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Spinal disease presenting as acute abdominal pain: Report of two cases

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Lesson of the Week

Spinal disease presenting as acute abdominal pain: report of two cases

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Successive generations of surgeons have been taught the importance of considering spinal disease in the differential diagnosis of abdominal pain[1] but with the passing of tabes dorsalis as a clinical problem and the increasing rarity of infective lesions of the spine the lesson seems to require reinforcing. We recently managed two patients with spinal lesions who initially presented with abdominal pain leading in both cases to an inappropriate surgical exploration before the onset of cord compression revealed the true nature of the problem.

Lesions of the dorsal spine may present as abdominal pain before the onset of myelopathy and this may result in an inappropriate laparotomy. It is important to bear the syndrome in mind, particularly in diabetics and children

Case reports

Case 1—A 48 year old man with mild diabetes was admitted to hospital on 4 September 1982 with severe right hypochondrial pain and fever. His diabetes had been controlled by diet alone and he was well until three weeks before admission when he developed lower dorsal back pain which progressively increased in severity and became girdle-like with radiation to the front of the trunk, particularly on the right. In the three days before admission the anterior component of his pains became exacerbated and in addition he had fever, sweating, and nausea. Though he looked unwell there were no unequivocal abdominal signs and he was observed for 24 hours. His haemoglobin concentration was 13 g/dl, the white cell count was $12{\cdot}0 \times 10^9$/l, and the blood sugar concentration 12·5 mmol/l (225 mg/100 ml). His pains continued and in addition the abdomen distended with dilated loops of bowel seen on a radiograph. Treatment was started with metformin and he underwent an exploratory laparotomy which gave negative results. His back pain continued and he developed an abdominal wound infection but was allowed home, only to be readmitted on 29 September. On this occasion his dorsal pain radiated to the lower chest on the left and was associated with pain and weakness in the left leg. Deep venous thrombosis with pulmonary embolism was suspected and he was given anticoagulants. This treatment was discontinued two days later when findings of the chest *x* ray examination and a ventilation-perfusion scan failed to substantiate the diagnosis. The weakness of his leg became a paraparesis with extensor plantar responses. On 7 October he was noted to have a sensory level at D7 and was paraplegic and incontinent of urine. A myelogram now showed a complete block at D9; laminectomy was undertaken immediately, and an extradural abscess was evacuated. *Staphylococcus aureus* was grown from the pus. His pains have subsequently abated but he remains paraplegic and incontinent.

Case 2—A 73 year old woman was admitted with spinal cord compression. She gave an eight year history of lower dorsal back pain which soon after onset began to radiate round the flank to the right iliac fossa; at times the anterior component of her pains would occur independently. Her back pains would be worst at night when she lay down and they sometimes forced her to get up and walk about. Coughing and sneezing were particular aggravating factors. During one particularly severe bout of pain of the iliac fossa, four years after onset, she was admitted to hospital with acute abdominal pain and a normal appendix was removed. She then had an intravenous pyelogram, which failed to show an abnormality. Her pain remained unchanged, though intermittent, until six months before admission when first the left and then the right leg became paretic with paraesthesiae in a sciatic distribution. Eventually sphincter failure resulted in a neurological assessment when a mild paraparesis with absent ankle jerks, extensor plantars, and a sensory level at D11 were found. Three café au lait spots were noted on the trunk. A myelogram showed an intradural extramedullary tumour at T10 and at operation a schwannoma was removed. She suffered increased weakness of the left leg immediately after operation but at follow up was walking with a Zimmer frame and was improving.

Comment

Two thirds of patients with intradural extramedullary tumours and about a third of patients with extradural lesions suffer neuralgia referred to a part of the chest, abdomen, or the extremities.[2] In lesions between the eighth and twelfth thoracic segments root pain, if it occurs, may be felt on the surface of or within the abdominal cavity. Patients with these lesions may present to the general surgeon and are likely to undergo an unnecessary laparotomy, particularly in the absence of any gross motor or sensory deficit or reflex changes.

Paying careful attention to the chronology of historical details and character of the pain is the most important element in making a correct diagnosis of the spinal lesion. The back pain in case 1 was misinterpreted as referred pain from a diseased gall bladder while case 2 gave an excellent account of pain character-

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istic of an intradural tumour, which was worse on lying down, could be walked off, and was aggravated by coughing and sneezing. The diligent examiner may find unilateral weakness of the abdominal muscles and diminution of the abdominal reflex in these cases.

Patients with diabetes are known to suffer abdominal pains from thoracic radiculopathy[3] but the onset of back pain in these patients should always suggest an epidural abscess until proved otherwise. The presentation may be subacute, as in case 1, with no objective neurological signs. Waiting for such signs to evolve may result in disastrous cord infarction. Children are another group of patients in whom recurrent abdominal pains may be the initial symptom of a spinal cord tumour and be misdiagnosed as abdominal migraine or abdominal epilepsy.[4] The well known difficulties of examining children should never prevent a neurological examination in atypical abdominal pain.

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Communicable Diseases

Plague

Prepared by the Public Health Laboratory Service Communicable Disease Surveillance Centre

There have been three worldwide pandemics of plague. The first occurred in the sixth century; the second began in the fourteenth century and during the ensuing 300 years killed millions of people in Europe, and the first attempt at quarantine began in Venice in 1377.[1] The third pandemic began in China in the 1860s and reached Hong Kong by 1894. It was at that time that the epidemiological features of the disease first began to be elucidated, and the causative organism *Yersinia pestis* (now called *Y pseudotuberculosis* ssp *pestis*) was identified simultaneously by Yersin and Kitasato.[2 3] Since then the spread of the disease has waned owing both to a natural decline and to more efficient control measures based on a scientific explanation of transmission.

Plague is one of the diseases notifiable to the World Health Organisation. Areas with such cases may be designated as infected, and restrictions may be enforced on the movements of people in and out of these areas.

Worldwide occurrence

At present plague is known to occur naturally in rodents in parts of the United States, southern Africa, south eastern Russia, central Asia, and South America. In the past decade countries in other areas—for example, Vietnam[1]—have reported cases of the disease in man; these episodes are continuations of endemic disease present since the third pandemic.

Wild (sylvatic) plague occurs in wild rodents of different types depending on the locality. In the United States ground squirrels and prairie dogs are the main reservoir, while marmots are important in central Asia, and rodents of the family Gerbillinae are important in south eastern Russia and southern Africa.[2] Direct spread may occur from wild rodents or their fleas to hunters and others in close contact with the animals. If there is an epidemic (an epizootic) in the wild rodent population then the disease may spill over into the domestic animal population or to certain wild animals which live close to man. Transmission to domestic animals in rural environments may occur even when only a few wild animals are affected—an enzootic phase.

Various rodents have been implicated in the spread of the disease from wild rodents to man, and these include the semidomesticated brown rat and bandicoots, notably in India. Domestic rodents such as mice and rats (especially *Rattus rattus* and *Mastomys natalensis*) may either be infected directly from the fleas of wild rodents or from semidomesticated rodents. Transmission from one rodent population to another takes place when the infected animal dies and its infected fleas leave it and migrate and infect another animal. A number of species of fleas and other arthropods have been implicated in this transmission, but the most important is *Xenopsylla cheopis*. An infected flea becomes "blocked" and thereupon regurgitates the plague bacilli into the creature being bitten. *Xenopsylla* appears to be particularly likely to become blocked in this way.[3] It can be seen that spillage from wild enzootics or epizootics into semidomesticated rodents would be of importance only when the latter populations are large and increasing and human contact with them is possible. This is the classic setting for epidemics of the disease in man.

Man may become infected by direct contact with infected rodents or fleas. Bubonic plague is manifested by a lymphadenitis in nodes draining the site of a flea bite or other contact and may progress to a septicaemic form. Secondary spread to the lungs, pneumonic plague, may result in man to man transmission and cases of primary pneumonic disease.

Pattern since 1970

The smallest number of cases (191) of human plague notified to the World Health Organisation since 1970 was reported in 1981, but the trend has not been continuously downward (see table). Madagascar, a country with a natural rodent focus, was the only nation in Africa consistently reporting cases since 1970. There were, however, outbreaks of the disease in Namibia, which reported 102 cases in 1974; in Kenya outbreaks occurred in 1978 and 1979; and in 1979 Sudan reported 226 suspect cases.[5-7]

In the American region up to 1976 Brazil reported large numbers of cases, and a substantial number were reported again