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CLINICAL IMAGE

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Relapsing polychondritis after COVID-19 vaccination

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Abstract

Relapsing polychondritis (RP) is a rare inflammatory disorder involving immune-mediated destruction of cartilaginous structures. Herein, we present the first report of a strong association between COVID-19 vaccination and RP development. Clinicians should be aware that RP is among the autoimmune diseases that can develop after mRNA vaccination.

KEYWORDS
COVID-19, relapsing polychondritis, vaccine

CLINICAL IMAGE

A 69-year-old man with no respiratory or autoimmune disease history developed increased sputum production 1 week after receiving the first coronavirus disease 2019 (COVID-19) vaccination (BNT162b2 + Omicron BA.4–5; Pfizer–BioNTech). Chest computed tomography (CT) and ^{18}F-fluorodeoxyglucose (FDG) positron emission tomography-CT revealed post-vaccination thickening of the tracheal cartilage wall with FDG accumulation (Figure 1A–C). Bronchoscopy revealed tracheobronchial stenosis (Figure 1D). Serum C-reactive protein levels, which were within normal range before vaccination, increased 1 week after administration. Additionally, serum anti-type II collagen antibody levels were elevated (>153 EU/mL). Tracheal biopsies revealed inflammatory cell infiltration around the cartilage (Figure 1E,F). Based on the examination findings, the patient was diagnosed with relapsing polychondritis (RP) induced by the COVID-19 vaccine. After oral prednisolone administration, the patient's symptoms, cartilage wall thickening, and bronchial stenosis improved (Figure 2). Due to a flare-up of tracheochondritis observed during steroid monotherapy, azathioprine was added and remission was maintained. COVID-19 vaccine-induced autoimmune disease is speculated to be caused by cross-reactivity between severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and autoimmune target proteins.[1] The SARS-CoV-2 spike protein has been reported to cross-react with tissue collagen, a tracheal cartilage component.[2] This cross-reactivity may have caused the RP in our case.

AUTHOR CONTRIBUTIONS

Takunori Ogawa was responsible for drafting the work; conception and design of the work; and acquisition, analysis, and interpretation of the data. Koki Ito and Shunya Igarashi collected the clinical data and drafted the original manuscript. Kosuke Miyai, Kimiya Sato, and Akihiko Kawana critically revised the manuscript for important intellectual content. Yoshifumi Kimizuka was responsible for revising the manuscript critically for important intellectual content. All authors have confirmed the final manuscript and agreed to publication.

CONFLICT OF INTEREST STATEMENT

None declared.

DATA AVAILABILITY STATEMENT

Data available on request from the authors.

ETHICS STATEMENT

The authors declare that appropriate written informed consent was obtained for the publication of this manuscript and accompanying images.

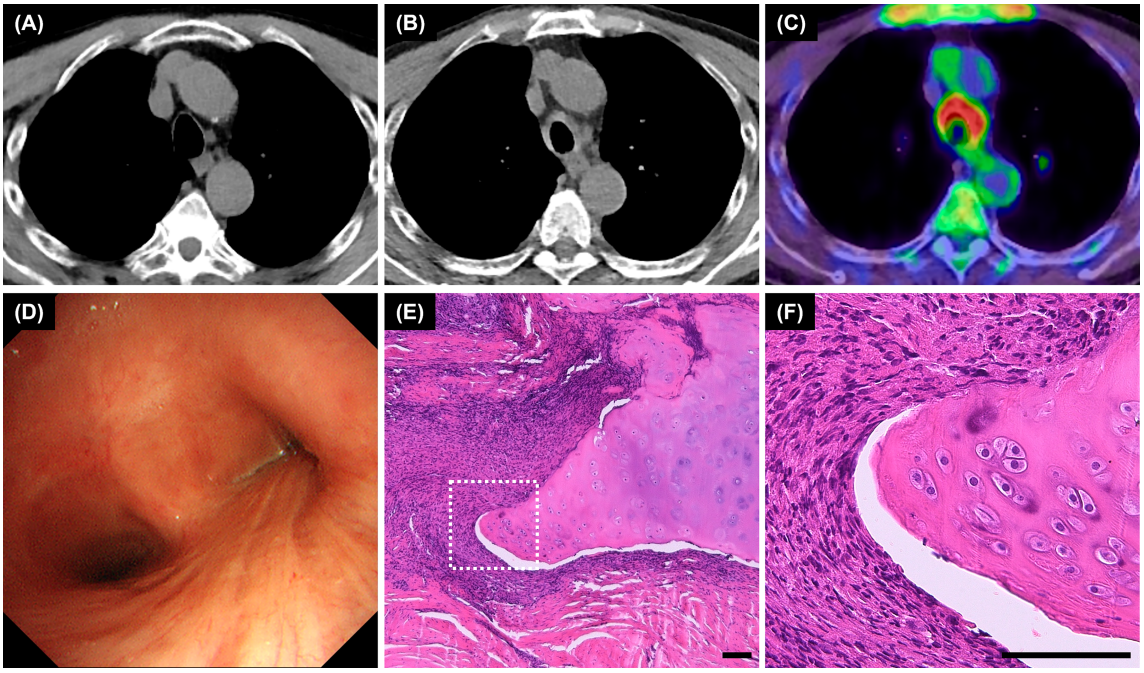

FIGURE 1 (A–C) Chest computed tomography (CT) and ^{18}F-fluorodeoxyglucose (FDG) positron emission tomography (PET)-CT demonstrating findings suggestive of tracheal cartilage inflammation. CT image done for periodic health checkups 2 months before vaccine administration (A). CT (B) and PET-CT (C) images 1 month after vaccine administration showed thickening of the tracheal wall with FDG accumulation except in the membranous portion. (D) The bronchoscopic image showed tracheal stenosis extending into the bilateral main bronchi. (E and F) Histologic examination of the surgical biopsy specimen from the trachea demonstrated lymphocyte and plasma cell infiltration around the tracheal cartilage. Low-power (E) and high-power (F) images of the haematoxylin and eosin stain. Scale bars: 100 μm.

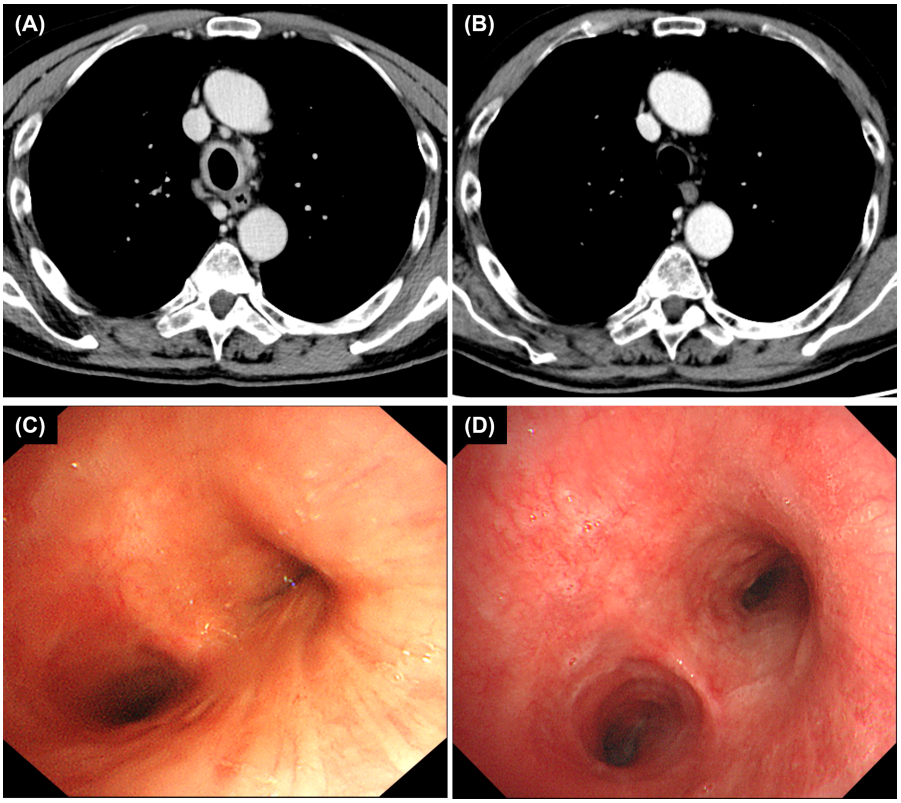

FIGURE 2 Chest computed tomography images (A and B) and bronchoscopic images (C and D) demonstrated improvement in tracheobronchial relapsing polychondritis after treatment with oral prednisolone. The images show findings before (A and C) and 1 month after (B and D) treatment.

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