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Neuralgic amyotrophy after administration of tetanus toxoid

Sir: I report a case of neuralgic shoulder amyotrophy. This condition has been described after passive immunisation against tetanus with horse-serum, and was formerly referred to as "serum neuritis". There are very few cases in the literature¹⁻⁴ mentioning this condition after active immunisation with tetanus toxoid. The onset usually follows booster-immunisation. The syndrome consists of severe neuralgia followed by the onset of paresis in the related muscles of the shoulder girdle. The pattern of paresis is peripheral, not radicular, and the prognosis is generally considered to be fair. Diagnosis is usually dependent on the natural course⁵ because there are no specific findings to enable the physician to come to an early diagnosis.

A twenty-year-old soldier, who had just joined the Army, was routinely administered two tetanus toxoid vaccinations at a four-week interval. Two weeks after the administration of the "booster-shot" he noticed the sudden onset of severe left-sided neuralgia spreading to the left shoulder muscles and into the left biceps. At the same time slight right-sided shoulder neuralgia was noticed. Four days after the onset of the neuralgic syndrome almost total paresis of the left deltoid muscle occurred. The soldier was referred to a military hospital, where a cervical CT scan suggested a left-sided cervical disc protrusion extending into the left intervertebral foramen at the level C 5/C 6. This was thought to be consistent with a diagnosis of a left-sided C 6 root compression syndrome and the patient was referred to our neurosurgical unit to undergo a cervical Cloward procedure. On admission the patient presented with a complete paresis on the left deltoid muscle, a severe weakness of the left triceps, and moderate weakness of the left pronator teres. The left triceps reflex was absent with all other reflexes being normal. No sensory deficit could be found and thorough neurological examination revealed no other abnormalities. Electromyographic studies showed denervation of the affected muscles the sensory pathways not being affected. We repeated the cervical CT scan and performed a cervical myelogram with consecutive CT,⁶ both of which were normal. During the first week as an inpatient the neuralgia gradually ceased. As all laboratory findings (CSF, immune electrophoresis) were normal, we made the diagnosis of neuralgic shoulder amyotrophy after tetanus toxoid administration.

The few cases in the literature describing the condition of neuralgic shoulder amyotrophy after tetanus toxoid seem to share a similar pattern. The patients were usually young, healthy subjects, all of whom had been vaccinated against tetanus several times before.⁷ It was always the "booster-shot" that led to the onset of the neuralgic amyotrophy.

JÜRGEN CW KIWIT
Abt. XII/Neurochirurgie,
Bundeswehrzentralrankenhaus,
Rübenacher Straße 170,
5400 Koblenz,
West Germany

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Painful hyperaesthesia following resection of the lateral cutaneous nerve of the thigh

Sir: A patient is described in whom hyperaesthesia followed resection of a peripheral nerve. Functional reorganisation of the dorsal horn neurons seemed to be the best explanation for this phenomenon.

In 1980 the patient, a 25-year-old married woman, fell down in the street, fractured the processus transversus of the fourth lumbar vertebra, and developed a haematoma in the left thigh. The present illness began in May 1981 with burning pain and paraesthesiae in the left thigh. In July 1981 a neurologist found that she had a hyperaesthetic painful area which covered the region innervated by the lateral femoral cutaneous nerve. Clinical examination and electromyography revealed that the lesion was situated at the peripheral level. There was no denervation activity in the muscles innervated by roots L2-4. On the left side the sensory evoked potential was not large enough to be measured. The diagnosis of meralgia paraesthetica was made, with an assumption that it was due to trauma.

Since conservative therapy with mild analgesics and physical therapy proved unsuccessful an orthopedic surgeon was consulted in October 1981; he resected a 20 cm long piece of the affected nerve. After the operation the hyperaesthetic region was much smaller, but within a few weeks the hyperaesthetic area began to grow until it was of the original size. It was suspected that there were still some nerve branches left. Therefore, local anaesthetic was injected at the inguinal level; after this injection the size of the hyperaesthetic region decreased about 50%, confirming the suspicion. The patient was referred for re-exploration. The orthopedic surgeon found that just distally to the inguinal ligament the nerve divided into two branches. The medial branch had been resected in the first operation and there were no connections from the resected nerve stump which could explain the regrowth of the hyperaesthetic area. An

intact lateral branch of the nerve was found which was now resected. The main nerve trunk was cut as proximally as possible (several cm). After this new operation the hyperaesthetic area was considerably smaller although still present. However, within a few weeks the size of the hyperaesthetic area again grew and reached the original size. Three months after the reoperation the area of the left lateral femoral cutaneous nerve was so painful that the patient had much trouble wearing clothes. Detection thresholds tested with a piece of cotton were higher on the affected side; but even a light touch with fingers produced considerable pain. The patient did not discriminate between warm and cold in the affected region. There was no false localisation.

There remained a small oval area (diameter about 2 cm) in the middle of the affected region which was totally anaesthetic. In addition, the patient had another, qualitatively different, lancinating pain which disappeared after injection of local anaesthetics at the inguinal level; therefore this pain was considered to be due to a neuroma. Even a massive injection of local anaesthetic at the inguinal level did not affect the hyperaesthesia in the left thigh. Analgesics, carbamazepine, amitriptyline, propranolol and guanethidine proved ineffective. Transcutaneous electrical nerve stimulation applied to the right (normal) thigh made the pain and hyperaesthesia worse, even when it was applied at low intensities. Sympathetic blockade at the L2-4 level produced temporary relief of the lancinating component of the pain.

Regeneration of the resected nerve does not explain the present findings. At reoperation it was found that the resected nerve branch did not reach the periphery and, moreover, it was sectioned again. A plausible explanation could be the phenomenon described by Devor and Wall in rats and cats:^{1,2} transection of a peripheral nerve produces a reorganisation of the receptive fields of the spinal dorsal horn neurons. This reorganisation is based on unmasking of normally present but ineffective afferent terminals, through which the dorsal horn neurons receive impulses from the neighbouring intact skin nerves after the transection. Trophic mechanisms most probably have a major role in this unmasking,^{1,2} although other kinds of mechanisms, such as the inhibition by the lateral division of Lissauer's tract,³ may contribute. The central connections of the corresponding dorsal horn neurons remain intact if the transection is made distally to the soma of the primary afferent neurons.⁴

In the present case a nerve lesion at the peripheral level had first produced meralgia paraesthetica. After nerve resection, the dorsal horn neurons which normally mediated signals from the resected nerve began to mediate signals from the neighbouring intact skin nerves which either had pre-existing overlap of innervation areas covering the denervated region or, more probably, had sprouted to the denervated area.⁵ Small diameter afferents presumably are a dominant group in the sprouting nerves (cf ref. 5) which could explain the higher detection thresholds in the hyperaesthetic region. The production of pain by light touch could have been caused by lack of afferent inhibitory control at the spinal level. Lack of false localisation supports the theory that signals from the hyperaesthetic region are mediated via second order neurons of the affected nerve. Our case resembles the cases described by Noordenbos and Wall⁶ and reconfirms their recommendation that a resection should not be done in this kind of condition.

T NURMIKKO

Department of Neurology,
University of Tampere,
33520 Tampere 52

A PERTOVAARA

Department of Physiology,
University of Helsinki,
00170 Helsinki 17, Finland

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Cryptococcal meningitis for 15 years

Sir: A case of cryptococcal meningitis is reported where the diagnosis was made, after 14 years of a chronic relapsing illness, by the isolation of *Cryptococcus neoformans* from the CSF. The organism was non-encapsulated, a finding that may be related to the relatively benign clinical course.

Mr C was an Indian, born in Bombay in 1934, who came to this country at the age of 26 years. He first presented in September 1966 with headaches and a left lateral rectus palsy which resolved after three weeks. At that time chest and skull radiographs, ESR, WR and full blood count were all normal. In June 1967 he was seen with a right hemiparesis of sudden onset which subsequently improved. Routine biochemical and haematological tests were again normal but his cerebrospinal fluid showed a protein of 1.6 gm/l, WBC 70/mm³ mainly lymphocytes, and a glucose of 1.0 mmol/l. An air encephalogram, EEG and isotope brain scan were normal. CSF culture was negative. No firm diagnosis was made at this time but he remained generally well; he was seen once in 1971 with sacro-iliac pain which settled spontaneously, but was then lost to follow-up until 1978. In 1978 he presented with an episode of vertigo related to head posture which improved over four weeks. At that time he had mild residual right upper motor neurone signs with bilaterally brisk reflexes. In August 1979 he had some perioral and right arm paraesthesiae which lasted for a few days. Following this a CT scan showed a communicating obstructive hydrocephalus with symmetrical enlargement of the third, fourth and lateral ventricles but no other lesion. Again routine biochemistry, haematology, skull and chest radiographs were normal, but the CSF showed a pressure of 14 cm of CSF, protein 1.3 g/l, WBC 20/mm³ mainly lymphocytes and a glucose of 0.6 mmol/l (blood glucose 4.3 mmol/l); culture, cryptococcal antigen and India ink stain were all negative. A Mantoux test was positive and a Kveim test negative, making sarcoid unlikely. He was then started on anti-tuberculous chemotherapy comprising streptomycin 1G, ethambutol 1G, rifampicin 600 mg, isoniazid 300 mg and pyridoxine 50 mg daily. However, by January 1980 all TB cultures had proved negative and he had developed visual impairment, numbness of his feet and vertigo possibly related to the ethambutol, isoniazid and streptomycin respectively. The anti-tuberculous chemotherapy therefore was stopped. In February 1980 he relapsed with malaise and headaches, so was started on prednisolone 60 mg a day