CASE REPORT

The clinical conundrum of diagnosing and treating systemic sarcoidosis in a high TB burden area

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SUMMARY

A 53-year-old woman from Southern India presented with weight loss, anorexia, fever and asthenia. Whole body positron emission tomography/computed tomography (PET-CT) showed fluorodeoxyglucose-avid mediastinal and abdominal lymphadenopathy with hepatic, splenic, parotid and lacrimal glandular inflammations. Endoscopic ultrasound-guided fine needle aspiration of subcarinal lymph node showed non-caseating granulomas. Initial serum ACE level was elevated but with normal calcium. Despite the suspicion of sarcoidosis, a trial of antituberculosis therapy was started empirically due to similar presentations of disseminated tuberculosis (TB) in this high endemic area. The patient subsequently deteriorated and was admitted with symptomatic hypercalcaemia. Her subsequent ACE levels were very high, supportive of a diagnosis of systemic sarcoidosis. She was given steroid pulse therapy, and 5 months later had fully recovered. This case highlights the challenges faced by physicians in high TB-endemic areas when managing granulomatous diseases as they are concerned about missing TB, the difficulties in diagnosing sarcoidosis and the role of pulse steroid therapy.

BACKGROUND

To highlight the difficulty of excluding tuberculosis (TB) and diagnosing systemic sarcoidosis in patients with granulomatous disorders in a high TB-endemic area such as the Indian subcontinent and the concerns of starting empirical systemic steroids and immunosuppressive therapy.

To highlight the fact that hypercalcaemia, raised serum ACE levels and hypercalciuria are associated with other granulomatous disorders including TB, which reduces their diagnostic value in sarcoidosis.

To highlight the importance of checking serial serum calcium and ACE levels as the levels may be normal in early sarcoidosis.

To highlight the use of combined fluorodeoxyglucose (FDG) positron emission tomography/computed tomography (PET-CT) scan as a valuable tool in the assessment of systemic sarcoidosis for diagnosis and response evaluation.

To highlight the role of endoscopic ultrasound-guided fine needle aspiration (EUS-FNA) of mediastinal lymph nodes to demonstrate the histopathological features of sarcoidosis.

To highlight the use of methylprednisolone pulse therapy in refractory systemic sarcoidosis.

CASE PRESENTATION

A 53-year-old woman from rural Southern India, with only a history of diabetes and on no regular treatment, developed progressive loss of appetite, nausea, intermittent fevers and generalised weakness. In a period of 6 months, she worsened to the state of being bed bound. On examination, she was asthenic and had a 10 cm hepatomegaly; there were no other physical signs.

INVESTIGATIONS

Pretreatment biochemistry

Bloods showed systemic inflammatory features including raised LDH-743 (normal <450 IU/L), C reactive protein 30.8 (normal <10 mg/dL) and erythrocyte sedimentation rate 44 mm/hour (0–20 mm/hour); globulins were elevated and were polyclonal. Initial corrected serum calcium was normal at 2.69 mmol/L (normal 2.3–2.74 mmol/L) but on the subsequent admission very high at 3.29 mmol/L. Serum PTH was suppressed to 5.9 pg/mL (normal 10–65 pg/mL). 1,25(OH) vitamin D assay was not available at our centre, but 25(OH) vitamin D level was normal at 40 ng/mL (normal 25–80 ng/mL). Twenty-four hour urinary calcium excretion was high at 364 mg (up to 240 mg, normal).

Serial serum ACE levels (ref 8–52 U/L)

Before steroids (on first presentation): 238 U/L.
 After antituberculous therapy (ATT) trial: 122 U/L.
 After 2 weeks of daily oral steroids: 960 U/L.
 After 8 weeks of daily oral steroids: 1900 U/L.

Liver function test profile

Pretreatment: AST 67 IU/L (8–33), ALT 31 IU/L (4–36), gamma-glutamyl transpeptidase (GGT) 297 IU/L (5–40) and alkaline phosphatase (ALP) 235 IU/L (20–130).

Post-treatment 3 months of oral prednisolone: AST 34 IU/L, ALT 34 IU/L, GGT 338 IU/L and ALP 167 IU/L.

Post-treatment six pulses of methylprednisolone: AST 40 IU/L, ALT 46 IU/L, GGT 123 IU/L and ALP 84 IU/L.

Positron emission tomography/computed tomography

Initial scan showed (figures 1–5) FDG-avid mediastinal/hilar (figures 1, 2) and abdominal lymphadenopathy (figures 1, 3), and liver and splenic enlargement (figures 1, 3) with increased uptake in both parotids (figures 1, 4) and lacrimal glands (figure 5).

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Figure 1 Maximum intensity projection image (MIP) showing increased fluorodeoxyglucose (FDG) uptake in bilateral parotid glands, enlarged mediastinal, hilar, periportal and small pelvic nodes and diffuse increased FDG uptake in the liver and spleen.

Three-month follow-up PET-CT (figures 6–10) after daily oral steroids showed significant metabolic regression with minimal morphological response in the mediastinal/hilar (figures 6, 7) and abdominal lymph nodes (figures 6, 8) with persistent high uptake in the liver, spleen (figures 6, 8), and parotid (figures 6, 9) and lacrimal glands (figure 10).

EUS-FNA of subcarinal lymph node (figure 11) showed non-caseating granulomas composed of epithelioid histiocytes and occasional multinucleated histiocytes. Lymph node biopsy TB PCR was negative.

DIFFERENTIAL DIAGNOSES

(1) Disseminated tuberculosis and (2) systemic sarcoidosis.

TREATMENT

Patient was given a trial with ATT as per the WHO standard regimen of 2HREZ/4HR as disseminated TB commonly presents in our region with these clinical features.

The patient progressively deteriorated within a fortnight after initiation of the ATT and was readmitted as an emergency when she was found to have severe hypercalcaemia. At this stage, the

Figure 3 Transaxial PET/CT showing FDG uptake in enlarged periportal lymph nodes, liver and spleen. FDG, fluorodeoxyglucose; PET, positron emission tomography.

clinical deterioration despite the ATT combined with the constellation of abnormal calcium metabolism plus parotid/lacrimal FDG PET uptake plus serum ACE level elevation and the negative PCR for TB on the node biopsy led us to safely consider a diagnosis of systemic sarcoidosis and prescribe her steroids and immunosuppressive therapy. The patient was started on oral prednisolone 60 mg/day, which gave good resolution of her acute symptoms and the hypercalcaemia. Patient was discharged on oral weekly methotrexate, prednisolone tapering therapy and hydroxychloroquine.

OUTCOME AND FOLLOW-UP

Patient was readmitted for response evaluation after 3 months of prednisolone 20 mg daily, weekly methotrexate and hydroxychloroquine; she had improved with slightly better mobility and 3 kg weight gain but still remained asthenic with low appetite. Inflammatory markers had improved and albumin increased, but alkaline phosphatase (ALP) and gamma-glutamyl transpeptidase (GGT) were still high, suggestive of persistent cholestatic hepatitis. Her calcium level had normalised, but ACE levels were considerably higher (960 and 1900 U/L, respectively) than before treatment. A follow-up whole body PET-CT was done, which showed significant metabolic resolution of lymphadenopathy consistent with the clinical response (figure 6) but persisting inflammation in the liver (figures 6, 8), spleen (figures 6, 8), parotid (figures 6, 9) and lacrimal glands (figure 10).

Figure 2 Transaxial PET/CT showing FDG uptake in enlarged paratracheal lymph nodes. FDG, fluorodeoxyglucose; PET, positron emission tomography.

Figure 4 Transaxial PET/CT showing increased FDG uptake in bilateral parotid glands. FDG, fluorodeoxyglucose; PET, positron emission tomography.

Figure 5 Transaxial PET/CT showing increased FDG uptake in bilateral lacrimal glands. FDG, fluorodeoxyglucose; PET, positron emission tomography.

The initiation of steroids and immunosuppressive drugs had given some clinical improvement after 3 months; however, the presence of persistent inflammatory activity in the liver and spleen (figure 8), the residual symptoms, the persistent cholestatic liver function tests (LFTs) and the initial response to steroids prompted the use of methyl prednisolone pulse therapy 1000 mg intravenous infusion 30 mg/kg once a week for 6 weeks,^{1–3} along with maintenance dose of steroids (10 mg oral prednisolone daily) to try to achieve full clinical remission and heal the persistent residual hepatosplenic and glandular inflammation. After the 6 weeks of pulse steroids, the patient became completely asymptomatic, had gained weight and returned to her baseline mobility, and the cholestatic LFTs had significantly improved. She was advised to continue the 10 mg prednisolone daily along with methotrexate and hydroxychloroquine.

Figure 6 Maximum intensity projection image (MIP) showing metabolic resolution of the previously noted mediastinal, periportal and pelvic adenopathy but persisting uptake in liver, spleen and parotids.

Figure 7 Transaxial PET/CT showing enlarged paratracheal node with no significant FDG avidity. FDG, fluorodeoxyglucose; PET, positron emission tomography.

DISCUSSION

India accounts for 23% of global TB incidence and the highest country incidence in numbers.⁴ Common presentations of extrapulmonary/disseminated TB include generalised lymphadenopathy and hepatosplenomegaly. Systemic sarcoidosis has similar clinical, radiological and histopathological features as that of TB.^{5,6}

This is a case that was probably consistent with systemic sarcoidosis at initial presentation (elevated serum ACE levels, parotid/lacrimal uptake in PET and non-caseating granulomas) but was cautiously treated with an ATT trial initially because of fears of missing TB or exacerbating a TB infection with steroids and immunosuppression^{7–9} due to the similarities aforementioned and the lack of specificity of supportive tests for sarcoidosis. Matters are further complicated by reports of coexistent sarcoidosis and TB or 'tuberculous sarcoidosis'/overlap cases.¹⁰

The presence of non-caseating granulomas, although characteristic of sarcoidosis, can also be seen in TB.⁶

In the tuberculosis spectrum, bilateral parotid and lacrimal involvement are rare but not unknown.^{11–16} On retrospective research, we came across the WASOG sarcoidosis organ assessment

Figure 8 Transaxial PET shows diffuse increased FDG uptake in the liver and spleen that persists but no FDG avidity in the periportal/abdominal nodes. FDG, fluorodeoxyglucose; PET, positron emission tomography.

Figure 9 Transaxial PET/CT showing persistent FDG uptake in bilateral parotid glands. FDG, fluorodeoxyglucose; PET, positron emission tomography.

instrument, which was a Delphi methodology study involving sarcoidosis experts¹⁷ in which an increased uptake in both parotid glands in PET-CT had 70% expert consensus as ‘Highly Probable’ and the rest (30%) as ‘At Least Probable’ for sarcoidosis involvement, thereby conferring the ‘positive parotid PET uptake’ a ‘High Probability’ manifestation for sarcoid involvement.

Serum ACE elevation is also known to have low specificity^{18–21} and can be increased in other granulomatous disorders including TB.²² In this patient the first ACE level was 238 U/L but dropped to 122 U/L during the ATT trial when she deteriorated with active sarcoidosis; it later increased multifold—960 and 1900 U/L during the daily oral steroid therapy when she was clinically improving; this is reflective of the low sensitivity and specificity of this test in sarcoidosis.

Serum calcium level was normal on initial assessment but subsequently increased after a few weeks; patient was also found to have hypercalciuria.^{23,24} It is important to recognise that hypercalcaemia and hypercalciuria can also be associated with granulomatous disorders including TB and therefore not specific for other granulomatous sarcoidosis.^{25–31} The WASOG sarcoidosis organ assessment instrument¹⁷ expert consensus is more than 70% (highly

Figure 10 Transaxial PET/CT showing persistent FDG uptake in bilateral lacrimal glands. FDG, fluorodeoxyglucose; PET, positron emission tomography.

Figure 11 Histopathology of lymph node biopsy demonstrating epithelioid histiocytes and inflammatory cells forming granulomas.

probable) for hypercalcaemia (or hypercalciuria) + normal PTH + normal/increased 1,25(diOH) vitamin D+ low 25(OH) vitamin D. Abnormal calcium–vitamin D metabolism is considered an end-organ sarcoid involvement.¹⁷

This case also highlights the role that whole-body PET-CT scan could play, both as a tool for diagnosing systemic sarcoidosis and for assessing the therapeutic metabolic response.^{32–40} In addition, increased PET uptake in both parotid glands has consensus by experts as highly or at least probable for sarcoidosis involvement (WASOG organ assessment instrument).¹⁷

EUS-FNA, in available centres, can be used to obtain nodal aspirates in a minimally invasive method and can demonstrate the histopathological features of sarcoidosis.^{41–45} EUS-FNA yields for sarcoidosis diagnoses could be comparable or even higher to conventional bronchoscopic methods⁴⁶ provided the lymph nodes are EUS accessible.

This patient, although had improved after 3 months with oral prednisolone, still had disabling residual symptoms and persistent hepatosplenic and glandular inflammation with persistent cholestatic hepatitic LFTs. Methylprednisolone pulse therapy—1000 mg intravenous infusion 30 mg/kg once a week for 6 weeks—resulted in complete relief of symptoms and improvement of the cholestatic LFTs. Efficacy of pulse steroid therapy in systemic sarcoidosis and refractory neurosarcoidosis has been reported.^{1–3} It remains to be seen whether the effects

of the pulse therapy in this patient are sustained or only transient.¹

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Contributors KT, LA and PR contributed to writing the clinical and pathological aspects of the report; PC contributed to the radiological aspect of the report.

Competing interests None declared.

Patient consent Obtained.

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Patient's perspective

I suffered a lot by this disease called sarcoidosis. Before the diagnosis, I could not eat and was always bed bound due to tiredness. I could feel hungry but could not eat. If I forced myself to eat, I would vomit. So, I myself reduced my eating and so had lost around 18 kg of weight in a few months. I assumed it was due to my diabetes and delayed getting doctor's help, but as I got worse I was referred to a city hospital where, after several tests, sarcoidosis was diagnosed and treatment was given. Now I am getting better and can eat well now.

Learning points

- ▶ Anti-tuberculous therapy (ATT) trial as first intervention in cases of uncertainty, as this is likely to be safer than steroids/immunosuppressive therapy for systemic granulomatous diseases. The need to develop objective guidelines for ATT trial and define its outcomes when facing similar clinical conundrums exists.
- ▶ To propose the measurement of serial serum ACE levels²¹ and serial serum calcium to increase the sensitivity as they can fluctuate in sarcoidosis.
- ▶ To check 24-hour urine calcium, as hypercalciuria (30%–50%) is more frequent than hypercalcaemia (10%–20%) in sarcoidosis.^{23 24}
- ▶ Positron emission tomography/computed tomography (PET-CT) uptake of parotids and lacrimal glands are strong indicators for a sarcoid diagnosis given their rarity in the tuberculosis disease spectrum.
- ▶ To reflect on the fact that none of the tests are very specific for sarcoidosis but, put together, can increase specificity of a sarcoidosis diagnosis. The WASOG organ assessment instrument¹⁷ could be used as a valuable tool to assign probability for each clinical manifestation as representative of sarcoidosis organ involvement.
- ▶ PET-CT could be a valuable tool in the initial diagnosis and to monitor response to treatment.
- ▶ Endoscopic ultrasound-guided fine needle aspiration in available centres could be used as a minimally invasive method to obtain aspirate samples from accessible mediastinal nodes for sarcoidosis diagnosis.
- ▶ Intravenous methylprednisolone pulse therapy 30 mg/kg can produce clinical remission in systemic sarcoidosis refractory to low-dose daily corticosteroid and immunosuppressive therapy.

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REFERENCES

- 1 Wallaert B, Ramon P, Fournier EC, *et al*. High-dose methylprednisolone pulse therapy in sarcoidosis. *Eur J Respir Dis* 1986;68(4):256–62.
- 2 Allen RK, Merory J. Intravenous pulse methyl prednisolone in the successful treatment of severe sarcoid polyneuropathy with pulmonary involvement. *Aust N Z J Med* 1985;15:45–6.
- 3 Babu K. Sarcoidosis in tuberculosis-endemic regions: india. *J Ophthalmic Inflamm Infect* 2013;3:53.
- 4 WHO. Global tuberculosis report. 2015 http://apps.who.int/iris/bitstream/10665/191102/1/9789241565059_eng.pdf.
- 5 Badar F, Azfar SF, Ahmad I, *et al*. Diagnostic difficulties in differentiating sarcoidosis from tuberculosis. *Oman Med J* 2011;26:210–1.
- 6 Litinsky I, Elkayam O, Flusser G, *et al*. Sarcoidosis: tb or not TB? *Ann Rheum Dis* 2002;61:385–6.
- 7 Kleinschmidt RF, Johnston JM. Miliary tuberculosis in a cortisone treated patient: case report with autopsy(MILIARY tuberculosis in a cortisone treated patient: case report with autopsy*). *Ann Intern Med* 1951;35:702:694.
- 8 King EQ, Johnson JB, BATTEN GS, *et al*. Tuberculosis following cortisone therapy; report of a case of rapidly progressive pulmonary tuberculosis following cortisone therapy for rheumatoid arthritis. *J Am Med Assoc* 1951;147:238–41.
- 9 Perchanok M, Bull TS. Activation of healed pulmonary tuberculosis incidental to the use of cortisone in rheumatoid arthritis. A case report. 1951;6:321–8.
- 10 Wong CF, Yew WW, Wong PC, *et al*. A case of concomitant tuberculosis and sarcoidosis with mycobacterial DNA present in the sarcoid lesion. *Chest* 1998;114:626–9.
- 11 Madhukar K, Bhide M, Prasad CE, *et al*. Tuberculosis of the lacrimal gland. *J Trop Med Hyg* 1991;94:150–1.
- 12 Thakur J, Thakur A, Mohindroo N, *et al*. Bilateral parotid tuberculosis. *J Glob Infect Dis* 2011;3:306–9.
- 13 Garg R, Verma SK, Mehra S, *et al*. Parotid tuberculosis. lung India : official organ of indian chest society. 2010;27:253–5.
- 14 Erkan AN, Cakmak O, Kayaselçuk F, *et al*. Bilateral parotid gland tuberculosis. *Eur Arch Otorhinolaryngol* 2006;263:487–9.
- 15 Sen DK. Tuberculosis of the orbit and lacrimal gland: a clinical study of 14 cases. *J Pediatr Ophthalmol Strabismus* 1980;17:232–8.
- 16 Agrawal PK, Nath J, Jain BS. Orbital involvement in tuberculosis. *Indian J Ophthalmol* 1977;25:12.
- 17 .WASOG.(<http://aasog.net/wp-content/uploads/2015/01/WASOG-Organ-Assessment-Instrument.pdf>)
- 18 Studdy PR, Bird R. Serum angiotensin converting enzyme in Sarcoidosis--its value in present clinical practice. *Ann Clin Biochem* 1989;26:13–18.
- 19 Ungprasert P, Carmona EM, Crowson CS, *et al*. Diagnostic utility of Angiotensin-Converting enzyme in sarcoidosis: a Population-Based study. *Lung* 2016;194:91–5. Epub 2015 Nov 13.
- 20 Khan AH, Ghani F, Khurshid M, *et al*. Pages with reference to book, from 131 to 133 role of serum angiotensin converting enzyme in sarcoidosis. *Journal of Pakistan medical association* 1998;48:131–3.
- 21 Studdy P, Bird R, James DG. Serum angiotensin-converting enzyme (SACE) in Sarcoidosis and other granulomatous disorders. *Lancet* 1978;2:1331–4.
- 22 Iyengar KB, Udyavara Kudru C, Kaniyoor Nagiri S, *et al*. Tuberculous mediastinal lymphadenopathy in an adult. *Case Rep Child Meml Hosp Chic* 2014;2014:bcr2013200718.
- 23 Adams JS. Vitamin D metabolite-mediated hypercalcemia. *Endocrinol Metab Clin North Am* 1989;18:765–78.
- 24 Sharma OP. Vitamin D, calcium, and sarcoidosis. *Chest* 1996;109:535–9.
- 25 Martinez ME, Gonzalez J, Sanchez-Cabezudo MJ, *et al*. Evidence of absorptive hypercalciuria in tuberculosis patients. *Calcif Tissue Int* 1993;53:384–7.
- 26 Gkonos PJ, London R, Hendler ED. Hypercalcemia and elevated 1,25-dihydroxyvitamin D levels in a patient with end-stage renal disease and active tuberculosis. *N Engl J Med* 1984;311:1683–5.
- 27 Cadranel J, Garabedian M, Milleron B, *et al*. 1,25(OH)2D2 production by T lymphocytes and alveolar macrophages recovered by lavage from normocalcemic patients with tuberculosis. *Journal of Clinical Investigation* 1990;85:1588–93.
- 28 Kuno Y, Iyoda M, Aoshima Y, *et al*. A case of tuberculous peritonitis in a hemodialysis patient with high serum soluble interleukin-2 receptor and CA-125 levels. *Intern Med* 2010;49:1783–6.
- 29 Abbasi AA, Chemplavil JK, Farah S, *et al*. Hypercalcemia in active pulmonary tuberculosis. *Ann Intern Med* 1979;90:324–8.
- 30 Chan TY. Differences in vitamin D status and calcium intake: possible explanations for the regional variations in the prevalence of hypercalcemia in tuberculosis. *Calcif Tissue Int* 1997;60:91–3.

- 31 Shek CC, Natkunam A, Tsang V, *et al.* Incidence, causes and mechanism of hypercalcaemia in a hospital population in Hong Kong. *Q J Med* 1990;77:1277–85.
- 32 Lewis PJ, Salama A. Uptake of fluorine-18-fluorodeoxyglucose in sarcoidosis. *J Nucl Med* 1994;35:1647–9.
- 33 Kobayashi A, Shinozaki T, Shinjyo Y, *et al.* FDG PET in the clinical evaluation of sarcoidosis with bone lesions. *Ann Nucl Med* 2000;14:311–3.
- 34 Nishiyama Y, Yamamoto Y, Fukunaga K, *et al.* Comparative evaluation of 18F-FDG PET and 67ga scintigraphy in patients with sarcoidosis. *J Nucl Med* 2006;47:571–6.
- 35 Love C, Tomas MB, Tronco GG, *et al.* FDG PET of infection and inflammation. *Radiographics* 2005;25:1357–68.
- 36 Bleeker-Rovers CP, de Kleijn EMHA, Corstens FHM, *et al.* Clinical value of FDG PET in patients with fever of unknown origin and patients suspected of focal infection or inflammation. *Eur J Nucl Med Mol Imaging* 2004;31:29–37.
- 37 Aide N, Allouache D, Ollivier Y, *et al.* Early 2'-deoxy-2'-[18F]fluoro-D-glucose PET metabolic response after corticosteroid therapy to differentiate cancer from Sarcoidosis and sarcoid-like lesions. *Mol Imaging Biol* 2009;11:224–8.
- 38 Imperiale A, Riehm S, Veillon F, *et al.* FDG PET coregistered to MRI for diagnosis and monitoring of therapeutic response in aggressive phenotype of sarcoidosis. *Eur J Nucl Med Mol Imaging* 2011;38:983–4.
- 39 Braun JJ, Kessler R, Constantinesco A, *et al.* 18F-FDG PET/CT in Sarcoidosis management: review and report of 20 cases. *Eur J Nucl Med Mol Imaging* 2008;35:1537–43.
- 40 Basu S, Zhuang H, Torigian DA, *et al.* Functional imaging of inflammatory diseases using nuclear medicine techniques. *Semin Nucl Med* 2009;39:124–45.
- 41 Fritscher-Ravens A, Sriram PV, Topalidis T, *et al.* Diagnosing sarcoidosis using endosonography-guided fine-needle aspiration. *Chest* 2000;118:928–35.
- 42 Walsh PR, Williams DB. Mediastinal adenopathy: finding the answer with endoscopic ultrasound-guided fine-needle aspiration biopsy. *Intern Med J* 2005;35:392–8.
- 43 Mishra G, Sahai AV, Penman ID, *et al.* Endoscopic ultrasonography with fine-needle aspiration: an accurate and simple diagnostic modality for sarcoidosis. *Endoscopy* 1999;31:377–82.
- 44 Wildi SM, Judson MA, Fraig M, *et al.* Is endosonography guided fine needle aspiration (EUS-FNA) for sarcoidosis as good as we think? *Thorax* 2004;59:794–9.
- 45 Jue TL, Sharaf RN, Appalaneni V, *et al.* Role of EUS for the evaluation of mediastinal adenopathy. *Gastrointest Endosc* 2011;74:239–45.
- 46 von Bartheld MB, Dekkers OM, Szlubowski A, *et al.* Endosonography vs conventional bronchoscopy for the diagnosis of sarcoidosis: the GRANULOMA randomized clinical trial. *Jama* 2013;309:2457–64.

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