JCN Open Access LETTER TO THE EDITOR

pISSN 1738-6586 / eISSN 2005-5013 / J Clin Neurol 2020;16(2):336-337 / https://doi.org/10.3988/jcn.2020.16.2.336

Seropositive Rheumatoid Arthritis Presenting with Reversible Parkinsonism and Dysphagia

Seojun Im[a]
Min Ok Kim[a]
Soo-Kyoung Kim[a,c]
Heejeong Jeong[b]
Heeyoung Kang[a,c]

[a]Department of Neurology, Gyeongsang National University Hospital, Jinju, Korea
[b]Department of Neurology, Changwon Gyeongsang National University Hospital, Changwon, Korea
[c]Institute of Health Sciences, Gyeongsang National University College of Medicine, Jinju, Korea

Received April 18, 2019
Revised December 1, 2019
Accepted December 4, 2019

Correspondence
Heeyoung Kang, MD, PhD
Department of Neurology and Institute of Health Sciences, Gyeongsang National University College of Medicine,
15 Jinju-daero 816beon-gil, Jinju 52727, Korea
Tel +82-55-750-8077
Fax +82-55-755-1709
E-mail miranda75@naver.com

Dear Editor,

Autoimmune parkinsonism has been reported in systemic rheumatic diseases such as Sjögren's syndrome and systemic lupus erythematosus, and also in paraneoplastic syndromes with neuron-specific antibodies.[1] However, the central nervous system is rarely involved in rheumatoid arthritis (RA).[2] Here we report a case of seropositive RA presenting with rapid- onset parkinsonism and dysphagia with no previous diagnosis of RA.

A 57-year-old man was admitted with a 3-week history of progressive dysphagia. He had a history of insomnia and a familial history of Parkinson's disease. At admission he was unable to swallow and needed to be kept on a Levin tube. He showed voice change, tremor, rigidity, and slow gait, and these symptoms did not respond to levodopa. An evaluation was performed that included extensive imaging and electrophysiological studies such as esophagography, duodenofiberscopy, cervical spine radiography, brain MRI, CT of the chest and abdomen, nerve conduction study, and electromyography, but theses did not reveal any structural lesion to explain the dysphagia and parkinsonism. Laboratory tests revealed elevations in leukocytes (13,030/mm^3), the erythrocyte sedimentation rate (41 mm/h), and C-reactive protein level (14 mg/L). Tests of autoantibodies for rheumatoid diseases revealed positivity for rheumatoid factor (40.9 IU/mL) and anticyclic citrullinated peptide (189.6 U/mL). No abnormality was observed in a cerebrospinal fluid examination. Tests for paraneoplastic antibodies and neuron-specific antibodies were negative in both the serum and cerebrospinal fluid. The patient had a history of arthralgia, and hand radiography showed erosion of the carpal bone (Fig. 1), and so he was diagnosed with seropositive RA. We treated the patient with methylprednisolone at 1 g/day for 5 days, after which oral prednisolone was initiated for maintenance therapy. Sulfasalazine was administered as a disease-modifying antirheumatic drug. His dysphagia, tremor, and slow gait completely recovered 1 month after initiating the steroid therapy, but mild trunk stiffness and limb rigidity remained.

This case is notable for the presence of rapidly progressive parkinsonism with dysphagia and poor levodopa responsiveness, which is atypical for idiopathic Parkinson's disease. RA is associated with various neurological manifestations including rheumatoid meningitis. Several cases of secondary parkinsonism associated with rheumatoid meningitis have been reported.[3] However, the present patient was unlikely to have rheumatoid meningitis because he had been newly diagnosed with RA, and his cerebrospinal fluid and brain MRI findings were normal. We considered the intense manifestation of RA as being the cause of dysphagia, rigidity, and slow gait. RA can mimic parkinsonism due to the presence of joint stiffness and gait abnormalities. Typical RA presents with gradual-onset morning stiffness, but some patients can experience acute atypical stiffness.[4] One study observed the extrapyramidal type of rigidity in one-quarter of patients in RA.[5] Stiffness and joint destruction in the lower body result in gait abnormalities, while RA and Parkinson's disease manifest similarly with nonmotor symptoms such as fatigue, depression, and sleep change, as well as motor symp-

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (https://creativecommons.org/licenses/by-nc/4.0) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

 Copyright © 2020 Korean Neurological Association

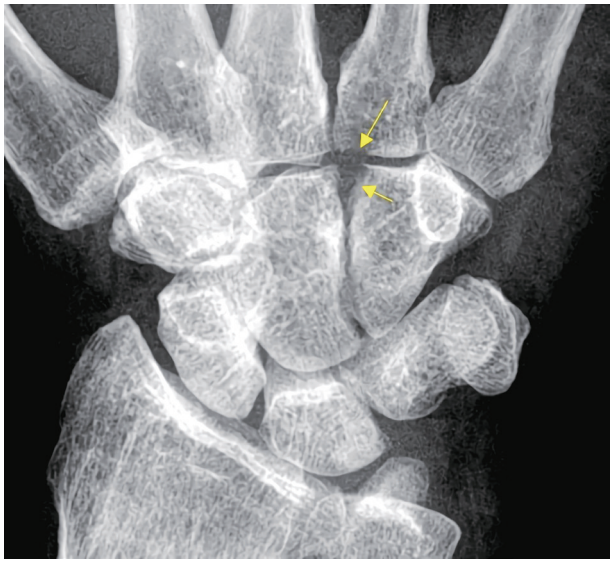

Fig. 1. Anteroposterior view of the left hand shows the marginal bony erosions between 4th metacarpal and hamate bone (arrows).

toms. The flare-up of RA represents the transient acceleration of disease activity and increases joint stiffness, pain, and fatigue-in our case this was mimicking parkinsonism, and early immunotherapy produced a good prognosis. We considered that the dysphagia and voice change of our patient were caused by the laryngeal involvement of RA. Laryngeal involvement is frequent in RA, and the clinical presentations vary from asymptomatic to dysphagia.[6]

In conclusion, this case showed that the atypical symptom of a sudden increase in RA disease activity in a patient without a history of RA is similar to parkinsonism and improves with immunotherapy. The inflammatory markers of the erythrocyte sedimentation rate and C-reactive protein level, radiography of the hand and wrist, and autoantibody tests were useful for diagnosing of RA.

Author Contributions

Conceptulization: Heeyoung Kang. Investigation: Seojun Im, Min Ok Kim. Supervision: Heejeoung Jeong, Soo-Kyoung Kim. Writing—original draft: Seojun Im, Heeyoung Kang. Writing—review & editing: Heeyoung Kang.

ORCID iDs

Seojun Im	https://orcid.org/0000-0001-5249-7459
Min Ok Kim	https://orcid.org/0000-0003-4438-3018
Soo-Kyoung Kim	https://orcid.org/0000-0003-0994-8503
Heejeong Jeong	https://orcid.org/0000-0001-6531-1860
Heeyoung Kang	https://orcid.org/0000-0001-5404-1674

Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

Acknowledgements

None.

REFERENCES

1. Kannoth S, Anandakkuttan A, Mathai A, Sasikumar AN, Nambiar V. Autoimmune atypical parkinsonism–a group of treatable parkinsonism. *J Neurol Sci* 2016;362:40-46.
2. DeQuattro K, Imboden JB. Neurologic manifestations of rheumatoid arthritis. *Rheum Dis Clin North Am* 2017;43:561-571.
3. Pellerin D, Wodkowski M, Guiot MC, AlDhukair H, Blotsky A, Karamchandani J, et al. Rheumatoid meningitis presenting with acute parkinsonism and protracted non-convulsive seizures: an unusual case presentation and review of treatment strategies. *Front Neurol* 2019;10:163.
4. Lee DM, Weinblatt ME. Rheumatoid arthritis. *Lancet* 2001;358:903-911.
5. Ertan S, Fresko I, Apaydin H, Ozekmekçi S, Yazici H. Extrapyramidal type rigidity in rheumatoid arthritis. *Rheumatology* 1999;38: 627-630.
6. Hamdan AL, Sarieddine D. Laryngeal manifestations of rheumatoid arthritis. *Autoimmune Dis* 2013;2013:103081.