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Case Report Open Access

Tourniquet Paralysis may Reflect Injury of Muscle Spindles

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Abstract

Objective: Knee operation using pneumatic tourniquet may cause weakness of the quadriceps femoris muscle. A patient with a paresis of the quadriceps femoris muscle for two months postoperatively was studied.

Methods: ENMG and lumbar magnetic stimulation (MEP) follow-up.

Results: There was no distinct lesion of the motor axons of the femoral or other nerves in ENMG or MEP six weeks postoperatively. After a follow-up of eight months, the paresis was subsided and a complete recovery took place in two years.

Conclusion: High pressure of the tourniquet may injure muscle spindles. Decreased Ia-afferent activity may severely inhibit voluntary motor effort.

Keywords: Tourniquet paresis; ENMG; Motor evoked potential; Muscle spindle; I_a-afferent; Knee surgery

Introduction

The advantage of femoral pneumatic tourniquet in knee operation is a bloodless surgical field. However, the use of a tourniquet may cause hemodynamic, metabolic or local complications [1]. Some patients suffer from postoperative thigh weakness, especially when a high tourniquet pressure and long tourniquet time are used [2]. In a systematic ENMG study of patients with anterior crucial ligament operations with tourniquet, the changes in neurography were slight, when pre- and post-operative investigations were compared. The most marked change was seen in the EMG interference pattern of maximum voluntary effort of the vastus medialis muscle. The maximum voluntary activation was reduced to about 50 % postoperatively, compared with the preoperative values. None of these patients suffered from any distinct weakness of the quadriceps femoris muscle. There were no postoperative signs of axonal destruction, such as fibrillation potentials, in needle EMG [3]. A case of a patient with a nearly total bilateral paresis of the quadriceps femoris muscles six weeks after a bilateral knee operation with pneumatic tourniquet is described.

Case Report

A 40 year old athletic man was operated because of bilateral patellar chondrodysplasia. The main symptom was pain caused by extension of the knee joint when rising up from squatting position. A day-surgery operation (DBX-reconstruction) [4] was performed bilaterally using pneumatic tourniquet (dx, 52 min; sin, 43 min), with a relatively high pressure level (250 mm Hg on the right side, the pressure level on the left side is not known). When anaesthesia subsided the patient felt unbearable pain in the lower extremities. He required four extra days of hospitalization. Pain medication had to be continued for 16 days. The postoperative phase was complicated by a total bilateral paresis of the quadriceps femoris muscles. Initially, the paresis was not observed, because the patient had to avoid squatting, i.e., flexion of the knee joint with pressure. In the outpatient ward five weeks postoperatively, the patient walked clumsily with a pair of crutches and kept full extension of the knees. He complained of pain in the lower extremities and there was a suspicion of venous thrombosis of the legs. In the sitting position the patient could not straighten his legs. From the supine position the patient could not elevate the straight lower extremities from the bed. Sensibility was normal as well as the function of the foot and toe muscles. There was no edema of the muscles. Venous thrombosis was not found in the ultrasonography study. The lumbar MRI was normal.

ENMG study was performed six weeks postoperatively because the bilateral quadriceps femoris paresis was not relieved (Medelec Synergy, Oxford Instruments Medical, Old Woking, Surrey, U.K.). The patient also had persistent pain and allodynia of the knee and lower leg region. Patellar reflex study could not be performed because of patellar allodynia and pain.

Motor conduction velocities and F-responses of the deep peroneal nerves were bilaterally normal. The motor response of the femoral nerve in the right vastus medialis muscle could not be recorded because of pain elicited by electric stimuli in the thigh. The other sites studied were not painful and sensory conduction velocities and amplitudes of the responses of the right superficial peroneal nerve, as well as bilateral saphenous and sural nerves were normal. The H-reflexes of the tibial nerves were normal. In needle EMG, there were occasional fibrillation potentials in the vastus medialis and lateralis muscles bilaterally, but the insertion activity was otherwise normal. With maximal effort the patient could activate only 1-2 motor unit potentials for 2-3 s in both vastus medialis and the left vastus lateralis muscle. The right vastus lateralis muscle was totally paretic. In contrast with the vasti muscles, the right iliopsoas muscle and the tibialis anterior muscles were activated normally with a full interference pattern and there was no fibrillation activity. In the right extensor

hallucis longus muscle severe pain was caused by the EMG needle, which had to be removed soon, but there were no fibrillation potentials and the insertion activity was normal. EMG study was not painful in the other muscles studied.

Because the M-response of the femoral nerves could not be studied with electric stimuli, a lumbar MEP study was performed, with surface recording of the vastus lateralis (Figure 1) and vastus medialis muscles (Magstim, Whitland, Wales, U.K.), as well as the anterior tibial muscles. The latencies and amplitudes of the MEP responses were normal according to the control values of our laboratory.

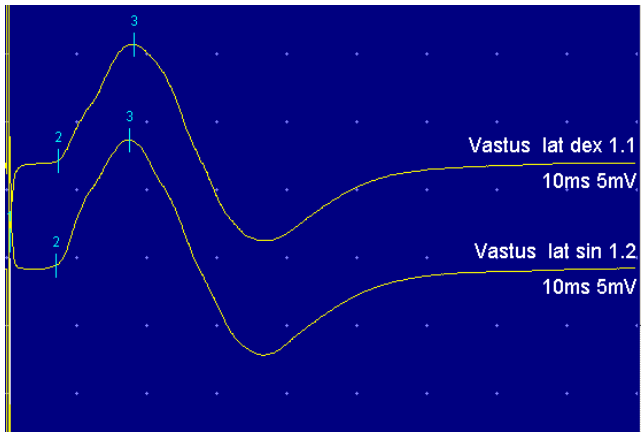

Figure 1: Bilateral MEP-responses of the vastus lateralis muscles six weeks postoperatively. The latency left vs. right was 6.8 and 6.9 ms and the amplitude 8.6 and 9.2 mV, respectively. The magnetic stimulation was performed to the lumbar L 3-4 level.

The patient trained the thigh muscles and 12 weeks postoperatively he could manage 10 kg with horizontal foot press. In the outpatient ward 14 weeks, postoperatively the strength of the thigh muscles was further improved and the patient could press a weight of 30 kg by stretching the legs from the angle of 110 to 180 degrees. The patient still used crutches because of fatigue of the thigh muscles during a long walk. Follow-up ENMG and MEP studies were performed 9 months postoperatively. The patient walked normally and he had returned to his work as a warehouseman. There were still problems with heavy work; the thigh muscles had not regained full endurance. The patellar reflexes were weak, but could be elicited bilaterally. The vastus lateralis and medialis muscles were slightly atrophic and activated with a fair but not full interference pattern. There were no fibrillation potentials and the configuration and amplitude of the motor unit potentials were normal. The MEP latencies were normal from the lumbar root level to the vastus lateralis (Figure 2) and vastus medialis muscles. The amplitudes were decreased, compared to the previous values. The patient complained of pain in the left foot and in an ENMG study two years postoperatively EMG of the right vastus lateralis, left gastrocnemius and left interosseus dorsalis pedis IV muscles were normal, as well as the paraspinal muscles at the left L5 and S1 levels. Motor conduction values and F-responses of the left deep peroneal nerves and H-reflexes of the tibial nerves were normal. The sensory conduction values of the left superficial peroneal and sural nerves were normal. In quantitative sensory testing (QST) of the upper and lower extremities, the sensory thermal and vibration thresholds were normal, but there was cold allodynia in the dorso-lateral foot area bilaterally: The cold sensory threshold dx/sin was 26.1/29.7°C and pain threshold 22.7/26.6°C, respectively (normal cold sensory threshold is >25.3°C and cold pain threshold is <16.2°C in our laboratory).

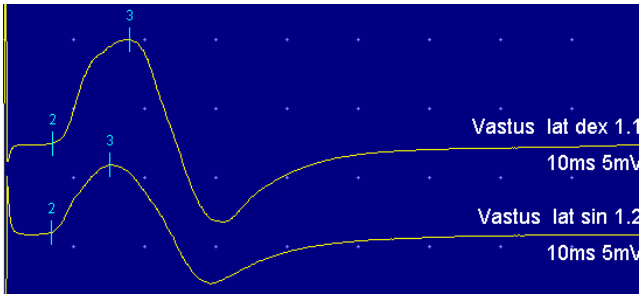

Figure 2: Bilateral MEP-responses of the vastus lateralis muscles 9 months postoperatively. The latency left vs. right was 6.8 and 6.6 ms and the amplitude 7.5 and 4.9 mV, respectively.

Discussion

Postoperative weakness of the quadriceps femoris muscle after operations using pneumatic tourniquet are common and the weakness has been attributed to axonal compression syndrome [2]. The patient described here is an extreme example: a nearly total bilateral postoperative paresis of the quadriceps femoris muscles for two months and a gradual improvement thereafter. The neurophysiological studies could not reveal any distinct axonal compression syndrome: the M-responses of the vasti muscles studied with lumbar MEP were normal in spite of the nearly total inability to activate these muscles voluntarily. There were only occasional fibrillation potentials, but axonal destruction should elicit abundant fibrillation activity in six weeks [5]. The saphenous nerve conduction values were normal indicating intact A beta fibers in the femoral nerves. Nerve conduction, as well as F- and H-responses were normal in the other leg nerves, without any signs of axonal involvement or conduction block of the motor nerve fibers or sensory A alpha or A beta nerve fibers. Pain and allodynia in the lower extremity pointed out involvement of thin sensory A delta and C nerve fibers. In the follow-up, there was fair but not full interference pattern in the vasti muscles eight months postoperatively and a complete normalization of EMG of the right vastus medialis muscle was observed two years postoperatively. There were no signs of collateral reinnervation in the configuration or amplitude of motor unit potentials of the vastus medialis muscle. The lumbar MEP latencies were normal, but decreased amplitude in the follow-up study 9 months postoperatively could reflect disuse atrophy. QST study was not performed in the acute stage, but QST two years postoperatively did not reveal any residual neuropathy of the thin sensory nerve fibers, but there was hypersensitivity of the A delta fibers presenting as altered cold pain threshold on the dorso-lateral foot areas.

Experimental studies have revealed regional necrosis of muscle fibers below the pneumatic tourniquet with a clinically relevant cuff inflation pressure [6]. However, the probable destruction of muscle spindles beneath the tourniquet was not studied. Different muscle fibers may have different sensitivities to pressure. The Type IIB fast glycolytic fibers seem to be most vulnerable [6]. The long chain fibers of the muscle spindles are innervated by static beta motor axons, which also innervate the fast glycolytic extrafusal muscle fibers [7]. Thus the

long chain fibers may be especially vulnerable to even clinically relevant pressure levels. It is conceivable, that the high pressure of the pneumatic tourniquet affected the proprioceptive afferent function, especially the Ia and II-afferents of the thigh muscle spindles of the patient. Muscle spindles are surrounded by a tight membrane and the intrafusal capillaries are poorly permeable, compared to the extrafusal ones [8]. This may predispose the development of "intrafusal compartment syndrome", which may even lead to severe hypoxia and finally necrosis of the muscle spindle beneath the tourniquet. Muscle spindles and spindle capsules have somatic C-nerve fibers [9] and damage to the spindles may cause postoperative pain. The long nuclear chain fibers extend out of the sleeves of the muscle spindles [10]. If the spindles are in necrosis, the denervation of these relatively few but vital pieces of muscle fibers may be the reason for occasional fibrillation potentials, observed in EMG of the quadriceps femoris muscles of the patient.

How the poor postoperative voluntary activation of the quadriceps femoris muscles can be explained? Motor neuron exitability is normally adjusted by persistent inward currents (PICs). The PIC is subject to regulation by a descending neuromodulatory system releasing monoamines serotonin and noradrenaline. Without PIC, the motor neurone exitability is very low. But the focusing of descending PICs is poor and needs local synaptic integration to meet the demands of different motor tasks. The PIC is exquisitely sensitive to reciprocal inhibition mediated by the length sensitive muscle spindle Ia afferents and Ia interneurons [10].

Conclusion

Thus, the reciprocal inhibition from posterior thigh muscles may cause the initial paresis on the quadriceps femoris muscles, which lack the corresponding proprioceptive activity because of the decreased muscle spindle afferent firing. On the other hand, motor neuron activation may also require proprioceptive activity from the muscle. Acute decrease of afferent firing seems to cause extreme inhibition of muscle activity in voluntary effort, as seen in acute sensory axonal neuropathy [11]. The alleviation of symptoms takes several weeks, as in our patient. Thus it is recommended, that the tourniquet pressure should be kept as low as possible in knee operations. Anterior cruciate ligament surgery may even be performed without the use of tourniquet [12].

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