# Ocular Nerve Palsies with Headache in Diabetes Mellitus

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Isolated cranial nerve lesions are reported as occurring with greater frequency in patients suffering from diabetes mellitus than in a nondiabetic population.[1] Cranial nerves III and VI appear to be involved most frequently, and not VII as stated by Joslin *et al.*[2]

The association of severe headache or periorbital pain with the ocular nerve palsies in diabetic subjects appears to have escaped attention until the recent report by Jackson.[3] The possibility of an accompanying trigeminal neuritis is mentioned by Martin[4] and Goodman *et al.*[5]

The three cases reported here illustrate association of unilateral headache and ocular nerve paresis; they form part of a small series of six patients with ophthalmoplegias seen in this clinic in the last five years.

## CASE REPORTS

*Case 1.* A man aged 59, known to have had diabetes for 24 years, was admitted because of frontal headaches of recent onset. His diabetes was inadequately controlled on 40 units of an insulin mixture daily. The past medical history was noncontributory, and there was no evidence of alcoholism. Neurological examination revealed absence of knee and ankle jerks and loss of vibration sense at the ankles. The blood pressure was 120/80.

Within a few days of admission he developed diplopia, and his headache increased in severity, becoming localized over the left eye. Examination revealed left sixth nerve palsy which became complete over a period of about 48 hours. During the following week he developed a right sixth nerve palsy which was also gradual in onset. There was no objective evidence of involvement of the trigeminal nerve, although the distribution of pain strongly suggested this. Radiographs of the skull and sinuses and air encephalography were normal. The cerebrospinal fluid was normal, and the Wassermann reaction was negative in both blood and cerebrospinal fluid. Tests for sweat secretion showed loss of sweating over the lower extremities, and a cystometrogram showed a "pseudotabetic" curve as seen in diabetic patients with autonomic neuropathy. Both of the ocular palsies disappeared within three months without specific therapy other than the control of his diabetes.

*Case 2.* A woman 61 years of age, diabetic for the previous 6 years, was admitted with a history of 3 days of severe left frontal headache and diplopia. She complained also of pain down the left arm. Her diabetes was moderately controlled on 70 units of zinc protamine insulin daily. There was nothing significant in her past medical history, and she took no alcohol.

Examination showed complete sixth nerve paralysis on the right side, with ipsolateral loss of corneal reflex. There was wasting and tenderness of the left deltoid and triceps muscles and loss of the left triceps jerk. The blood pressure was 200/110, and there was cardiomegaly.

Investigations which included radiographs of the skull and examination of the cerebrospinal fluid were normal. The Wassermann reaction was negative in the cerebrospinal fluid and blood.

Recovery from both headache and diplopia was complete within three months, and there has been no recurrence in the last two years. Treatment was confined to the stabilization of her diabetes.

*Case 3.* A man aged 68 was known to have had diabetes for 14 years. His diabetes was indifferently controlled on about 24 units of protamine zinc insulin daily. He was also known to have had peripheral neuritis with left foot drop, and he suffered from intermittent attacks of nocturnal diarrhea with rectal incontinence. He had had no other significant illness, and his alcohol consumption was minimal.

In 1954 he was admitted complaining of diplopia and severe pain around the left eye. Examination revealed left third nerve palsy with ptosis, diminished left corneal reflex, and impairment of sensation to light touch over the first sensory division of the left trigeminal nerve. The ankle jerks were absent, and there was loss of sweating over both legs below the knee. The blood pressure was 130/80.

Radiographs of the skull and sinuses were normal; the cerebrospinal fluid was normal. The Wassermann reaction was negative in both cerebrospinal fluid and blood.

Insulin dosage was adjusted to improve diabetic control, but no other treatment was instituted.

Recovery began within three weeks, and was complete at the end of four months. There has been no recurrence.

## DISCUSSION

There is still little agreement on the etiology of cranial nerve lesions which occur in diabetic subjects. Collier,[6] reporting the largest series at that time, considered these lesions to be of vascular origin because his patients were mostly in the latter half of life. Weinstein and Dolger[7] considered that the most likely cause of paresis of the extra-ocular muscles was hemorrhage into the ocular nerve nuclei. It seems possible to challenge this view on the following grounds. Ocular nerve palsies have been reported in young subjects with severe diabetes where there was little evidence of vascular degeneration.[8] Further, multiple and recurrent cranial nerve palsies associated with severe and labile diabetes,[9] and the associa-

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tion of a trigeminal nerve lesion suggest a process involving the peripheral nerves rather than the nuclei. Furthermore, the onset is not invariably sudden as would be expected if the cause were always vascular. This point is particularly well illustrated by the patient in Case 1 who was under observation in hospital when his ocular nerve palsies occurred. In this instance it was possible to observe the gradual and progressive development of both sixth nerve lesions.

The neurologic investigations performed in this group, except for Case 1, were limited, but extensive investigations were not thought to be indicated in these cases since the prognosis is known to be good. Therefore, although vascular and space-taking lesions cannot be said to have been excluded, the clinical course did not suggest the presence of such lesions. This small series demonstrates that the ocular nerve palsies of diabetes mellitus may be gradual in onset, and that there may be evidence of an associated trigeminal nerve involvement. This supports the view that they may be "neuritic" in nature and not, as has been previously asserted, always of vascular origin. It is notable that in all three cases there was evidence of peripheral or autonomic neuropathy, either accompanying the ocular nerve lesions or in the past. This association of cranial and peripheral neuropathy was observed by Root.[10]

## SUMMARY

The association of unilateral headaches, presumed to be due to trigeminal nerve involvement with ocular nerve palsies, in patients with diabetes mellitus is described. In one the ocular palsies were of gradual onset suggesting a "neuritic" rather than a vascular pathology. Experience suggests that this is not an uncommon pattern of cranial nerve lesions in diabetic patients.

## SUMMARIO IN INTERLINGUA

*Paralyses del Nervo Ocular con Mal de Capite in Diabete Mellite*

Es describite le occurrentia, in patientes de diabete mellite, del association de unilatere mal de capite—supponitemente debite al affection del nervo trigemine—con paralyses del nervo ocular. In un caso le paralyses ocular esseva characterisate per un declaration gradual, lo que pareva indicar un pathologia "neuritic" plus tosto que vascular. Experientias passate indica que iste manifestation de lesiones cranio-nerval non es incommun in patientes diabetic.

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