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Multiple adverse reactions following metrizamide myelography

Sir: Metrizamide, a nonionic contrast medium, is still widely used for routine myelography and cisternography although there are now other agents such as iohexol with fewer complications and less expensive.1 There are several reports based on human and animal observations concerning metrizamide’s adverse reactions;2–11 however, from the work of Killebrew et al12 it appears that there may still be side effects which have not yet been adequately emphasised. We report a patient with a mild cervical cord lesion who developed generalised convulsions, neurobehavioural disturbances and persistent myelopathy following metrizamide lumbar myelography.

A 50-year-old farmer was admitted with a history of having woken up 15 days earlier complaining of numbness, pins and needles in the arms and legs and mild difficulty in using both upper and lower limbs, without any bladder dysfunction. He had been well the day before and there was no relevant past history. On examination he had mild pyramidal signs in all four limbs consisting of increased tendon reflexes, sustained ankle clonus on the right and unsustained on the left, mild difficulty in fine movements of both hands without any detectable weakness in the muscles and equivocal plantar reflexes. The cranial nerves were intact, the abdominal reflexes were present, there were no cerebellar signs or objective sensory disturbances. Normal laboratory data, including blood count, erythrocyte sedimentation rate, blood urea and electrolytes, VDRL, serum B12, urine analysis, ECG, chest radiographs, computed brain tomography and CSF obtained during myelography. Blood sugar was slightly increased and the glucose tolerance test showed mild diabetes mellitus. Plain cervical radiographs showed degenerative changes and small posterior osteophytes between C1–C2 and C4–C5.

The patient’s symptoms and signs remained unchanged for the following three weeks. Then lumbar myelography was carried out. Three g (0.33 g/ml) of metrizamide were administered. This showed narrowing of the spinal canal in the cervical region and indentation of the contrast medium at the level of C2–C3 and C4–C5, due to posterior osteophytes. The contrast medium passed freely through the cervical canal. During the investigation there was intracranial spill of the contrast medium with high concentration of metrizamide in the posterior fossa. Twenty minutes later the patient had severe generalised convulsions. He was treated with intravenous diazepam, phenytoin and steroids and in the next few hours he had three more generalised seizures with an hour interval between. For the following 24 hours he was severely confused and gradually by the end of 48 hours he recovered. The next morning following the myelogram he was found to have considerable deterioration of his spastic tetraparesis with urinary retention and he was catheterised for a few days. He had moderate to severe weakness of shoulder girdle muscles which gradually became atrophic with widespread and frequent fasciculations. He also had a sensory level to pin-prick at C4. The patient refused at that time to undergo a cervical laminectomy. Although his clinical state remained unchanged, a decompressive cervical laminectomy at C2–C3 and C4–C5 level was carried out 3 months later in another hospital. Seven months later the patient’s clinical condition has not changed.

Headache, nausea and vomiting are fairly frequent side effects following metrizamide myelography. Other less common adverse reactions include seizures, diplopia, menigism, fever, neuro-behavioural disorders (hallucinations, confusion, anxiety and disorientation) and EEG changes. Lower extremity myoclonus has been reported as a very rare complication.12 All adverse reactions of metrizamide have been reversible and usually do not last longer than a few days. Metrizamide’s adverse reactions are probably due to meningeal-neuronal cerebral and spinal irritation and it has been shown that metrizamide penetrates the brain substance.14 There may be several explanations for our patient’s sudden deterioration of cervical myelopathy. The sudden onset of the spinal symptoms in addition to the normal CSF suggests probably a vascular mechanism. Demyelination is rather unlikely as there have been no previous episodes and the patient was over 50 years of age. Deterioration due to the lumbar puncture itself on myelography was not possible as the cervical spondylosis was not severe and there was no spinal block. The patient’s sudden deterioration may have been caused by a vascular accident at myelography or during the generalised convulsion; on the other hand myelopathy due to metrizamide cannot be excluded and could be the possible explanation particularly in a patient apparently sensitive to metrizamide. All the reported metrizamide’s adverse reactions due to toxic encephalopathy are reversible within a few days. Reports of cases with spinal irritation or myelopathy due to metrizamide are very rare and it is possible that this reaction may last much longer.13

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Th PAPAPETROPOULOS

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The role of childbirth on the natural history of occult spinal dysraphism

Sirs: Occult spinal dysraphism is the term used for a variety of embryological malformations of the spinal neural structures and neighboring tissues which, in contrast with spinal bifida aperta, are less or not obvious externally. The condition may go unnoticed for many years, especially when symptoms start in adult life. We report the case of a woman with a lumbosacral lipoma associated with a widened dural sac and posterior spina bifida, in whom urinary symptoms developed shortly after the birth of her first child and exacerbated after a second childbirth. This unusual presentation led to an initial incorrect diagnosis of post-partum stress incontinence and resulted in a needless urological operation.

A 36-year-old woman was referred to the urology clinic because of unexplained urinary sphincter disturbances. Symptoms started at age 30, when after the delivery of her first child, she intermittently noted loss of urine during sexual intercourse. Three years later, she gave birth to a second son, and soon afterwards, she complained of urinary frequency and loss of urine during physical strain. Symptoms were attributed to stress incontinence due to the successive deliveries. Pelvic floor exercises were unsuccessful and her gynaecologist decided to perform an anterior colporrhaphy and urethroplasty. After the intervention, she had urinary retention and overflow incontinence. Since then she has required intermittent self-catheterisation. Urodynamic examination revealed a hypotonic bladder with vesico-urethral dysfunction.

She was then referred to the urology department. For the first time, she admitted to have noticed minimal faecal incontinence during recent months. Although she only complained of sphincter disturbances, examination showed saddle hypesthesia (S5 to S3 on the right, and S5 to S2 on the left), absent ankle jerks, a hypotonic anal sphincter, bilateral vesical reflex and a small operative scar at the lumbosacral region, from the removal of a small "skin nodule" shortly after her birth.

Radiographs of the lumbosacral spine showed an enlarged canal at the lower lumbar spine and sacral levels, with posterior sacral spine bifida. Myelography with metrizamide showed a marked widening of the sacral dural sac which contained a multiloculated mass. Combined computed tomographic scanning revealed that the mass had a density of fat (fig). There was no evidence of a tethered cord since the conus terminated at the lower edge of L1.

Surgical exploration confirmed the radiological findings. The lipoma had no extension outside the dural sac. Because the lipoma was merged with the nerve roots, only a small proportion could be dissected. The thin filum terminale was sectioned, although it was not under traction. Histological examination showed normal adult adipose tissue. Postoperatively, the sphincter disturbances remained unchanged.

Stress incontinence is a common manifestation of a neurogenic bladder. Stress incontinence occurring after childbirth is usually due to weakness of supporting tissues of the urethra and base of the bladder. Our patient suffered stress incontinence from an autonomous neurogenic bladder, but the relation of this symptom to the successive deliveries initially suggested the latter cause to be responsible.

A few other anecdotal cases of occult spinal dysraphism, in which symptoms started after childbirth, have been reported. Pool described a case which shows some similarities with ours. His patient had an intrasacral meningocele with a lipoma that compressed the adjacent nerve roots and tethered the spinal cord. Symptoms started at age 22 years, when after her first childbirth, she developed pain and paraesthesia in the legs. Eight years later, she developed urinary frequency followed by stress incontinence. She also underwent a needless urological intervention. Joseph and McKenzie reported the case of a 27-year-old woman with an occult intrasacral meningocele who developed symptoms shortly after the delivery of her fourth child. Although the spinal cord was tethered by a small terminal lipoma, surgical intervention was restricted to bony decompression. Postoperatively there was rapid regression of symptoms and abnormal signs. They postulated that the intensive straining during childbirth could have caused the arachnoid to herniate through a possible dural defect. In their series of adults with a tethered cord syndrome, Pang and Wilberger included three female patients in whom symptoms started shortly after childbirth. Injury of the conus, due to traction during the straining associated with childbirth, is the postulated mechanism in these patients.

In our patient there was no evidence of a tethered conus, but the sacral roots were compressed by the lipoma. Normally, when a traction is applied on the lumbosacral roots, they can to some extent move out of their foramina. During straight leg raising it has been demonstrated that the L5 and S1 roots can move out of their foramina for some millimeters. In our case, the lower sacral roots were merged with the lipoma, so that outwards movement through their foramina was impossible. During the straining of childbirth, momentary stretching of the tethered sacral roots could have caused permanent injury to them. Another factor to be considered is that the condition of pregnancy itself could have played a role in the growth of the lipoma. It is well
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