

LETTER TO THE EDITOR

R-SUBTYPE OF EARLY ANTIGEN OF THE EPSTEIN-BARR VIRUS IN THE DIAGNOSTICS OF THE CHRONIC FORM OF INFECTIOUS MONONUCLEOSIS

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Serum samples from 69 patients afflicted by infectious mononucleosis were collected during the period from 1989 to 1991. Examinations of specific anti-D and anti-R subtypes of antibodies to the early antigen (EA_D, EA_R) of EB virus revealed the presence of anti-EA_R antibodies. Examinations were performed by use of lymphoblastoid cell line Raji which expresses EA antigen in the presence of tumor producing agent sodium butyrate. One half of the preparations was fixed with acetone (detection of anti-EA_D IgG) and the other one with methanol (detection of anti-EA_D and anti-EA_R IgG). The method of indirect immunofluorescence (1) was employed.

From each of the 69 patients a series of 3 serum samples were taken during 20 months from the onset of the disease. One group included 33 patients with an acute form of infectious mononucleosis. Only two patients of this group had anti-EA_R IgG in the third serum samples (titers 1:20 – 1:40) taken shortly after reconvalescence. The group with subacute manifestation of infectious mononucleosis included 23 patients. Only 1 patient of this group had in his third serum sample anti-EA_R IgG. In comparison to these two groups, the third one of 13 patients with chronic form of infectious mononucleosis yielded in prevalence anti-EA_R IgG (titers 1:20 – 1:80). In the first serum samples (6 – 8 months), two patients were detected to have anti-EA_D IgG (15.4%) and three had anti-EA_R IgG (23.7%). The second serum samples (8 – 14 months) yielded in 2 patients the presence of anti-EA_D IgG (15.4%) and in 10 patients anti-EA_R IgG (76.9%). The third serum samples were positive for anti-EA_D IgG in 2 patients (15.38%) and for anti-EA_R IgG in 11 patients (84.61%).

Some of the 13 patients displayed a change in the presence of specific antigens in the individual samples of the series, namely from anti-EA_R IgG in the first and second samples to anti-EA_R IgG in the third samples. This swap of IgG from anti-D to anti-R is quoted also in the literature

IgG	The positivity (%) of serum samples of 13 patients with chronic form of infectious mononucleosis taken at various intervals after the onset of the disease		
	I (6-8 months)	II (8-14 months)	III (14-20 months)
anti-EA _R	23.1%	76.9%	84.6%
anri-EA _D	15.4%	15.4%	15.4%

referring to patients with chronic manifestation of infectious mononucleosis or fatigue syndrome (2). Recently, the investigation of the antibodies response to the EA is carried out frequently, namely in order to distinguish the response toward D and R subtypes of EA in patients with prolonged manifestation of infection by Epstein-Barr virus. At present it is generally accepted that the specific anti-EA IgG D and R subtypes are the crucial markers in the serologic diagnosis of the etiologic agent of the so-called chronic syndrome of mononucleosis (3). In comparison with patients afflicted with the acute or subacute forms of infectious mononucleosis, the group of patients with the chronic form of infectious mononucleosis described here yielded most frequently the presence of anti-EA_R but not the anti-EA_D IgG. This difference is mostly apparent in the data for the second and third serum samples. Since the serum samples of these patients were taken predominantly during the period of deterioration of their state, e.g. during exacerbation, we may assume on the basis of the presented evidence that the deterioration of symptoms in our patients could coincide with reactivation of chronic infection by Epstein-Barr virus. The increased occurrence of anti-EA_R IgG, detected in the 13 patients with chronic form of infectious mononucleosis also in the present study, is considered also by other authors to represent the main marker of chronic form of infectious mononucleosis and fatigue syndrome (4,5).

References

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ERRATUM

In the article “Characterization of the virions of Mopyridone-sensitive wild strain and Mopyridone-resistant mutant of influenza virus A (H3N2)” by L. Wassilewa *et al.* in *Acta virol.* **39**, 79-84 (1995), the following parts of the article were incorrectly printed due to inadequate corrections of proofs which for the Editorial Office takes apology :

Incorrectly	Correctly	Site
HAU/ μ g	HAU/mg	p. 80, right column, line 19, Table 1
SW11	SW41	p. 80, left column, line 18
MCU-r	MCU-s	p. 80, right column, line 16
pepsin, lysozyme, β -lactoglobulin	Should be omitted	p. 80, left column, lines 26-27
serum albumin, pepsin, lysozyme, β -lactoglobulin	Should be omitted	Fig. 2, legend