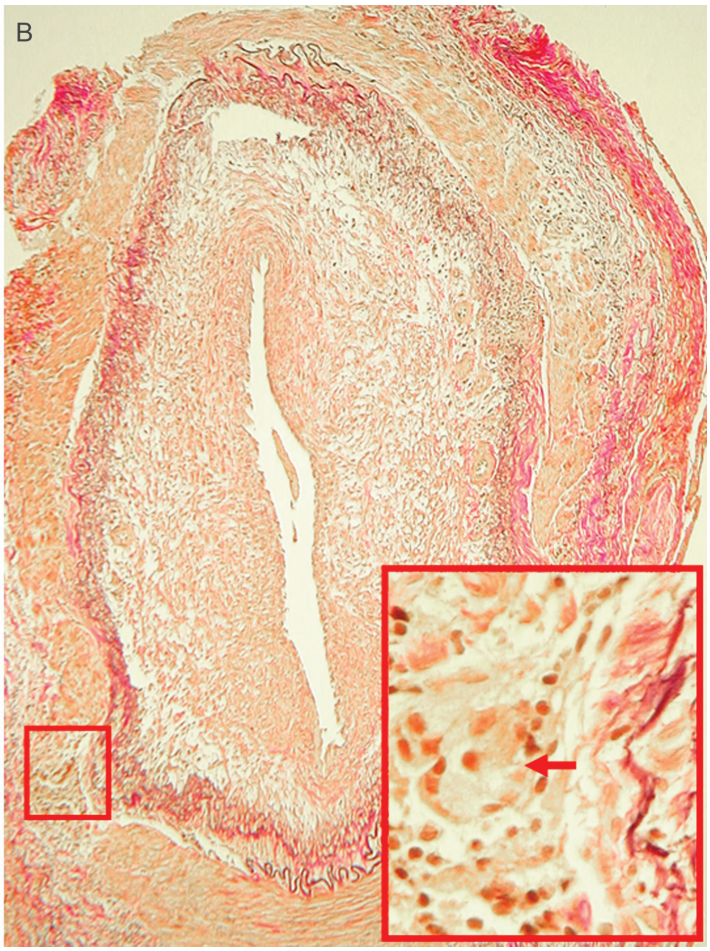

FIG. 1. Continued. Histological confirmation of giant cell arteritis, by finding of giant cell infiltration (arrow) with thickening of all temporal artery layers and consecutive stenosis of the lumen.

cell count was 12 100/mm^3, the haemoglobin concentration was 9.4 g/dl, the haematocrit was 29.1%, the platelet count was 494 000/mm^3 and the C-reactive protein was 141 mg/l. Gastroscopy, colonoscopy and CT scanning showed no abnormalities.

For further evaluation, the patient was admitted for [^{18}F]FDG-PET, which clearly demonstrated pathologically elevated glucose uptake within the vessel wall of the whole aorta (Fig. 1A, arrow) and its main thoracic and abdominal branches. The diagnosis of large-vessel vasculitis was subsequently confirmed by the finding of giant cell infiltration (Fig. 1B, arrow) with thickening of all temporal artery layers and consecutive stenosis of the lumen. The patient was treated with prednisone and recovery was unremarkable.

Due to its high sensitivity, especially in the state of active inflammation [1], [^{18}F]FDG-PET might become a valuable diagnostic tool in the management of large-vessel inflammation.

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Congenital heart block associated with a maternal anti-HsEg5-like autoantibody

SIR, Congenital heart block (CHB) is a rare (1:20 000) disease, and in 70% of cases there are no coexisting cardiac malformations [1]. In this subsect, up to 90% [2, 3] are associated with, if not directly caused by, maternal autoantibodies (Abs) against SSA/SSB (cardiac manifestation of neonatal lupus erythematosus), independently of whether maternal SLE or SS is manifested. Via active placental transfer (increasing after 16 weeks' gestation), IgG antibodies (Abs) gain access to the fetal heart during a vulnerable phase (from week 16 until shortly after birth), when the main cardiac development is complete and physiological apoptotic