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LATE ILEUS IN PARAPLEGIA

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Abstract. Ileus is a common complication of spinal cord injury, resolving within one week of injury in most cases. Two cases are reported in which the ileus developed as a more serious later complication and proved to be very intractable to treatment. Causes and treatment are discussed.

Key words: Quadriplegia; Ileus.

ILEUS, a paralysis of the gastro-intestinal tract resulting in abdominal distension, is a common complication of spinal cord injury, especially in the upper thoracic and cervical levels of the cord. It is uncommon in injuries of the lower dorsal and lumbar levels of the cord. It is difficult to understand the mechanism of development of this complication in high injuries.

Peristalsis is governed by the usual antagonistic action between the two elements of the autonomic nervous system—the sympathetic reducing peristalsis and digestion and the parasympathetic increasing peristalsis and function of digestive juices. There is also the autonomous intramural nervous control of Meissner's and Auerbach's plexuses.

The sympathetic innervation of the gastro-intestinal tract derives from the intrathoracic sympathetic chain and ganglia, receiving fibres from the 5th thoracic to the 3rd lumbar spinal segments and transmitting fibres via the splanchnic nerves to the coeliac and mesenteric ganglia and plexuses, from which post-ganglionic fibres travel with the blood vessels to the gut.

The parasympathetic supply to the gut is by the 10th cranial nerve, the vagus, whose fibres also pass through the coeliac plexuses to reach the small and large intestines.

In an injury of the upper thoracic and cervical levels of the cord, there should be a paralysis of the sympathetic system resulting in a lowered ability to reduce peristalsis, while the parasympathetic system should continue to function to stimulate peristalsis; without the antagonistic action of the sympathetic, peristalsis should be enhanced in upper thoracic and cervical injuries. Most of these injuries do not develop paralysis of the peristalsis, they do not vomit, they absorb oral fluids well and soon take food after 2 or 3 days. But a certain proportion of them, probably one-quarter to one-third, develop distension of the abdomen, lose bowel sounds and vomit. In cervical injuries this can be very serious and life-threatening, especially if acute dilatation of the stomach should develop, with subsequent interference with diaphragmatic movement and reduction of respiratory capacity. Treatment with naso-gastric tube and suction, with i.v. fluids and rectal tube usually resolves the clinical situation within 3–4 days, although some authorities advocate neostigmine to stimulate peristalsis.

Why do some patients develop ileus? Their parasympathetic systems are not affected by the level of their injury and peristalsis should be enhanced if changed at all.

One possible explanation is that the abrupt loss of sympathetic innervation in an acute injury upsets the usual delicate balance between the two antagonistic parts of the autonomic system, the paralysis of the one causing inhibition of the other, bearing in mind that both parts act through the same plexuses in the abdomen.

However, the two cases I am going to report present a different clinical picture as far as the date of onset and intractability of the complication were concerned and there would appear to be a different mechanism of action in these cases.

Case 1 was a young girl of 16 years who sustained a quadriplegia below C7 with sensory sparing only, due to a burst fracture of C.V.7 in a diving accident. She was treated with skull traction and transferred to Sheffield 3 days later in good general condition. She remained well for 5 weeks and then suddenly developed fever, rigors, vomiting and distension of the abdomen. Clinical examination did not disclose any focus for this feverish upset but the white cell count showed a polymorph leucocytosis of 13,700 per cub. mm. It could have been the onset of a urinary infection and she was treated with ampicillin. Her vomiting continued, bowel sounds disappeared and she developed acute dilatation of the stomach. She was treated with naso-gastric tube and suction and i.v. fluids and rapidly improved as far as her general state of health was concerned but the abdomen remained silent. This clinical picture continued for over 1 week. She remained generally well, gastric suction produced bile-stained fluids and i.v. fluids were necessary. X-ray of the abdomen demonstrated air in the stomach only. Distigmine (Ubretid), was tried with no effect on the 9th day. Neostigmine was tried with some effect on the 10th day as it produced some small bowel sounds but the stomach did not function and suction had to be continued. By the 14th day no real food had been taken and i.v. feeding with a full diet of Protein CHO and fat was given to a calorific value of 2500 cals daily.

Metoclopramide (Maxolon) was given at this time and almost immediately there was a return of bowel sounds and all oral fluids were absorbed. She made rapid progress thereafter and the metoclopramide was discontinued after 2 weeks. Diarrhoea was a problem then for the next 3 weeks until controlled with drugs, the most useful being Codeine Phosphate. From the 12th week onwards her gastro-intestinal tract was satisfactory.

The unusual feature of this first case was the late onset of ileus 5 weeks after injury and the longer duration lasting over 2 weeks, so that parenteral feeding had to be started.

Case 2 was a soldier of 26 years with a complete D6 paraplegia as a result of a bullet wound in the right chest. He sustained serious chest injuries, requiring a thoracotomy, massive blood transfusion and postoperative assisted ventilation through a tracheostomy. He had a stormy convalescence and came under our care in Sheffield 10 weeks after injury. He had to be treated in bed for another 12 weeks for repair of a sacral sore and also developed myositis ossificans at the right hip.

Twenty-six weeks after injury he became feverish, with rigors and tachycardia. There was no obvious focus of infection, his W.B.C. was 16,000 with a polymorph leucocytosis and he was treated with antibiotics. Two days later he began to vomit, sweated profusely, bowel sounds were reduced and his urine output fell. He was treated with i.v. fluids, Stemetil and a naso-gastric tube with suction of bile-stained fluid and recovered in 4 days, when he suddenly felt better. His tachycardia reverted to normal and he had a large diuresis. He began to eat at once. Three days later he became ill again with fever, sweating, clammy skin, tachycardia and vomiting. He became agitated and frightened. Treatment with gastric suction and i.v. fluids was restarted and he again recovered well in 3 days. An I.V.P. at this time showed normal kidney function.

Two days later a third episode occurred, similar to the previous two, perhaps a little milder and resolving in 3 days. Five days later a fourth episode of ileus occurred and a barium meal X-ray at that time showed complete atony of the stomach and first

part of the duodenum with a functional obstruction at the level of the third part of the duodenum, with no barium passing that level. There was a total lack of peristalsis, even after the administration of metoclopramide (Maxolon). At this stage parenteral feeding was required as well as i.v. fluids.

There was a gap of 9 days before episode five occurred with the same clinical picture. Again there was tachycardia with clammy sweating and vomiting and a polymorph leucocytosis of 15,000. He needed blood transfusion at this time and the episode lasted three days as before. Five days later the sixth and last episode started with vomiting, tachycardia, sweating, oliguria. He was treated this time with a Beta blocking drug, Ox prenolol (Trasicor) and the episode was less severe than the previous ones with resolution within 2 days.

A barium meal performed 2 days after resolution showed a normal gastrointestinal tract with rapid peristalsis. The dosage of Ox prenolol was increased and thereafter he ate normally and felt well. His prognosis was very satisfactory, he was mobilised into his wheelchair and was able to go home a month later.

The Ox prenolol was continued for 6 months in reduced dosage but there has never been any recurrence of the ileus.

The ileus in this case was different from that in Case 1 in that it was delayed till the 26th week after injury, it was composed of six separate distinct attacks, each lasting 2 to 4 days with normal intestinal function for 3 to 9 days between attacks. The attacks were dramatic, sudden in onset and he felt very ill at the time—he stated that he felt he was going to die. Tachycardia and clammy sweating were prominent features of each episode, as well as the vomiting and resolution usually occurred rapidly.

The cause of this dysfunction could only be an upset in the normal antagonistic interaction between the sympathetic and parasympathetic parts of the autonomic nervous system. At first I suspected that the bullet wound and subsequent thoracotomy had damaged the vagus nerves in the chest, thereby allowing the sympathetic system to act unchallenged. But the clinical picture, especially the tachycardia, sweating and clamminess pointed to sympathetic overactivity and this was confirmed when the problem was resolved by the exhibition of Beta-blocking drugs.

In summary then, these two cases of ileus developing as late complications of upper dorsal and cervical spinal cord injury proved to be somewhat intractible to treatment, compared with the commoner type of ileus occurring in the first days after injury and which resolves quickly with gastric suction. The cause in the late cases would appear to be intense sympathetic over-activity as the ileus was accompanied by severe vaso-motor disturbances. The actual precipitating factor in triggering off this autonomic imbalance is unknown. Both cases started their illnesses with fever, rigors and a polymorph leucocytosis and infection of the urinary tract may have been responsible.

Summary

Two cases are reported in which ileus developed as late complications at 5 and 6 weeks after upper dorsal and cervical spinal injury. The clinical picture differed from that commonly associated with ileus in the first week of injury when resolution occurs rapidly with gastric suction. Both cases proved intractible to treatment and gave cause for alarm. It was considered that sympathetic overactivity was responsible for the ileus as it was accompanied by severe vasomotor disturbances but the precipitating factor in triggering off the autonomic imbalance was not certain, although infection may have played a part.

Résumé

On présente deux cas de paralytique ileus qui se manifestait comme une complication tardié, 5 et 26 semaines après une trauma médullaire thoracol et cervicol. Le développement

clinique se distingue de l'ileus post traumatique qui résait rapidement avec aspiration stomecale. Les deux cas ici, se provent trés résistantes au traitement conservative. Une hyperactivité orthosympathique est probablement le cause de l'ileus car au même temps il y avait des symptomes vasomotorique. Le cause immediate qui précipites le reaction autonomique n'est pas établis mais une infection peut-être jouait un rôle.

Zusammenfassung

Bericht über zwei Falle von paralytischem Ileus, der 5 bzw. 26 Wochen nach hoher dorsalen und zervikalen Rückenmarksverletzung auftrat. Das klinische Bild unterschied sich von dem, das man gewöhnlich im Zusammenhang mit Ileus in der ersten Woche nach Unfall seiht, wenn Magenaspiration schnell zur Losung des Problems führt. Beide Falle sprachen nicht auf Behandlung an und die Prognose schien sehr ungünstig.

Die Erklärung liegt wahrscheinlich in erhöhter Aktivitat des sympathetischen Nervensystems, da der Ileus von schweren vasomotorischen Störungen beleitet war. Die unmittelbare Ursache, die die autonomische Gleichgewichtsstörung auslöste, bleibt unsicher; Infektion könnte eine Rolle gespielt haben.