The progression of lesions in synovial chondromatosis has some similarities. In early phases, there is a proliferation of stem cells in the stratum synoviale. Later, cells resembling fibroblasts form a primitive chondroid matrix and they are metabolically active. Then they remain free as a loose body and this condition may be followed by resolution or full resorption. Histological examination shows fibroblasts in the subintimal layer of synovium that undergo cartilaginous metaplasia, giving rise to multiple nodules of hyaline cartilage (Murphy et al. 1962, Milgram 1977, Ono et al. 1994).

If cellular activity exists in the foci of osteopoikilosis, it is conceivable that a malignant lesion could develop (Mindell et al. 1978, Van de Stadt et al. 1984, Grimer et al. 1989). This is also true of synovial chondromatosis (Murphy et al. 1962, Lichtenstein 1977, Milgram 1977, Ono et al. 1994).

In 1977, Verbov described dermatofibrosis lenticularis disseminata as the skin manifestation of osteopoikilosis. By taking into account the similarities, we can speculate that chondromatosis is the synovial manifestation of osteopoikilosis (synosteopoikilosis) and increasing evidence suggests that, when osteopokilosis is detected in a case, other associated disorders, especially lesions of fibroproliferative origin, should be sought.

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Slow-onset subdural hematoma, evolving into paraplegia, after attempted spinal anesthesia—a case report

Paolo Cabitza and Matteo Parrini

5° Orthopaedic Department of the University of Milan Medical School, Ospedale Clinicizzato San Donato, Milan, Italy. Correspondence: Dr. P. Cabitza, Viale Sabotino, 13, IT-20135 Milan, Italy. Tel +39-2-58319234. Fax -55600234
E-mail: paolo.cabitza@unimi.it
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Epidural and subdural hematomas after spinal anesthesia are rare and are reported mainly as a consequence of needle-induced trauma during a difficult procedure and in patients with abnormal coagulation (Dupeyrat et al. 1990, Sanama et al. 1992, Coquet et al. 1993, Taylor and Major 1993). The first signs of this complication are vague and indistinguishable from the frequent symptoms of meningeal irritation and liquoral hypotension that often follow this type of anesthesia (Tarkkila and Kaukinen 1991, Carpenter et al. 1992, Lynch et al. 1994, Dahlgren and Tornebrandt 1995, Lomartire et al. 1995). Since the onset of an epidural or subdural hematoma can have serious neurological consequences, in the worst case irreversible paraplegia, it is important to recognize the first symptoms.

We observed a rare case of delayed subdural hematoma, which resulted in paraplegia after attempted spinal anesthesia.

MRI of the lumbosacral segment.

T1 weighted sequence, axial. Blood in the posterior subdural space at L1–L2 and L2–L3 leves. Note that the inferior portion of the conus is slightly deformed.

T1 weighted sequence, sagittal.

Case report

A 75-year-old woman was admitted to our department for a fracture of the right patella requiring surgical osteosynthesis. Due to the age of the patient and to the lack of specific contraindications, spinal anesthesia was planned. No antithrombotic prophylaxis was given. The procedure was difficult to carry out and after 3 attempts at puncture at L2–L3 level using a 22 gauge needle, it was given up (no bleeding was observed). Reduction and osteosynthesis were performed under general anesthesia, without any further problem (neither hypo- nor hypertensive episodes nor blood loss; a tourniquet was applied for 40 minutes). The leg was put in a plaster leaving the foot free. The following morning the patient complained of sciatic pain and weakness of her left foot with no sensory or reflex loss; both symptoms resolved spontaneously in a few hours.

On the third postoperative day, the patient presented with slight rigidity of her neck, mental confusion and fever, but showed no peripheral neurological defects. The consulting neurologist diagnosed a state of meningeal irritation due to the attempted spinal anesthesia and he prescribed antibiotic, and antinflammatory drugs. In the following days, a slow but steady improvement in the clinical signs was reported but, on the twelfth postoperative day the patient suddenly developed bilateral lower extremity paralysis. MRI of the thoracic and lumbar spine showed blood in the subdural space, with slight deformation of the conus at the L1–L2 level (Figure). The patient underwent a spinal exploration and decompression at the L1–L2 and L2–L3 levels (a subdural hematoma was present). She never recovered from the paraplegia.

Discussion

No patient with a spinal hematoma has been reported in over 150,000 spinal or epidural anesthesias (10,000, if we only consider controlled studies) (Lund 1966, Gustafsson et al.1988, Modig 1992). Irreversible neurological sequelae are even more unusual and, among them, evolution into paraplegia can be considered exceptional: 7 such cases have been reported in more than 1,720,000 cases of spinal and epidural procedures (Bonica et al. 1957, Moore and Bridenbaugh 1966, Bergquist et al. 1992, Aromaa et al. 1997). 3 paraplegias caused by spinal hematomas (in 17,733 central blocks) have occurred in patients with an altered hemostatic capacity (Dahlgren and Tornebrandt 1995).

Paraplegia seldom occurs several days after lumbar puncture, while Pryle et al. (1996) reported such a

complication after 8 days. The case that we describe is the first to develop a paraplegia 12 days after attempted lumbar puncture.

This complication is notorious for its lack of specific early signs, resembling in all details those resulting from meningeal irritation or hypotension in the cerebrospinal fluid system. Both such complications are relatively common sequelae of lumbar puncture (0.5%–3.5%) and are usually benign, disappearing in a few days, generally within a couple of weeks (Phillips et al. 1969, Kane 1981). Radiating back pain, sensory changes and muscular weakness that progress to paraplegia should arouse suspicion of an epidural hematoma (Gustafsson et al. 1988). CT cannot always detect the presence of a hematoma, as demonstrated in two cases reported by Dupeyrat et al. (1990) and Coquet et al. (1993) while MRI has higher diagnostic value (Bergquist et al. 1992). In our case, the agitation of the patient made MRI examination difficult and the patient had to be reexamined a few hours after the first attempt to obtain diagnostic images.

If induction of lumbar anesthesia is difficult, requiring multiple punctures, the presence of blood in the needle, the use of large or traumatic needles, especially in association with blood dyscrasias, thrombocytopenia, antiplatelet therapy and anticoagulation, can increase the risk of bleeding and even of this complication (Gustafsson et al. 1988, Coquet et al. 1993). In the first hours following anesthesia, the patient should be monitored for any symptom of meningeal irritation and any sensory abnormality, especially in the saddle region. In case of a symptomatic medullary compression, local spinal decompression should be performed without delay, even though many cases of spontaneous resolution of such symptoms are reported (Boukobza et al. 1994).

After the onset of motor deficits, less than half of the reported cases recovered following surgery, often only partially (Owens et al. 1986, Dupeyrat et al. 1990).

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