Case report

Influenza A (H3N2)-induced rhabdomyolysis complicating anterior compartment syndrome: Serial changes in muscle MRI T2 fat suppression imaging

Tadanori Hamano a,b,*, Akiko Matsunaga a, Osamu Yamamura a, Masako Nakamura d, Yasutaka Kawamura c,d, Itsuro Higuchi e, Masaru Kuriyama a,1, Yasunari Nakamoto a

a Second Department of Internal Medicine, Faculty of Medical Sciences, University of Fukui, Fukui, Japan
b Department of Radiology, Faculty of Medical Sciences, University of Fukui, Fukui, Japan
c Life Science Innovation Center, University of Fukui, Fukui, Japan
d Department of Radiology, Faculty of Medical Sciences, University of Fukui, Fukui, Japan
e Fukui Prefectural Institute of Public Health and Environmental Science, Japan
f Third Department of Internal Medicine, Kagoshima University, Kagoshima, Japan

A R T I C L E   I N F O

Article history:
Received 9 March 2017
Accepted 24 March 2017
Available online 27 March 2017

Keywords:
Influenza A (H3N2)
Rhabdomyolysis
Muscle MRI
T2-fat suppression imaging
Anterior compartment syndrome

A B S T R A C T

Background: Rhabdomyolysis with influenza infection is rarely reported in adults. We report here influenza A induced rhabdomyolysis and anterior compartment syndrome (ACS).

Case report: This case report describes a 43-year-old woman exhibiting influenza A induced rhabdomyolysis. High levels of creatine kinase (97,000 IU/L) and high titer of anti-influenza A virus antibody (H3N2) (320×) with negative anti-influenza B virus antibody were observed. T2 fat suppression muscle MRI imaging showed high-intensity signals in rectus femoris, vastus lateralis, adductor magnus, and semimembranosus (SM) muscles. The existence of ACS was suspected out. Muscle biopsy showed that fiber size variations exist without infiltration of inflammatory cells. The symptoms and muscle MRI findings of T2 fat suppression imaging was markedly improved.

Conclusions: Muscle MRI T2 fat suppression imaging is a useful method to monitor influenza A induced rhabdomyolysis. We should keep in mind the possibilities of rhabdomyolysis and ACS in patients with influenza A infection presenting serious muscle pain.

© 2017 The Authors. Published by Elsevier B.V. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

1. Introduction

Rhabdomyolysis with influenza infection is rarely reported in adults [1–4]. We report here a 43-year-old woman who developed rhabdomyolysis in association with influenza A (H3N2) virus infection. In muscle MRI T2 fat suppression imaging, the affected muscles were clearly detected in both legs. Anterior compartment syndrome (ACS) was complicated with this patient. Clinical symptoms and MRI results were markedly improved. We should consider the possibility of rhabdomyolysis and compartment syndrome in patients with influenza A virus infection.

2. Case description

A 43-year-old woman with no special past history or family history, including metabolic disorders, exhibited high fever, headache, cough and bilateral ankle pain. She was admitted to a local hospital, and diagnosed with influenza infection by the rapid diagnosis kit for influenza type A virus. She had not received a vaccination for influenza A that year. She was prescribed oseltamivir. Ten days later, her fever subsided, but cough and sputum still persisted. As she noticed severe thigh pain, she admitted to our hospital. On admission, she had body temperature of 36.0 °C, blood pressure of 132/60 mm Hg, and regular pulse of 96 beats/min. She had normal respiratory and heart sounds, but exhibited leg edema. Neurologically, her consciousness level, mentality, and cranial nerves were normal. There were no meningeal irritation signs. Muscle strength was normal in the upper extremities, but there was moderate weakness at the proximal portion of the legs. She demonstrated severe muscle tenderness in the proximal portion of the lower limbs. Deep tendon reflexes were brisk in the lower limbs, with no Babinski signs. Her sensation and cerebellar functions were normal. By routine laboratory examination, she had a WBC of 16,500/μL, RBC of 5.62 × 106/μL, her
sodium concentration was 129 mEq/L, and UN was 35 mg/dL. Creatine kinase (CK) was 1197 IU/L (normal, 67–251) on admission. On the third day after admission, her CK elevated to 97,000 IU/L. Anti-influenza A antibody (H3N2) was ×320, but influenza B was negative (× 10). Thyroid function was normal. On CSF examination, the cell count was 0/mm³, protein concentration was 18 mg/dL, and IgG was 1.8 mg/dL. In the EMG study, myogenic changes, including short duration and low amplitude, were observed in rectus femoris (RF), tibialis anterior and first dorsal interosseous muscles.

Fat suppression-T2 weighted imaging (WI) of muscle MRI on day 2 showed high-intensity signals in RF, vastus medialis (VM), adductor magnus (AM), gracilis (G), and sartorius (SA) muscles. Iliotibial tract of the thighs on day 7 (Fig. 1A arrowhead), and in the right TA and tibialis posterior (TP) of legs (Fig. 1A) also showed high-intensity signals. The right TA muscle also exhibited marked swelling (Fig. 1A). This finding was suggestive of ACS [5]. The interosseous membrane of the legs showed high-intensity signals and thickness. On day 13, biceps femoris longus (BL), vastus intermedius (VI), and semimembranosus (SM) muscles demonstrated marked atrophy on T1-weighted imaging (Fig. 1D). All thigh muscles showed high-intensity signals at FS7-T2 WI, except VI, BL, and SM muscles (Fig. 1C). The thickness and high-intensity signals of the crural fascia (double arrowhead) and fascia between S and GM (arrow head) became severe on day 13. Edema in the subcutaneous tissues worsened as well.

Muscle biopsy findings on the 2nd day following admission. Hematoxylin eosin staining demonstrated variation of the muscle fibers, and no infiltration of inflammatory cells, including fascicles, was observed. The patient was given methylprednisolone (1 g/day) for 3 days. Clinical symptoms, and atrophy of BL and SM muscles by muscle MRI were markedly improved after the treatment (Fig. 1E, F).

3. Discussion

ACS was suspected in this patient [5]. Compartment syndrome results from increased pressure within an indispensable space. Trauma, burns, heavy exercise, extrinsic pressure, intramuscular hemorrhage, or rhabdomyolysis [4,5,6] may initiate a vicious cycle of increasing pressure within confining fascia that leads to venous occlusion, muscle and nerve ischemia, arterial occlusion, and tissue necrosis [7]. As this patient exhibited chronic ACS (>48 h), in contrast to acute trauma, urgent decompression fascia was not performed according to the consultation with the orthopedist [5]. Early MRI findings included extremity swelling and diffuse edema within the affected compartment, like our case. Fat suppression T2-weighted imaging of muscle MRI was a sensitive and useful method for estimating affected muscles and degree of influenza A rhabdomyolysis. The observation of rhabdomyolysis is caused by numerous disorders, including acute polymyositis, acute myositis by virus infection [4,6], and hypokalemic myopathy, carnitine palmitoyltransferase I deficiency, or rhabdomyolysis complicating with hypothyroidism [5]. To our knowledge, this is the first report describing the serial change of muscle MRI in influenza A induced rhabdomyolysis, complicated with ACS. We concluded that muscle MRI is useful in the follow-up of influenza A rhabdomyolysis. We also reconfirmed that T2-weighted fat suppression imaging is a useful method to detect affected muscles. The possibility of rhabdomyolysis or ACS in patients with influenza A infection presented with marked muscle pain should be considered.

Ethical standards

The patient gave her informed consent in accordance with ethical standards stated in the Declaration of Helsinki and its later amendments.

Conflict of interest

None.

Funding

A part of this study was supported by the JSPS KAKEN Grant Number JP (25460893, 15K08904, 16K09235), JST (AS242Z03676Q), and a research grant from the University of Fukui.

Acknowledgements

We are grateful to Dr. Arimi Harasawa, Department of Radiology, University of Teikyo, Tokyo, Japan, and Dr. Mikio Hirayama, Department of Neurology, Kasugai Municipal Hospital, Aichi, Japan, for their helpful comments.

**Fig. 1.** Serial examination of muscle MRI. On day 2, fat-suppressed T2-weighted imaging (FS T2-WI) detected high-intensity signals in the rectus femoris (RF), vastus medialis (VM), adductor magnus (AM), gracilis (G), and sartorius (SA) muscles. The right TA (arrow) and bilateral tibialis posterior (TP) muscles exhibited high-intensity signals and swelling (A). On day 13, the vastus lateralis (VL), RF, vastus medialis (VM), SA, AM, and semitendinosus (ST) muscles showed high-intensity signals (C, D). The high intensity signals of thigh and calf muscles, and atrophy of BL and semimembranosus (SM) muscles were markedly improved after therapy (E, F). PE: peroneus muscle, ED: extensor digitorum. (A) and (B): day 2, (C) and (D): day 13, and (E) and (F): day 209. (A), (C), and (E): FS T2-WI, (B), (D), and (F): T1-WI. AM: adductor magnus, VM: vastus medialis, RF: rectus femoris, G: gracilis.
References

[1] L. Berry, S. Braule, Influenza A infection with rhabdomyolysis and acute renal failure - a potentially fatal complication, Postgrad. Med. J. 67 (1991) 389–390.
[2] N.M. Simon, R.N. Rovner, B.S. Berlin, Acute myoglobinuria associated with type A2 (Hong Kong) influenza, JAMA 212 (1970) 1704–1705.
[3] E. Cunningham, R. Kohli, R.C. Venut, Influenza-associated myoglobinuric renal failure, JAMA 248 (1979) 2428–2429.
[4] J.C. Swaringen, J.G. Seiler 3rd, R.W. Bruce Jr., Influenza A induced rhabdomyolysis resulting in extensive compartment syndrome, Clin. Orthop. Relat. Res. 375 (2000) 243–249.
[5] P. Muir, M.S. Choe, M.S. Croxson, Rapid development of anterotibial compartment syndrome and rhabdomyolysis in a patient with primary hypothyroidism and adrenal insufficiency, Thyroid 22 (2012) 651–653.
[6] C.E. Paletta, R. Lynch, A.P. Knutsen, Rhabdomyolysis and lower extremity compartment syndrome due to influenza B virus, Ann. Plast. Surg. 30 (1993) 272–273.
[7] D.A. May, D.G. Disler, E.A. Jones, et al., Abnormal signal intensity in skeletal muscle at MR imaging: patterns, pearls, and pitfalls, Radiographics 20 (2000) S295–S315.