The interlinking background of multiple sclerosis and coronary artery disease. Authors' reply

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We appreciate readers' interest in the presented case and gladly take the opportunity to respond [1]. The comments on our article highlight key issues and bring an interesting perspective, for which we would like to thank. Since the clinical vignette form has its limitations, we were not able to fully report on the complex background.

As mentioned by Polat et al. [2], the complex pathophysiology with multifactorial etiology of acute coronary syndrome (ACS) does not allow us to establish a certain cause of myocardial infarction (MI) using standard diagnostic tools in each case. Finding the answer to the question about the triggering factor and mechanism of atherosclerotic plaque rupture could be unattainable. Expanding the diagnosis to include intravascular ultrasound or infrared imaging could bring new data, but both techniques have their restrictions and would not influence the treatment.

Growing evidence shows that vascular disease and multiple sclerosis (MS) share similar pathogenesis, which includes autoimmunity and pro-inflammatory pathways. Alterations to endothelial functions have been observed in other chronic immune-mediated diseases such as rheumatoid arthritis, psoriasis, and atopic eczema, most likely as a result of inflammatory activity. Endothelial dysfunction may be a precursor to lesions both in MS and atherosclerosis. Changes in endothelium cause increased adherence of immune cells that are involved in the formation of atherosclerotic plaques and enhancement of procoagulant properties due to platelet activation. Higher levels of lipoprotein-associated phospholipase A2 and homocysteine, linked with cardiovascular disease, are also common findings among MS patients. The local inflammatory component in atherosclerotic plaques plays a significant role in most cases, especially in active, unstable plaques; and while systemic inflammation may have contributed to plaque rupture, it does not appear to be the primary factor in the settings of the moderate inflammatory response (white blood count 11.38×10^3/µl, C-reactive protein 74 mg/l) [3]. Another aspect that should be taken into account is dysfunction of the autonomic nervous system and cardiac repolarization, which have been observed in several studies, and could contribute to increased prevalence of MI. Finally, the treatment of MS and its side effects cannot be ignored. Systemic glucocorticoids are the cornerstone of treating relapses and are associated with a significantly increased risk of stroke, MI, and atrial fibrillation. Cardiotoxicity has been reported for disease-modifying therapies (DMTs) such as interferons, mitoxantrone, natalizumab, and fingolimod, which are all used in the treatment of MS. Post-vaccination ACS and MS relapses have been observed in some cases, but the relationship between them is uncertain and requires further study, as mentioned in the original article. Perhaps, even if the histological examination was performed, the cause would still remain unclear. The overlapping burden of comorbidities makes this process even more difficult, and although many hypotheses could be made, pinpointing the exact cause would require a much broader investigation and would not necessarily provide a definitive answer.

Many reports on cases of Takotsubo syndrome (TTS) in the course of multiple sclerosis exacerbation have been published. Indeed, ACS associated with relapse of MS can present as TTS but is not in fact TTS. There have been reports of MI-induced TTS or TTS with secondary plaque rupture, but these conditions occur where acute regional wall motion abnormalities are more extensive than the culprit coronary arteries territory and correspond to the Takotsubo pattern. Coronary multivessel lesions, high troponin I levels, and presented echocardiography likely exclude this diagnosis [4]. However, the pathophysiology of MI is often associated with catecholamine release, microvascular spasm, metabolic impairment, and stunning, resulting in a Takotsubo-like pattern [5]. Perhaps these definitions may need to be changed in the future.

Finally, patients in the acute phase of a MI admitted to the cardiac intensive care unit are closely monitored within 48 hours as part of standard management. Although MS relapse is associated with arrhythmia risk, in this clinical situation, ACS and reperfusion are particularly predisposing, thereby longer surveillance could be considered for this sub-population. No arrhythmia was observed in our case, however, the important nature of these considerations should be borne in mind, as they empasize value of interdisciplinarity in the evaluation of patients. Regardless of progress, this shows how many limitations and unanswered questions remain in medicine.

Article information

Conflict of interest: None declared.

REFERENCES

1. Łagosz P, Biegus J, Gruszka E, et al. The surprising course of multiple sclerosis relapse in a patient after SARS-CoV-2 vaccination. Kardiol Pol. 2022; 80(2): 237–238, doi: 10.33963/KP.a2022.0005, indexed in Pubmed: 35014011.
2. Polat MO, Yalta K, Yalta T, et al. Acute cardiovascular conditions in the setting of multiple sclerosis relapse: Practical implications. Kardiol Pol. 2022; 80(2): 382–383, doi: 10.33963/KP.a2022.0025, indexed in Pubmed: 35114003.
3. Bentzon JF, Otsuka F, Virmani R, et al. Mechanisms of plaque formation and rupture. Circ Res. 2014; 114(12): 1852–1866, doi: 10.1161/CIRCRESAHA.114.302721, indexed in Pubmed: 24902970.
4. Redfors B, Råmunddal T, Shao Y, et al. Takotsubo triggered by acute myocardial infarction: a common but overlooked syndrome? J Geriatr Cardiol. 2014; 11(2): 171–173, doi: 10.3969/j.issn.1671-5411.2014.02.001, indexed in Pubmed: 25009569.
5. Lameris TW, de Zeeuw S, Alberts G, et al. Time course and mechanism of myocardial catecholamine release during transient ischemia in vivo. Circulation. 2000; 101(22): 2645–2650, doi: 10.1161/01.cir.101.22.2645, indexed in Pubmed: 10840018.