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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.

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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