

THE SYNDROME OF PARSONAGE AND TURNER DISCUSSION OF CLINICAL FEATURES WITH A REVIEW OF 8 CASES

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A clinical analysis of 8 patients with brachial plexus neuropathy is presented. The disease may involve the upper, the lower, or the entire plexus. There is a higher incidence in men than in women. The syndrome is not uncommon but is frequently diagnosed incorrectly. The fairly typical pattern of symptoms and signs includes sudden onset with severe pain along one side of the shoulder girdle, followed in a few hours or days by atrophic paralysis of muscles over the affected shoulder. The disorder is occasionally bilateral. The paresis persists for months or even years. The overall prognosis is excellent despite the severity and extent of the lesion. The etiology is unknown, but decreased physical resistance is a predisposing factor.

Keywords: syndrome of Parsonage and Turner ; plexus brachialis.

Mots-clés: syndrome de Parsonage et Turner ; plexus brachial.

RÉSUMÉ

B. VANERMEN, M. AERTGEERTS, M. HOOGMARTENS et G. FABRY. Le syndrome de Parsonage et Turner. Aspects cliniques. Revue de 8 cas.

Les auteurs ont revu 8 malades présentant une neuropathie du plexus brachial. Ils décrivent les aspects cliniques de cette affection qui peut atteindre le plexus supérieur, inférieur ou sa totalité. L'affection est plus courante chez l'homme que chez la femme. Le syndrome n'est pas rare mais le diagnostic exact est rarement posé. Le tableau clinique est assez typique et caractérisé par un début brutal avec douleur intense à la ceinture scapulaire, suivie après quelques heures ou quelques jours d'une paralysie avec atrophie des muscles de la ceinture scapulaire.

L'affection peut être bilatérale. La paralysie, complète ou incomplète, persiste pendant des mois ou même des années. Le pronostic est en général bon, malgré la gravité initiale et l'étendue de la lésion. L'étiologie est inconnue mais une diminution de la résistance physique semble un facteur prédisposant.

SAMENVATTING

B. VANERMEN, M. AERTGEERTS, M. HOOGMARTENS et G. FABRY. Het syndroom van Parsonage en Turner. Klinische tekens. Bespreking van 8 gevallen.

Een klinisch bilan van 8 patiënten met plexus brachialis neuropathy wordt gepresenteerd. Deze aandoening kan het bovenste, het onderste deel of gans de plexus aantasten. Er wordt een hogere incidentie vastgesteld bij mannen dan bij vrouwen. Het klinisch verloop is typisch. Het begin is abrupt en wordt gekenmerkt door hevige pijn t.h.v. de schoudergordel. Na enkele uren of dagen mindert deze pijn en treedt er een atypische paralyse op van bepaalde schouderpijpen. Deze paralyse kan enkele maanden tot 1 jaar duren. De prognose is gunstig. De etiologie is niet duidelijk gekend maar vermindering van de fysieke weerstand is een voorbeschikende factor.

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INTRODUCTION

The Parsonage-Turner syndrome comprises pain and flaccid paralysis of the muscles around the shoulder girdle. The basic clinical picture is simple : without any predisposing constitutional disturbance, pain starts suddenly across the top of the scapula* and may radiate down the lateral aspect of the upper arm and into the neck. This pain lasts from a few hours to a fortnight or more, and then flaccid paralysis of some of the muscles of the shoulder girdle and the arm develops. In some cases, there is a patch of numbness over the lateral aspect of the upper arm. When the paralysis appears, the severe pain usually stops, but a dull ache may persist considerably longer. However, this clinical picture is variable. This disorder of the brachial plexus has been described under such names as brachial plexus neuropathy, multiple neuritis, localized neuritis of the shoulder girdle, acute brachial radiculitis, localized nontraumatic neuropathy in military personnel, neuralgic amyotrophy, acute shoulder neuritis, paralytic brachial neuritis and syndrome of Parsonage and Turner. Remarkably little was published about this condition before 1942. Some cases of serratus anterior palsy were recorded after operation or infection (Bramwell and Struthers, 1903), but most of the cases were traumatic in origin. The standard neurological textbooks did not describe the condition beyond stating that a toxic neuritis of the long thoracic nerve and sometimes of the circumflex nerve may occur after infections such as typhoid fever and pneumonia.

In England, Richardson in 1942 drew attention to the increased incidence of serratus anterior palsy. Shortly afterwards, in the Middle East, Burnard and Fox (P1942) described cases of multiple neuritis of the shoulder girdle, and Spillane (1943) also analyzed 46 cases of localized neuritis of the shoulder girdle. One of the largest series of patients with this disorder and one of the most detailed reviews of the problem was reported by Parsonage and Turner in 1948. They suggested the name "neuralgic amyotrophy".

* This terminology is excellent for the lay public — it is condescending for our readers.

MATERIALS AND METHODS

Age and sex distribution

A series of 8 patients with brachial neuritis was reviewed. The youngest patient was 20 years, the oldest was 64 years. The mean age at the time of onset was 28 years. The patients spanned the range from 20 to 65 years fairly evenly. Large studies of cases were reported in military personnel, which of course showed a high incidence in men. In our series, a male predominance of 3 to 1 is also demonstrated.

Antecedents

Spinnale (12) noted that in 26 of his 467 cases, the condition developed while the patient was in hospital, usually in the convalescent stage of an infectious illness or following surgery. In this series of 8 patients only one was in the convalescent stage following surgery.

It is of interest that in 35% the syndrome developed after heavy physical activity. No patient had recent serum injections. There is a lower incidence of previous illness than that reported by Parsonage and Turner, who found that nearly half of their patients were hospitalized with other conditions when the shoulder girdle syndrome started.

Association was noted with either the acute phase or convalescence from such diseases as malaria, typhus, typhoid and dysentery.

Mode of onset

An important feature of the onset is the absence of fever and constitutional symptoms. Local pain is almost always the exhibited symptom. In this series almost all patients experienced the sudden onset of severe pain. This pain is constant rather than intermittent. Most commonly, it is experienced around the shoulder and upper arm, but in some cases, particularly those in which the musculocutaneous or the anterior interosseous nerves are involved, there may be a good deal of pain in the elbow and forearm.

At times, there is bilateral shoulder pain, although paralysis may only appear in one arm. The interval

between the onset of pain on the two sides varies, but most patients note bilateral pain either simultaneously or within 24 hours. The pain is usually aggravated by movements of the shoulder, but usually not by movements of the cervical spine. There may be considerable muscle tenderness. There is no exact relation between the position of the pain and the distribution of the subsequent muscle paralysis. The ordinary sequence of events is for the severe pain to last from a few hours to a week or two, and then to stop fairly suddenly as muscle paralysis appears.

Less severe pain may last considerably longer. In our series the interval between onset of pain and muscle paralysis ranged from 3 days to 21 days with a mean interval of 10 days.

Paralysis

The most striking feature is the rapid occurrence of muscle weakness after a variable period of pain. The weakness is usually maximal at the onset.

In bilateral cases, there may be a time interval between the motor involvement of the two sides. In many cases, the pathological process occurs in one or more peripheral nerves, while in others it must be localized in the nerve roots or in the spinal cord. Involvement of the long thoracic nerve, leading to paralysis of the serratus anterior muscle, and winging of the scapula, was the most common type in this series. There were 4 cases with unilateral involvement.

Disability from a lesion of the long thoracic nerve varied markedly from patient to patient. In some, winging of the scapula was the only complaint. In others there was considerable difficulty in raising the arm above the head.

In two cases the muscle involvement could be explained by a lesion affecting two nerves. The combination was paralysis of the spinati and deltoid muscles. In 2 cases, the involvement was explicable by a lesion of the nerve roots.

Objective evidence of cutaneous impairment was found in 3 patients; there was hypesthesia and hypalgesia on the lateral side of the arm in the distribution of the axillary nerve.

In two patients sensory loss followed the pattern of the C₇ and C₈ dermatomes.

ETIOLOGY

The etiology remains obscure. A viral infection has been suggested as the cause, but there are features unusual for virus infections of the nervous system, particularly the absence of constitutional symptoms at the onset and the findings of normal cerebrospinal fluid. A similar syndrome has occasionally been seen as a complication of serum injection. This usually develops from 7 to 10 days after the serum has been given and may be preceded by generalized urticaria.

The current thinking is that this form of neuropathy is either a manifestation of a systemic or localized infectious disorder, possibly viral, or the

Table I. — Clinical results

	Age	Sex	Precipitating causes	Onset	Pain	Interval	Motor involvement	Sensory changes
H. S.	20	Female	Physical activity	—	±	—	Serratus Ant. L. Rhomboid R. decreased	—
VM. R.	21	Male	Surgery	Sudden	Severe	14	R. Serratus Ant.	R. upper arm
S. R.	23	Male	Physical activity	Sudden	Severe	3	L. Deltoid + Supra + Infraspinat.	—
D. O.	26	Male	Physical activity	Sudden	Severe	4	R. Serratus Ant.	—
P. T.	35	Female	?	Sudden	Severe	21	Deltoid L. Supra + Infra- spinatus	—
M. W.	54	Male	?	Sudden	Severe	14	R. Serratus Ant.	—
R. A.	63	Male	?	Sudden	Severe	4	C ₇ C ₈ T ₁	Hand
M. A.	64	Male	?	Sudden	Severe	21	C ₈ T ₁	Hand

result of an allergic or hypersensitivity reaction. Laboratory tests for either of these mechanisms still do not exist. An important factor in the etiology appears to be a decrease in the physical resistance, usually seen some days or weeks before the onset.

LABORATORY STUDIES

Cerebrospinal fluid levels are normal. Roentgenograms of the cervical spine and shoulder show normal findings, except that mild hypertrophic arthritic changes can be noted compatible with the age. Immunologic studies (13) reveal no specific abnormalities. Biopsy of a cutaneous branch of the radial nerve was done in two patients with severe plexus involvement. The histologic findings are those of profound axonal degeneration (3). Electromyography is helpful in localizing the lesion. In our study, the localization was confirmed in all the patients. Eleven of the 99 patients in the study of Tsairis (13) with unilateral involvement on clinical examination had electrodiagnostic studies of their unaffected limb. Of these, 6 had

electromyographic, but not nerve conduction, abnormalities. This indicates that some patients with brachial plexus neuritis on one side may have subclinical involvement on the other side.

DIFFERENTIAL DIAGNOSIS

Anterior poliomyelitis

In the early stages of the shoulder girdle syndrome, constitutional symptoms are absent and the cerebrospinal fluid is normal. Moreover, in many cases, there are mild sensory changes, unlike poliomyelitis.

Prolapsed cervical intervertebral disc

Lateral prolapse of a disc usually affects only one root, most often C₇. The profound weakness and atrophy of the shoulder girdle syndrome do not occur. The feature that distinguishes neuralgic amyotrophy from an acute disc herniation is the involvement at multiple nerve root levels.

Table II. — Electromyographic examination and follow-up

	Bilateral	Differential diagnosis	E.M.G.	Treatment	Follow-up	Recurrence	Improvement after
H. S.	L > R	—	Denervation Serratus Ant. + L. Rhomboids	Mobilization	4 years	—	—
VM. R.	—	—	Denervation R. Serratus Anterior	Mobilization	2 2/12 y.	—	1 year
S. R.	—	—	Denervation potentials L. Axillary nerve	Mobilization	2 5/12 y.	—	2 3/12 years
D. O.	—	—	Fibrillation + positive sharp waves Ser. Ant.	K-I	1 8/12 y.	—	—
P. T.	—	<	Axillary nerve + Supraspin	—	18	—	6/12
M. W.	—	—	Serratus Anterior	Infiltration Mobilization	2 6/12	—	1 year
R. A.	—	Cervical osteoarthritis	C ₇ C ₈ T ₁	Ant. fusion	4 3/12	—	—
M. A.	—	Cerv. spin. stenosis	C ₈ T ₁	Mobilization	8/12	—	6/12

Brachial neuritis

In its strict sense, brachial neuritis implies a diffuse pathology of the brachial plexus, leading to pain in the arm, slight generalized weakness, diminution of the tendon jerks and diffuse sensory changes. Localized paralysis and wasting of muscles are not features of brachial neuritis.

Progressive muscular atrophy

The acute onset with pain, the rapid development of wasting, the absence of fasciculations, and the nonprogressive course of the disease are all entirely different from progressive muscular atrophy.

Cervical spondylosis

Subacromial bursitis and bicipital tenosynovitis

In the initial stage of the Parsonage-Turner syndrome, it may be difficult to distinguish it from subacromial bursitis and bicipital tenosynovitis.

TREATMENT AND PROGNOSIS

No specific treatment for the condition is known. In the early painful stages analgesics may be necessary.

Treatment by steroids is founded on the suspicion that the disorder may have a neuroallergic basis. The value of this treatment is uncertain.

The most important form of physical treatment is to put the shoulder joint through its full range of motion at least twice a day, to prevent stiffness, and to start active movements as soon as voluntary strength returns. When severe wasting occurs early and rapidly, the prospect of return of muscle strength is poor.

Recovery of voluntary strength in completely paralyzed muscles can certainly start 9 to 12 months after the onset, and probably even later, and may continue up to 2 years or longer. The muscle which is least likely to recover is the serratus anterior, where return of strength after complete paralysis is poor.

Functional recovery appears much earlier in patients with predominantly upper plexus lesions

than in those with lower plexus lesions. According to Tsairis (13), by the end of one year more than 60% of the patients with upper plexus lesions had recovered normal function, whereas none with predominantly lower plexus involvement had.

Overall, the rate of recovery was estimated to be 36% within one year, 75% by the end of the second year, and 89% by the end of the third year.

Minimal residual neurological deficits consisted of isolated winging of the scapula, slight proximal or distal weakness, mild sensory loss and slight decrease in tendon reflexes.

In this series, functional recovery was best in the deltoid muscle. The muscle regained its function after 3 to 6 months.

Recurrent attacks affecting the same shoulder are uncommon and may occur many years after the first attack. They are not as severe as the initial syndrome. In this series, there was no recurrence.

CONCLUSION

The essential clinical picture of the Parsonage-Turner syndrome consists of sudden onset of pain in the region of the shoulder or upper part of the arm, followed in days by atrophic paralysis of various muscles. Occasionally it is bilateral. The pain subsides but the paresis persists for months or even years. Overall prognosis is good. The etiology is unknown but decreased physical resistance is a predisposing factor.

REFERENCES

1. ALLEN I. M. The neurological complications of serum treatment. *Lancet*, 1931, 2, 1128-1131.
2. BACEVICH B. B. Paralytic brachial neuritis. *J. Bone Joint Surg.*, 1976, 58-A, 262-263.
3. BRADLEY W. Y., MADRID R., THIUS H. C., CAMPBELL M. J. Recurrent brachial plexus neuropathy. *Brain*, 1975, 98, 381-398.
4. BURNARD E. D., FOX T. G. Multiple neuritis of shoulder girdle. *N. Z. Med. J.*, 1942, 41, 243-247.
5. COMROE J. H., WOOD Fr. C., KAY C. F., SPOONT E. M. Motor neuritis after tetanus antitoxin with involvement of the muscles of respiration. *Am. J. Med.*, 1951, 10, 786-789.
6. GEIGER L. R., MANCAL E. L., PENN A. S., TUCKER S. M. Familial neuralgic amyotrophy. *Brain*, 1974, 97, 87-102.

7. GRUTTEN C. H., CRIELAARD J. M., FRANCHI-MONT P. Neuralgie amyotrophiant ou syndrome du Parsonage et Turner. *Rev. Med. Liège*, 1986, *XL*, N4.
8. JACOB J. C., ANDERMANN F., ROBB J. P. Heredofamilial neuritis with brachial predilection. *Neurology*, 1961, *11*, 1025-1033.
9. JAMES J. L., MILES D. W. Neurologic amyotrophy : a clinical and electromyographic study. *Brit. Med. J.*, 1966, *2*, 1042-1043.
10. MAGEE K. R., RUSSELL N., DEJONG. Paralytic brachial neuritis. *J.A.M.A.*, 1960, *174*, 1258.
11. PARSONAGE M. J., TURNER J. W. A. Neuralgic amyotrophy. The shoulder-girdle syndrome. *Lancet*, 1948, *1*, 973-978.
12. SPILLANE J. D. Localised neuritis of the shoulder girdle : report of 46 cases. *Lancet*, 1943, *2*, 532.
13. TSAIRIS P., DIJCK P. J., MULDER D. W. Natural history of brachial plexus neuropathy. *Arch. Neurol.*, 1972, *27*, 109-117.
14. TURNER J. W. A., PARSONAGE M. J. Neuralgic amyotrophy. *Lancet*, 1957, *2*, 209-212.
15. WEIKERS N. J., MATTSON R. Acute paralytic brachial neuritis : a clinical and electrodiagnostic study. *Neurology*, 1969, *19*, 1153.

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