CASE REPORT

Development of pulmonary sarcoidosis in Crohn’s disease patient under infliximab biosimilar treatment after long-term original infliximab treatment: a case report and literature review

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Abstract

Background: Inflammatory bowel disease (IBD) is chronic inflammation of the gastrointestinal tract, although its etiology has largely been unclear. Tumor necrosis factor inhibitors (TNF-I) are effective for the treatment. Recently, biosimilars of TNF-I, such as CT-P13, have been developed and are thought to possess equal efficacy and safety to the original TNF-I. Sarcoidosis is also a systemic granulomatous disease of unknown etiology. In steroid-resistant cases of sarcoidosis, TNF-I have been reported effective for achieving resolution. However, the progression of sarcoidosis due to the TNF-I also has been reported. We herein report a case of pulmonary sarcoidosis with a Crohn’s disease (CD) patient developed after a long period administration (15 years) of TNF-I.

Case presentations: A 37-year-old woman with CD who had been diagnosed at 22 years old had been treated with the TNF-I (original infliximab; O-IFX and infliximab biosimilar; IFX-BS). Fifteen years after starting the TNF-I, she developed a fever and right chest pain. Chest computed tomography (CT) revealed clustered small nodules in both lungs and multiple enlarged hilar lymph nodes. Infectious diseases including tuberculosis were negative. Bronchoscopic examination was performed and the biopsy specimens were obtained. A pathological examination demonstrated noncaseating granulomatous lesions and no malignant findings. TNF-I were discontinued because of the possibility of TNF-I-related sarcoidosis. After having discontinued for four months, her symptoms and the lesions had disappeared completely. Fortunately, despite the discontinuation of TNF-I, she has maintained remission.

Conclusions: To our knowledge, this is the first case in which sarcoidosis developed after switching from O-IFX to IFX-BS. To clarify the characteristics of the cases with development of sarcoidosis during administration of TNF-I, we searched PubMed and identified 106 cases. When developing an unexplained fever, asthenia, uveitis and skin lesions in patients with TNF-I treatment, sarcoidosis should be suspected. Once the diagnosis of sarcoidosis due to TNF-I
Background
Inflammatory bowel disease (IBD) is chronic inflammation of the entire gastrointestinal tract, although its etiology has largely been unclear. Tumor necrosis factor (TNF) inhibitors are known to be effective treatment for treating IBD patients with moderate to severe activity. The cost-effectiveness and efficacy of TNF inhibitors (TNF-I) have been demonstrated through their reduction in the rates of hospitalization and surgery [1]. Recently, biosimilars of TNF-I, such as CT-P13, have been developed and are thought to possess equal efficacy and safety to the original with dramatic cost benefits. Switching from the original to a biosimilar is thus considered an acceptable treatment [2, 3].

Similar to IBD, sarcoidosis is a systemic granulomatous disease of unknown etiology, affecting various organs, including the lung, heart, lymphatic system and skin. In many cases of sarcoidosis, steroids are effective for treatment, and in case of steroid resistance, TNF-I are reported to be effective. While some studies have reported that the administration of TNF-I caused the progression of sarcoidosis, no reports regarding the relationship between sarcoidosis and infliximab biosimilar (IFX-BS) have been published.

We herein report a case of pulmonary sarcoidosis in a Crohn’s disease (CD) patient during fifteen years administration of IFX-BS after switching from original infliximab (O-IFX). To our knowledge, this is the first case of sarcoidosis developing after switching from O-IFX to IFX-BS in a CD patient.

Case presentation
A 37-year-old Japanese woman was diagnosed with CD at 22 years of age. She had no relevant family history. At the onset of CD, she had symptoms of fever, abdominal pain, and frequent diarrhea. On total colonoscopy, she was found to have multiple longitudinal ulcers in the terminal ileum with stricture. Her symptoms were severe; thus, we administered O-IFX first, without steroid therapy. Clinical remission was obtained after 3 months of O-IFX treatment. She had maintained clinical remission without any adverse events for twelve years after the administration of O-IFX, and then O-IFX was switched to IFX-BS (CT-P13) after obtaining informed consent, because IFX-BS demonstrated equivalent efficacy and safety in the treatment of CD and the drug price was approximately half that of O-IFX in Japan. After switching to IFX-BS, clinical remission was still maintained for three years.

Fifteen years after starting the TNF-I (O-IFX and CT-P13), she developed a fever and right chest pain but had no respiratory symptoms, such as cough or sputum. Laboratory findings showed total bilirubin, 1.5 mg/dL; alanine aminotransferase, 248 U/L; aspartate aminotransferase, 105 U/L; gamma glutamyl aminotransferase, 192 U/L; alkaline phosphatase, 489 U/L; C-reactive protein (CRP), 0.44 mg/dL; anti-nuclear antibody, 1:160. Hepatitis B and C were negative. Chest X-ray and computed tomography (CT) revealed clustered small nodules in both lungs and multiple enlarged hilar lymph nodes (Fig. 1). The interferon gamma release assay and an acid-fast bacilli antibody were negative. A tuberculosis polymerase chain reaction (PCR) assay and bacterium culture of her sputum were both negative. Subsequently, a bronchoscopic examination was performed, and biopsy specimens were obtained from the middle and lower lobes of the right lung. A pathological examination demonstrated noncaseating granulomatous lesions and no malignant findings (Fig. 2). The serum angiotensin-converting enzyme and lysozyme levels were within normal limits, and soluble interleukin-2 receptor (sIL-2R) was increased to 627 U/mL (reference range 122–496 U/mL). Thus, the diagnosis of sarcoidosis was made. Her symptoms (low-grade fever and slight chest pain) could be sufficiently managed by the administration of an analgesic antipyretic. Therefore, we did not use steroids and, considering the possibility of TNF-I related sarcoidosis, TNF-I was discontinued. Subsequently, her symptoms improved gradually. In addition, the abnormal laboratory findings, including hepatobiliary enzymes, improved naturally without any treatment after one month. After having discontinued TNF-I for four months, her symptoms of sarcoidosis and the lesions detected by chest CT had disappeared completely (Fig. 3). Fortunately, despite the discontinuation of TNF-I, she has maintained clinical and endoscopic remission for 18 months after the discontinuation of TNF-I.

Discussions and conclusions
We herein report the pulmonary sarcoidosis after the long-term use of TNF-inhibitors in a CD patient.

Two possible causes were considered responsible for the development of sarcoidosis in our case: the
long-term IFX administration and the switching from the original agent to its biosimilar. To confirm the involvement of these causes and clarify the characteristics of patients likely to develop sarcoidosis under TNF-I therapy, we searched for case reports in PubMed concerning sarcoidosis due to TNF-I using the combination of the heading terms ‘infliximab’, ‘adalimumab’, ‘etanercept’, ‘certolizumab’, ‘golimumab’, ‘infliximab biosimilar’, with or without ‘Crohn’s disease’, combined with ‘sarcoidosis’. We identified 6 articles concerning 7 cases using the word combination with CD [4–9] and 65 articles concerning 99 cases using the word combination without CD (details in Additional file 1: Table 1) [10–74]. Regarding the impact of long-term TNF-I administration, in 103 cases, the median duration of developing sarcoidosis was 21 (range 1–90) months, and the maximum period was 90 months, according to previous reports. However, since those reports contained various diseases, we reviewed the seven cases with CD (excluding our own case). The median duration to the development of sarcoidosis was 24 (range 7–90) months, and the maximum period was 90 months. Our case was administered O-IFX for 144 months and then IFX-BS for 36 months. Based on the previous reports, the duration of 180 months until the development of sarcoidosis in our case was the longest, exceeding the longest period in previous reports by more than 7 years. Long-term IFX administration may affect the development of sarcoidosis, as the administration of TNF-I presumably induces cytokine imbalance (TNF-α suppression and excessive Interferon-α production) [75]. It took a very long time for the patient to develop sarcoidosis; however, it may have been a possible complication as the time to the induction of cytokine imbalance seems to show individual differences. In addition, infection is suggested to be a cause of sarcoidosis, and the treatment with TNF-I increases the risk of various infections [10–12], which can lead to the onset of sarcoidosis. Prudent follow-up should thus be performed, including observation of CD patients under long-term IFX administration. Regarding the impact of switching from O-IFX to IFX-BS, we thought that the possibility of sarcoidosis caused by changing O-IFX to IFX-BS would be quite low because of the very slight differences in the sugar chain sequences of O-IFX and IFX-BS and their almost equivalent immunogenicity [76].

Regarding other characteristics of CD patients, the median age was 30 (range 21–44) years, and the male:female ratio was 5:3 when including our case. The
causative TNF inhibitors were adalimumab (ADA) (n = 4, 50.0%, including a case switching from IFX to ADA) and IFX (n = 4, 50%, including our case). The symptoms depended on the organs involved. Our case showed an unexplained fever and chest pain because of pleuritis. Asthenia, uveitis, and skin lesions were also reported. There were no specific symptoms.

In this case, the serum ACE level was normal. It was reported that serum ACE shows low sensitivity and specificity in the diagnosis of sarcoidosis [77]. In addition, it is known that the serum ACE level is affected by genetic polymorphism [78, 79]. Clinicians should recognize that the serum ACE levels alone are not enough to diagnose sarcoidosis.

We also investigated the difference in the duration until the development of sarcoidosis under IFX and ADA therapy in the six CD cases without switching cases. The median duration to the development of sarcoidosis in CD patients under IFX (n = 3) and ADA (n = 3) therapy was 12 and 24 months, respectively. Patients receiving ADA therapy showed a longer duration until development than those receiving IFX, which might have influenced the difference in antibody type, such as chimeric human-mouse IgG monoclonal antibody and fully human monoclonal antibody. However, the number of such cases has been limited so far, so the accumulation of more cases will be required to verify whether or not differences in antibodies affects the development of sarcoidosis.

Regarding the therapy delivered after the development of sarcoidosis, six of eight cases discontinued TNF-I, two without steroid and four with additional steroid, and two cases continued TNF-I therapy while adding steroid therapy or topical therapy. All six cases who discontinued TNF-I with/without steroids showed the resolution of sarcoidosis. In one case, in which the patient continued TNF-I with steroid treatment, improvement of the lung lesion was observed. In one case in which the patient continued TNF-I with topical steroid treatment, improvement of the lung lesion, but not the skin lesion, was observed. In CD patients who develop sarcoidosis, discontinuation of TNF-I seems to be a feasible treatment. Among 107 cases with various diseases, 47 discontinued TNF-I without the addition of steroid therapy, and sarcoidosis was improved or resolved in 43 (91.5%), while 49 cases discontinued TNF-I with the addition of steroid therapy, and 46 (93.9%) showed improvement or resolution of sarcoidosis. These data support the notion that discontinuing TNF-I is effective for treating patients with chronic inflammatory disease, but the efficacy of additional steroid therapy is unclear.

Our case has maintained remission despite having discontinued TNF-I; however, TNF-I may be key drugs for CD patients. Thus, whether or not TNF-I can be re-initiated for these patients is important to clarify. Only one CD patient re-initiated the same TNF inhibitor (O-IFX) with no recurrence of sarcoidosis observed [8]. When expanding target diseases to include rheumatoid arthritis, ankylosing spondylitis, psoriatic arthritis, spondyloarthropathy, psoriasis, CD, juvenile idiopathic arthritis, SAPHO (synovitis, pustulosis, acne,
hyperostosis, osteitis), ulcerative colitis, 25 patients re-initiated TNF-I. Of 25 patients, 7 were administered the same TNF-I, and sarcoidosis consequently recurred in 4 cases (57.1%) [8, 11, 23, 27, 39, 63]. Eighteen cases started another TNF-I, and sarcoidosis recurred in 3 cases (16.7%) [13, 27, 64–74]. These data suggested that another TNF-I should be administered when re-initiating TNF-I in such patients.

In conclusion, should be aware that TNF-I can cause several drug related complications, including sarcoidosis. Sarcoidosis due to TNF-I can be caused by IFX-BS and develop even after long-term administration. Clinicians should be aware of the possibility of sarcoidosis in patients under anti-TNF therapy. The efficacy of additional steroid therapy remains unclear; however, TNF-I should be discontinued as soon as possible, even in CD patients who maintain long-term remission with TNF-I. When re-starting TNF-I, another TNF-I should be used for disease control, as relapse of sarcoidosis is frequent when patients are treated with the same TNF-I (Table 1).

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Authors’ contributions
SK and KM drafted the initial and final version of the manuscript and were involved in the care of this patient as gastroenterologists. KA and NU provided the corresponding author on reasonable request. HT were involved in the care of this patient as gastroenterologists and contributed to the intellectual review of the manuscript. SK and KM drafted the initial and final version of the manuscript and were involved in the medical management of this patient as physicians in charge. MF contributed to the critical revision for important intellectual content. All authors read and approved the final manuscript.

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Availability of data and materials
The datasets used and/or analyzed during the current study available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
This is a case report, and we did not receive any approval from the ethics review board. We obtained the patient’s written informed consent to publish the material.

Consent for publication
Written informed consent was obtained from the patient for the publication of this case report and accompanying images.

Competing interests
Dr. Fujiya reports grants and personal fees from Nippon Kayaku Co., Ltd., and Mitsubishi Tanabe Pharma Corporation as well as grants from AYUMI Pharmaceutical Corporation.

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References
1. van der Valk ME, Mangen MJ, Leenders M, et al. Health care costs of inflammatory bowel disease have shifted from hospitalisation and surgery towards anti-TNFα therapy: results from the COIN study. Gut. 2014;63:72–9.
2. Feagan BG, Lam G, Ma C, et al. Systematic review: efficacy and safety of switching patients between reference and biosimilar infliximab. Aliment Pharmacol Ther. 2019;49:31–40.
3. Ye BD, Pesegova M, Alexeeva O, et al. Efficacy and safety of biosimilar CT-P13 compared with originator infliximab in patients with active Crohn’s disease: an international, randomised, double-blind, phase III non-inferiority study. Lancet. 2019;393(10182):1699–707.
4. Simonetto DA, Papadakis KA. New-onset paresthesias in inflammatory bowel disease. Gastroenterology. 2015;148:906–7.
5. McDonnell MJ, Rutherford RM, O’Regan A. Sarcoidosis complicating treatment with adalimumab for Crohn’s disease. J Crohns Colitis. 2014;8:1140–1.
6. Kotze PG, de Barcelos IF, da Silva Kotze LM. Sarcoidosis during therapy with adalimumab in a Crohn’s disease patient: a paradoxical effect. J Crohns Colitis. 2013;7:e599-600.
7. Takahashi H, Kaneta K, Homma M, et al. Sarcoidosis during infliximab therapy for Crohn’s disease. J Dermatol. 2010;37:471–4.
8. Tae KK, Sun HK, Hee SM, et al. Pulmonary sarcoidosis that developed during the treatment of a patient with Crohn disease by using infliximab. Ann Coloproctol. 2017;33:74–7.
9. Decock A, Van Assche G, Vermeire S, et al. Sarcoidosis-like lesions: another paradoxical reaction to anti-TNF-α therapy? J Crohns Colitis. 2017;11:378–83.
10. Dhalie F, Viseux V, Caudron A, et al. Cutaneous sarcoidosis occurring during anti-TNF-alpha treatment: report of two cases. Dermatology. 2010;220:234–7.
11. Van Der Stoep D, Braunstahl G, Van Zeijen J, Wouters J. Sarcoidosis during anti-tumor necrosis factor-α therapy: no relapse after rechallenge in rheumatology and related fields. J Rheumatol. 2009;36.
12. Toussiet E, Pertuiset E, Kantelip B, Wendling D. Sarcoidosis occurring during anti-TNF-α treatment for inflammatory rheumatic diseases: report of two cases. Clin Exp Rheumatol. 2008;26:471–5.
13. Dairen CI, Monnier A, Claudepierre P, Constantin A, et al. Sarcoid-like granulomatosis in patients treated with tumor necrosis factor blockers: 10 cases. Rheumatology (Oxford). 2009;48:883–6.
14. Massara A, Cavazzini L, La Corte R, Trotta F. Sarcoidosis appearing during anti-tumoural phenomenon. Two case reports and literature review. Semin Arthritis Rheum. 2010;39:513–9.

15. Scaletieux L-M, Guedes C, Polard E, Perdriger A. Sarcoidosis after adalimumab treatment in inflammatory rheumatic diseases: a report of two cases and literature review. Presse Med. 2015;44:4–10.

16. Miyagi R, Ideguchi H, Soga T, et al. Development of pulmonary and cardiac sarcoidosis during etanercept therapy. Int J Rheum Dis. 2014;17:810–2.

17. Watrin A, Royer M, Legrand E, et al. Severe hypercalcemia revealing sarcoidosis precipitated by etanercept. Rev Mal Respir. 2014;31:255–8.

18. Duelt C-A, Feurer E, Piatat J-R, et al. Etanercept may induce neurosarcoidosis in a patient treated for rheumatoid arthritis. BMC NeuroL 2013;13:212.

19. Alhajri M, Aljumaah S, Aleyouni Y, et al. Sarcoidosis after adalimumab therapy. Rev Mal Respir. 2014;31:1095–8.

20. Lamrock E, Brown P. Development of cutaneous sarcoidosis during treatment with tumour necrosis alpha factor antagonists. Australas J Dermatol. 2012;53:87–90.

21. Unterstell N, Bressan AL, Serpa LA, et al. Systemic sarcoidosis induced by etanercept: first Brazilian case report. An Bras Dermatol. 2013;88(Suppl 1):197–9.

22. Tong D, Manolios N, Howe G, et al. New onset sarcoid-like granulomatosis developing during anti-TNF-therapy: an under-recognised complication. Intern Med J. 2012;42:89–94.

23. Fok KC, Ng WWs, Henderson C, et al. Cutaneous sarcoidosis in a patient with ulcerative colitis on infliximab. J Crohns Colitis. 2012;6:708–12.

24. Seki E, Hang X, Nishida K, et al. Adalimumab-induced sarcoidosis with hypercalcemia in a Japanese patient. Intern Med. 2013;52:1049–53.

25. Hanon M, Ryan JG, Haney S. Development of sarcoidosis 6-month post discontinuation of etanercept: coincidence or real association? Clin Rheumatol. 2011;30:1095–9.

26. Cui F, Ruiz-Esquide V, Xaubet A, et al. Lung sarcoidosis induced by TNF-α antagonists in rheumatoid arthritis: a case presentation and a literature review. Arch Bronconeumol. 2011;47:208–12.

27. Salviatierra J, Magro-Checa C, Rosales-Alexander JL, et al. Acute sarcoidosis as parotid fever in rheumatoid arthritis under anti-tumor necrosis factor-alpha therapy. Rheumatology (Oxford). 2011;50:1346–8.

28. Kerjuan M, Jouneau S, Lena H, et al. Pulmonary sarcoidosis developing during etanercept treatment. Rev Mal Respir. 2011;28:360–4.

29. Kanellopoulou T, Filotou A, Kranidioti H, et al. Sarcoid-like granulomatosis in patients treated with anti-TNFα factors. A case report and review of the literature. J Rheumatol. 2011;38:581–3.

30. Cucuchovic R, Hagan J, Khan T, et al. Tumor necrosis factor-alpha (TNF-α)-blockade-induced hepatic sarcoidosis in psoriatic arthritis (PsA): A case report and review of the literature. Clin Rheumatol. 2011;30:581–3.

31. Cucuchovic R, Hagan J, Khan T, et al. Tumor necrosis factor-alpha (TNF-α)-blockade-induced hepatic sarcoidosis in psoriatic arthritis (PsA): A case report and review of the literature. Clin Rheumatol. 2011;30:581–3.

32. Clementine RR, Lyman J, Zakem J, et al. Tumor necrosis factor-alpha antagonist-induced sarcoidosis. J Clin Rheumatol. 2010;16:274–9.

33. Pink AE, Fonia A, Smith CH, et al. The development of sarcoidosis on anti-tumour necrosis factor alpha therapy: a paradox. Br J Rheumatol. 2010;49:648–9.

34. Marcella S, Welsh B, Foley P. Development of sarcoidosis during adalimumab therapy for chronic plaque psoriasis. Australas J Dermatol. 2011;52:88–10.

35. Takatori S, Kamata Y, Muroski T, et al. Aplastic development of sarcoidosis with a prodomal increase in plasma osteopontin in a patient with rheumatoid arthritis during treatment with etanercept. Intern Med. 2010;37:210–1.

36. Sim JK, Lee SY, Shim JJ, Kang KH, et al. Pulmonary sarcoidosis induced by adalimumab: a case report and literature review. Yonsei Med J. 2012;53:272–3.

37. Glicà GE, Diaconescu S, Bălan GG, Timofoe Ș, Stănescu G, et al. Sarcoidosis associated with infliximab therapy in ulcerative colitis: a case report. Medicine (Baltimore). 2017;96:6156.

38. Wladis EJ, Tarasen AJ, Roth ZJ, et al. Orbital sarcoid-like granulomatosis in a patient receiving anti-TNFα therapy. Ophthalmology (Baltimore). 2017;96:6156.

39. Sturfelt G, Christenson B, Byrne G, et al. Neurosarcoidosis in a patient with rheumatoid arthritis during treatment with Silverman featuring research articles on clinical subjects from scientists working in rheumatology and related fields. J Rheumatol. 2007;34:12–6.

40. Suzuki J, Goto H. Uveitis associated with sarcoidosis exacerbated by etanercept therapy. Jpn J Ophthalmol. 2009;53:439–40.

41. Josse S, Klemmer N, Moreno-Swirc S, et al. Infliximab induced skin and pulmonary sarcoidosis in a rheumatoid arthritis patient. Joint Bone Spine. 2009;76:716–9.

42. Ishiguro T, Takayanagi N, Kurashima K, et al. Development of sarcoidosis during etanercept therapy. Intern Med. 2008;47:1021–5.

43. Louie GH, Chitkara P, Ward MM. Relapse of sarcoidosis upon treatment with etanercept. Ann Rheum Dis. 2008;67:896–8.

44. Farah M, Al Rashidi A, Owen DA, et al. Granulomatous hepatitis associated with etanercept therapy. J Rheumatol. 2008;35:349–51.

45. Farah RE, Shay MD. Pulmonary sarcoidosis associated with etanerpect therapy. Pharmacotherapy. 2007;27:1496–8.

46. Bachmeyer C, Blum L, Pettjejian B, et al. Granulomatous reaction to etanercept: a paradox. Br J Dermatol. 2010;163:648–9.

47. Morgan V, Jean-Charles C, Nicolas M, et al. Renal sarcoid-like granulomatosis disease following etanercept treatment for RA. J Rheumatol. 2007;34:648–9.

48. González-López MA, Blanco R, González-Vela MC, et al. Development of sarcoidosis during etanercept therapy. Arthritis Rheum. 2006;55:817–20.

49. O’Shea FD, Marras TK, Inman RD. Pulmonary sarcoidosis developing during infliximab therapy. Arthritis Rheum. 2006;55:978–81.

50. Hashkes PJ, Shajrawi I. Sarcoid-related uveitis occurring during etanercept therapy. Clin Exp Rheumatol. 2003;21:645–6.

51. Hübischer Q, Re R, Iotti R. Pulmonary rheumotid noulides in an etanerctic-treated patient. Arthritis Rheum. 2003;48:2077–8.

52. Peno-Green L, Lluberas G, Kingsley T, et al. Lung injury linked to etanercept therapy. Chest. 2002;122:1858–60.

53. Morgane V, Jean-Charles C, Nicolas M, et al. Renal sarcoid-like granulomatosis during anti-TNF therapy. Kidney Int. 2011;79:6156.

54. Dotta ZK, Ochieng P, Donald F, et al. Pulmonary sarcoidosis and latent tuberculosis in a patient with psoriasis treated with adalimumab. Dermatol Online J. 2015.

55. Nnodum BN, Hariri LP, Movrommati D, Dudley L, et al. A case of severe symptomatic central nervous system sarcoidosis secondary to treatment with adalimumab. Case Rep Rheumatol. 2019;7:21539.

56. Leventaki V, Flerange J. Sarcoid-like granulomatosis in a patient receiving anti-TNFα for psoriasis. Am J Hematol. 2018;93:722–3.

57. Sim JK, Lee SY, Shim JJ, Kang KH, et al. Pulmonary sarcoidosis induced by adalimumab: a case report and literature review. Yonsei Med J. 2016;57:272–3.

58. Glicà GE, Diaconescu S, Bălan GG, Timofoe Ș, Stănescu G, et al. Sarcoidosis associated with infliximab therapy in ulcerative colitis: a case report. Medicine (Baltimore). 2017;96:6156.
67. Olivier A, Gilson B, Lafontaine S, et al. Pulmonary and renal involvement in a TNFα antagonist drug-induced sarcoidosis. Rev Med Interne. 2012;33:e25–7.
68. Ognenovski VM, Ojo TC, Fox DA. Etanercept-associated pulmonary granulomatous inflammation in patients with rheumatoid arthritis. J Rheumatol. 2008;35:2279–82.
69. Dubosc AE, Perroud A-M, Bagot M, et al. Cutaneous granulomas during infliximab therapy for spondyloarthropathy. J Rheumatol. 2008;35:1222–3.
70. Phillips K, Weinblatt M. Granulomatous lung disease occurring during etanercept treatment. Arthritis Rheum. 2005;53:618–20.
71. Ando T, Kamiya K, Maekawa T, et al. Adalimumab as a successful alternative for the treatment of infliximab-induced sarcoidosis. J Dermatol. 2019;46:e360-362.
72. Berrios I, Jun-Oonnell A, Ghiran S, Ionete C, et al. A case of neurosarcoidosis secondary to treatment of etanercept and review of the literature. BMJ Case Rep. 2015;2015:bcr2014208188.
73. Jung JH, Kim J-H, Song GG. Adalimumab-induced pulmonary sarcoidosis not progressing upon treatment with etanercept. Z Rheumatol. 2017;76:372–4.
74. Vigne C, Tebib J-G, Pacheco Y, et al. Sarcoidosis: an underestimated and potentially severe side effect of anti-TNF-alpha therapy. Joint Bone Spine. 2013;80:104–7.
75. Cleynen I, Vermeire S. Paradoxical inflammation induced by anti-TNF agents in patients with IBD. Nat Rev Gastroenterol Hepatol. 2012;9:496–503.
76. Jung SK, Lee KH, Jeon JW, et al. Physicochemical characterization of Remsima. MAbs. 2014;6:1163–72.
77. Bargagli E, Mazzi A, Rottoli P. Markers of inflammation in sarcoidosis: blood, urine, BAL, sputum, and exhaled gas. Clin Chest Med. 2008;29:445–58.
78. Rigat B, Hubert C, Alhenc-Gelas F, et al. An insertion/deletion polymorphism in the angiotensin I-converting enzyme gene accounting for half the variance of serum enzyme levels. J Clin Invest. 1990;86:1343–6.
79. Biller H, Zissel G, Ruprecht B, et al. Genotype-corrected reference values for serum angiotensin-converting enzyme. Eur Respir J. 2006;28:1085–90.

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