Bell's Palsy - Herpes Simplex Virus Type-1 A Possible Causative Agent

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Abstract

The role of Herpes Simplex Virus Type 1 (HSV-I) in Bell's Palsy was evaluated according to clinical symptomatology and serologic findings in a series of 41 patients seen over two years period. Four fold rise in antibody titre between the acute and convalescent sera suggesting a primary infection by HSV-1 was seen in 21.9% cases, while arise in titre or a constantly high titre suggesting a reactivation or recurrent infection by HSV-I was obtained in 29.2% cases. In 48.9% cases there was a negative antibody titre. No seasonal clustering was observed. There was no relation between the recovery of facial nerve function with the antibody titre.

Key words: Bell's palsy. Herpes simplex virus.

Introduction

Acute idiopathic facial palsy (Bell's palsy) has intrigued physicians for years. There are numerous theories about its etiology and the most popular being the vascular, viral and immunological theories. The possibility of a viral aetiology has received increased attention with the Herpetoviridae or herpes group of viruses, as the primary candidate for a causal relationship. Herpes simplex Virus-1 (HSV-I) has been in the forefront of discussion of the aetiology of Bell's palsy (Mc. Cormick 1972, Djupesland et al. 1976, Adour et al. 1978), though no conclusive seriological evidence has seldom been obtained (Mulken et al. 1980, Tovi et al. 1980, Mees et al. 1981, Vahlne et al 1981). To the best of our knowledge there has been no In-

dian study on role of HSV-I in Bell's palsy. Hence, in the present study anti HSV-I antibody was measured in cases of Bell's palsy to explore its possible association with HSV-I infection.

Material & Method

This study was conducted on 41 cases of Bell's palsy seen over a two years period in the Department of Otolaryngology, Mahatma Gandhi Institute of Medical Sciences, Sevagram, Wardha, Maharashtra. All possible causes like intra-cranial tumours, systemic neurologic disorders, traumatic paralysis, otitis media, clinical herpes zoster oticus with facial palsy, pregnancy were excluded from the study. Detailed

history, otolaryngological and neurological examinations, complete haemogram, routine urine analysis, random blood sugar audio-vestibular investigation and topographical evaluation of facial nerve was carried out in all the cases. In recurrent palsy. X-ray study of the temporal bone was done. The severity of palsy on first presentation and during recovery were graded according to House & Brackmann (1985) grading scale.

Serum samples from each patient were obtained, immediately and on third week. These samples were examined for anti HSV-I antibody titre by indirect haemagglutination technique as described by Seth et al. (1978). In brief, sheep RBC (SRBC) was coated with HSV-I antigen using tannic acid. The serum was diluted in phosphate buffered saline (PBS) (pH 7.2) coated with HSV-I antigen to different concentration in microtitre plate. Antigen coated SRBC was then added to each well. The antibody titre was expressed in terms of log₂ of reciprocal of first dilution where no visible agglutination was observed. Controls consisted of (i) 1:8 dilution of serum plus tanned erythrocyte treated with PBS, (ii) 1:8 dilution of serum plus tanned erythrocyte sensitized with uninfected vero cells, (iii) a positive control immune serum (iv) diluent 1% NRS PBS as a negative control, (v) a known negative serum.

The cases were divided into three groups as recommended by Adour et al (1975) with some modifications:

- a) Primary infection of HSV-I (Group I): The cases were considered to be primary infection of HSV-I when titre in the 1st week was less than 6 (i.e. less than 1:64, dil, range 0 to 5) but there was rise in titre at least by 2 (i.e. at least 4 fold rise).
- b) Reactivation or recurrent HSV-I infection (Group II): The cases were considered to be reactivated or recurrent when the titre in the 1st week was more than 7 (i.e. > 1 : 128 dilution) irrespective of its extent of

rise in convalescent phase specimen.

- c) Non-HSV-I etiology (Group III): The cases were considered to have non HSV-I etiology when the titre was in between 0-1 both in acute and convalescent phase serum.

All cases were treated with steroid (Prednisolone), vasodilator (Nilidrinhydrochloride), neurotonic vitamins, facial exercise and proper eye care. The cases were followed up for a period of three months to assess the recovery of facial nerve function. Statistical analysis was performed by Mann-Whitney U test and a 'p' value of less than 0.05 was considered significant.

Results

A total of 41 patients were included in the study. There was a higher percentage of male (56%). Most of the patients in the series (82%) were under 40 years of age. Peak morbidity occurred in the 21-30 years of age. The cases reported in summer (Feb-March), in rainy season (June to September) and in winter (October to January) were 27.8%, 31.7% and 36.5% respectively. However on statistical analysis no seasonal clustering was observed. Topographical examination revealed pathology distal to the geniculate ganglion in 35 cases and proximal in 6 cases.

The antibody titre in various groups is shown in Table 1.

The primary infection of HSV-I was found in 21.9% of cases. In 66.6% cases, associated fever, malaise and upper respiratory tract infection in the form of coryza and sore throat preceded the onset of palsy. The interval between the viral illness and onset of palsy was within a week. Associated vesicles at the oral mucosa with cervical lymphadenopathy were present in 22.2% of these cases. Peripheral smear revealed lymphocytosis in 55.5% cases. The reactivation or recurrent infection group comprised 29.2% cases. Out of these 33.3% had a definite history of exposure to cold

preceding the palsy, 16.6% had evidence of viral infection, 16.6% presented with history of mental stress, 8.3% had recurrent palsy while (8.3%) had diabetes mellitus. HSV-I was not found to be responsible in 48.9% cases. Out of these 30% had evidence of viral infection clinically, whereas none of these had lymphocytosis (Table I).

Antibody titre in 3rd week was significantly high in comparison to that in 1st week in group I ($P < 0.001$) though, it was not significant in group II and III.

In group I, 7 of 9 patients (77.7%), in group II, 10 of 12 patients (83.4%) and in group III 16 of 20 patients (80%) had a satisfactory recovery of facial nerve function (Grade I & Grade II), during the follow-up period.

Discussion

The result of the present study demonstrates that HSV-I is a possible etiological factor in the causation of Bell's palsy as in more than 50% of cases (group I & II), there was evidence of either a primary infection or a reactivation of a latent HSV-I infection as is evident from the rise in antibody titre. The cases in group I which had clinical evidence of a viral infection as well lymphocytosis preceding the onset of palsy showed a four fold increase in antibody titre. The reactivation of HSV-I was due to various factors like upper respiratory tract infection, exposure to cold, sunburn, emotional upset and mental stress as evident from history elicited from the patient. In the non HSV-I infection

(group III), in spite of their being clinical evidence of a recent viral infection in 30% of the cases, the negative antibody titre for HSV-I suggests that it might have been due to viruses other than HSV-I.

In the present study the cases were sporadic and no obvious clustering of cases in a particular season, class of people, families or particular localities was observed.

Earlier reports (Adour et al. 1978, Tovi et al. 1980, Vahine et al 1981) give an incidence of HSV antibody in 75% to 100% of the cases. We observed the HSV-I infection in 50%, the possible reason may be due to the fact that these authors have included all groups of HSV, whereas we studied only HSV-I. A four fold rise in HSV-I antibody titre in 21.9% cases in the present study revealed a higher incidence of primary HSV-I infection as compared to previous reported figures.

The seronegativity of some patients for HSV-I indicates that this infection cannot be solely responsible for Bell's palsy. However the high proportion of HSV-I antibody found as well as clinical findings compatible with HSV-I infection makes it possible to assume that HSV-I is involved in the etiopathogenesis of some cases of Bell's palsy. However, this study with limited number of cases necessitates multicentric studies with a large number of cases with a paired control exploring etiologic role different viruses to get a clear picture of this interesting disorder.

Table 1. Anti HSV-I antibody titre from acute and convalescent phase sera in Bell's palsy cases.

Group (n)%	-log2 titre		Rise in titre
	1st week	3rd week	3rd wk titre - 1st wk titre
Group I (9) 21.9%	1.44 ± 0.75	7.55 ± 0.50*	6.11 ± 0.90a
Group II (12) 29.2%	9.17 ± 0.49	9.67 ± 0.41	0.5 ± 0.23
Group III (20) 48.9%	0	0	0

Values are mean ± SD * $P < 0.001$ in comparison to 1st week titre values a $P < 0.001$ in comparison to respective reactivation value, n = number of cases.

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