Guillain - Barre Syndrome Following Herpes Zoster

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HERPES ZOSTER'İ TAKİBEN GELİŞEN GUILLAIN-BARRE SENDROMU

Geliş Tarlhi:26 Mart 1987

A 52 year old man with polyneuritis follow mg herpes zoster is presented. This Guillain-Barre type of polyneuritis is quite rare as only 20 cases have been reported since the first report by Woftlwill in 1324. Varicellazoster virus is established to induce the aberrant immune response as Guillain-Barre syndrome. Recent developments in our knowledge of varicella-zoster infections showed thatrduring containtment, virus has a dynamic virus-host interaction, as evidenced by rises in levels of antibody in asymptomatic sub jects. This subclinical zoster should be considered in the etiology of many etiologieally unknown Guillain-Barre. syndromes.

Key words: Guillain-Barre syndrome, herpes zoster, :Vancella-zqster, polyneuritis

ÖZET

Bu yazımızda Herpes Zoster'! takiben gelişen bir poiinevrit vakası takdim edilmektedir. Bu Guillain-Barre tipindeki Herpes Zoster! izleyen patİKevnt oldukça nailır olup, ilk defa 1924 yılında Wohlwill tarafından yayınlanmasından beri sadece 20 vaka rapor edilmiştir, Varizella-Zoster virüsünün abeıran immun cevaba bağlı olarak Guillain-Barre sendromurid meydana getirdiği bilinmektedir. Vansellçı-Zoster mfeksıyona ile ilgili bılgılerımizdekı son gelişmeler, virusuii latent kaldığı dönemde, asemptomatik kişilerde kendisini antikor seviyelerinde artışta gösteren, dinamik bir virus-konak ilişkisi olduğunu göstermistir. Bu subklinik zoster, etyolojisı bilinmeydin bir çok Guillain Barr e şendrotrtunun sebebi olarak düşünülmelidir.

Anahtar kelimeler: GuiHain-**Barfe.** sendromü; Herpes **zö:şS**

T Kİ Tıp Bil Araş Dergisi C.6, S.l, 1988 53-56

T J Research Med Sei V.6, N.l, 1988 53-S6

INTRODUCTION

Besides painful cutaneous eruptions characteristic of zoster, various neurological involvement of herpes zoster is well documented (15). These include, segmental paralysis (3, *19)* encephalomyelitis (8,14,17), cranial nerve palsy (6, 13), meningitis (18), Guillain-Barre syndrome (7,8,15) and Fisher's syndrome (20). The report of a case with this rare Guillain-Barre syndrome following herpes zoster is presented.

CASE REPORT

A 52 year old man came to the hospital on February 2-1, 1986 with the complaints of weakness, urine retention and thoracal pain. Two weeks previously he had developed herpes zoster on the left side of the trunk strictly limited by the midline, front and back, in the D9 and D10 dermatome areas. The typical eruptions were later crusted. One week before admission he noticed weakness of his legs, which progressed rapidly and made him unable to walk and pass urine.

The neurological examination on admission showed signs of severe symmetrical polyneuritis. The arms were diffusely affected and paresis was more prominent in the legs. He was unable to walk or stand up. Skin sensation was diminished in the legs. Joint sense was impaired in the toes. Tendon reflexes were also diminished. Cranial nerves were not affected.

**Presented in XXII. National Psychiatry and Neurological Sciences Congress (29 October - 7 November 1986).*

Table -1

	Sex	Age (year)	Interval zoster-polyneuritis	Sensory-disturbance	Cranial nerve involvement	Onset	Lumbar CSF Cells (c.mm)	Lumbar CSF Protein (mg/100 ml)
1. Wohlwill (1924)	F	44	2 weeks	+		Acu te	Normal	Raised
2. Schubach (1930)	F	62	7 days		–	Acute	?	•>
3. Riser and Sol (1933)	M	30	2 months	+	–	Insidious	Normal	1,750
4. Gilpin, Moersch and Kernohan (1936)	M	52	Few days	+	Bilateral VII	Subacute	Normal	Raised
5. Maggi, Meeroff, Cosen and Hirschman (1956)	M	68	1 month	+	Right VIII	Insidious	Normal	Normal
6. Friart and Jeanty (1956)	F	72	?>	+	–	Subacute	25	3,300
7. Stammler and Struck (1958)	F	66	3 days	+	Bulbar	Acute	430	5,000
8. Palffy and Balazs (1959)	F	63	2 months	+	–	Insidious	9	146
9. Palffy and Balazs (1959)	F	53	7 days	+	Bulbar	Acu te	14	24
10. Duperrad and Pringued (1958)	M	53	2 days	0	Right VII	Acute	3	480
11. Knox Levy and Simpson (1961)	M	69	2 weeks	+	–	Insidious	3	50
12. Knox Levy and Simpson (1961)	M	54	7 weeks	+	–	Acute	8	89-150
13. Knox Levy and Simpson (1961)	F	65	7 weeks	+	Right III	Subacute	4	162
14. Levanti and Ledy (1963)	M	35	•>		Bilateral V, L. VI, R VII	Subacute	19	400
15. Bonduelle, Bouygues and Chemaly (1963)	F	63	2 months	+	–	Insidious	Normal	300
16. Nevsimal and Lehovsky (1963)	M	67		0	Bulbar Bilateral VII	–	3	122
17. Castellotti and Pittalugo (1965)	F	64		0	–	5	Normal	1,100
18. Dayan, Ogul and Graveson (1971)	M	67	1 week	+	Bilateral VII	Acute	–	
19. Dayan, Ogul and Graveson (1971)	M	53	5 days	+	R. V	Subacute	0	35
20. Gardner-Thorpe, et al. (1975)	M	45	14 days	+	–	Subacute	2	240

Examination of the lumbar CSF showed 15 cells/ mm^3 and a protein content of 78 mg/100 ml. On the eighth day of the polyneuritis, electromyographic examination showed conduction at 27,8 m/sec in the right tibial nerve and 43 m/sec in the right median nerve. M-responses were markedly dispersed.

Treatment was started with ACTH 80 units per day. On the second day of admission left VI. and VII. cranial nerve paresis developed. There were signs of sympathetic irritation with mild midriasis of the left pupil, hyperhydrosis and flushing of the left side of the face.

On the fifth hospital day mild respiratory distress developed, but no further extension of the polyneuritis occured thereafter. The following days a rapid improvement was observed, and he could walk short distance without help on his tenth day of admission. ACTH therapy was reduced and on the 15th day he returned home with ACTH twice a week for Iwoweeks.

DISCUSSION

The history and the clinical features of the patient was typical of herpes zoster causing firstly cutaneous eruptions which later crust. A polyneuritis of Guillain-Barre type has followed it. Despite the lack of serological confirmation we consider that this patient's neurological illness was due to varicella-zoster virus. Cultural and serological studies have previously provided firm support for the hypothesis that the etiological agents of varicella and zoster are identical and the agents are termed varicella-zoster virus (21). Earlier Garland had suggested that clinical zoster reflects the activation of a latent varicella virus (9). This concept is now generally accepted. Although the mechanism whereby the virus remains latent before recrudescence as zoster is still unknown (4, 5, 16), it appears that the virus-host interaction during containtment is a dynamic rather than a static relationship (5, 21). Rises in titers of spesific IgM antibody have been observed in "immune" subjects who have had varicella without evident zoster infection (2, 5, 10, 11, 12, 22). This subclinical zoster is worth noticing. Considerable evidence has been brought forward in recent years to suggest that Guillain-Barre syndrome represents an aberrant immune response (1). Varicella-zoster is considered in the etiology of Guillain-Barre syndrome besides other viruses amoung which are measles, mumps, rubella, influenza A and B, cytomegalovirus, infectious mononucleosis, vaccinia, variola, hepatitis B, coxsackie and ECHO (1). The viruses probably act indirectly to trigger an immunologic response, and the host immune response would then produce the disease secondarily with lymphocytic cellular infiltration and myelin destruction (1).

Guillain-Barre syndrome following herpes zoster is quite rare as only 20 cases have been reported (TableT) since the first account by Wohlwill in 1924 (7,8).

The very uncommon clinical occurrence of zoster polyneuropathy has attracted attention and it is attributed to either body's ability to prevent such an "autoimmune" reaction, or to remain at a subclinical level (1, 7). Recent studies, brings a new look to these considerations and emphasizes that Guillain-Barre syndrome following herpes zoster may be more than established and not in fact a rarity. It should be considered that subclinical zoster detected with only slight rises in titers of spesific antibody may be the underlying etiologic agent triggering this aberrant immune response in many etiologically unknown Guillain-Barre syndromes (10, 21). That may also explain why Guillain-Barre syndrome following herpes zoster is so rare (7), because no search for a preceeding subclinical herpes zoster has been made up to now. If so, it would be worthwhile examining Guillain-Barre syndromes of uncertain etiology for the presence of a preceeding subclinical varicella-zoster infection.

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