ACUTE APPENDICITIS ASSOCIATED WITH INTRAPERITONEAL HAEMORRHAGE FROM RUPTURE OF THE CORPUS LUTEUM

BY

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More than 300 cases of intraperitoneal bleeding from rupture of the Graafian follicle or the corpus luteum have now been recorded, most of them within recent years. In a review of the literature, however, I have been able to trace only one case in association with acute appendicitis—that of Hoyt and Meigs (1936). Accordingly, the following case may be considered worthy of record.

Case Report

A housewife aged 31 was sent to hospital on October 3, 1940, as a case of acute appendicitis. Two days before admission she complained of the sudden onset of umbilical pain, which soon became localized to the region of the right iliac fossa. She vomited several times. The bowels were opened daily and there was no disturbance of micturition. The menstrual cycle was a regular one of 28 days, with a catamenia of 4 or 5 days. The last menstrual period had occurred twenty-six days before admission and had been normal. There was no leucorrhoea. She had never been pregnant, and had never had any previous attack of a similar nature.

On examination the patient was seen to be a well-nourished woman. Her temperature was 99.4° and pulse rate 100. The tongue was covered with a dense fur, and the breath was heavy. Tenderness was present in both lower quadrants of the abdomen, but was very pronounced in the right iliac fossa, where there was appreciable muscular rigidity. Blumberg's sign was positive. Vaginal examination was difficult because of extreme tenderness in the right fornix and pouch of Douglas, but no gross abnormality was detected.

A diagnosis of acute appendicitis was made, and under ether anaesthesia abdominal section was performed through a right lateral rectus incision. On entering the peritoneal cavity it came as a great surprise to discover some free blood. Further investigation revealed that there was a small pelvic haematocele and that the source of the bleeding was a slight but persistent ooze from rupture of a corpus luteum in the right ovary. The appendix, somewhat injected and oedematous, was seen to occupy a pelvic position lying close up to but not touching the right ovary. One mattress suture sufficed to arrest the oozing from the corpus luteum. The pelvic haematocele was then evacuated and the appendix removed. No further pelvic pathology was noted. The abdomen was closed without drainage.

The post-operative course was uneventful until the eleventh day, when signs and symptoms of a small pulmonary embolus developed. These soon cleared up, and the patient was discharged from hospital on the twenty-third day. The pathological report on the appendix was "catarrhal appendicitis."

Commentary

It is worth remembering that in young women acute abdominal symptoms may be caused by bleeding from rupture of the Graafian follicle about the mid-menstrual phase or from rupture of the corpus luteum later in the menstrual cycle. In the former the haemorrhage is usually slight, while in the latter it may be profuse. That it is possible to make a correct pre-operative diagnosis may be seen from the fact that of a series of 58 cases collected by Hoyt and Meigs at the Massachusetts General Hospital the diagnosis was proved to be correct at operation in no fewer than 17. Clinically, three types can be recognized, depending on the amount of haemorrhage: (1) massive cases in which the clinical picture is dominated by shock,

resembling the acute fulminating intraperitoneal rupture of a tubal pregnancy; (2) discrete bleeding, with signs of peritoneal irritation; and (3) slow bleeding, with the formation of pelvic haematocele.

The only constant and characteristic feature is the sudden onset of abdominal pain—usually in the right lower quadrant, since analysis of the reported cases shows that the lesion is commoner on the right side. Thus, in the diagnosis, acute appendicitis, rupture of a tubal pregnancy, and torsion of an ovarian cyst are the main conditions to differentiate.

There has been much speculation as to the aetiology of the condition. Wilfred Shaw (1927) remarked on the effect of trauma in determining bleeding from rupture of an ovary that is the site of congestion, from chronic pelvic disease, or from a normal ovary showing pre-menstrual congestion. It is remarkable, however, that in the recorded cases trauma has rarely been a factor. In some instances the act of coitus seems to have been the cause. Bimanual examination has also been cited as the precipitating factor, and it is interesting to note that Forssner believed that such bleeding almost always resulted from rupture of an ovarian pregnancy. In the case described above it is possible that some abnormal hyperaemia of the ovary due to the proximity of an acutely inflamed appendix was responsible for the rupture and subsequent bleeding from the corpus luteum.

I am indebted to Dr. J. Clark, medical superintendent of the City General Hospital, Sheffield, for his encouragement and permission to publish the details of this case.

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Medical Memoranda

Tetanus treated with Sulphapyridine

There is some evidence to suggest that sulphapyridine is of value in the treatment of anaerobic infections (Singer, 1940; M.R.C. War Memorandum, No. 2, 1940). Bryant and Fairman (1940) used it in a series of cases of tetanus, with results which they considered encouraging, but evidence that it is of definite value is still inconclusive, and further observations are needed. The following description of a severe case in which sulphapyridine was given in addition to the routine treatment with antitoxin and sedatives is of interest:

CASE REPORT

The patient, a previously healthy girl aged 10, trod on a nail and sustained a slight penetrating wound of the plantar surface of the right big toe. The wound was healing satisfactorily. Seven days later she complained of pain in the left side of the chest, and after a few hours her throat was sore and her jaw so stiff that she could not open her mouth properly. Twenty-four hours later she was given 3,000 units of antitoxin by her doctor and sent to hospital.

On admission she also complained of abdominal pain, showed risus sardonicus, and could separate her teeth only a quarter of an inch. Her neck was stiff, and there was tonic rigidity of the muscles of the trunk, with arching of the back sufficient to allow the flat hand to be passed under the spine without difficulty when she was lying at full length. There was some tendency to reflex irritability, but no reflex spasms had occurred. The slight wound on the toe had almost healed and looked healthy. 200,000 I.U. of antitetanus serum was given intravenously and a further 10,000 units injected into the tissues round the base of the injured toe. There appeared to be nothing in the wound to treat. Sulphapyridine was also given, and she received 7 grammes in the first twenty-four hours and 6 grammes in the

second twenty-four hours. Her condition, however, became worse, and reflex spasms began to occur six hours after admission (thirty hours after the onset of trismus). These were satisfactorily controlled with rectal paraldehyde in doses of 4 drachms in normal saline, and this treatment was repeated when the reflex spasms tended to recur. In spite of the fact that the reflex spasms were comparatively easily controlled her condition steadily deteriorated, her pulse rate and temperature rose, and she died eighty hours after the onset of trismus. She never had a really severe reflex spasm, and death seemed to be due to cardiac failure.

Post-mortem examination showed slight congestion of the brain, lungs, and other organs, but no changes were found to suggest that the cause of death was anything but uncomplicated tetanus. The scar of the old wound was explored but no pus was found.

COMMENTARY

In this case an incubation period of seven days suggested a severe attack. On the other hand, the age of the patient, the slight wound on the foot, and the fact that there was only tonic rigidity without reflex spasms on admission twenty-four hours after the onset were all favourable signs. When, however, reflex spasms began to come on six hours later the prognosis became grave. It has been shown previously that if reflex spasms occur regularly within forty-eight hours of the onset of trismus the chances of recovery are slight and that there is a direct relation between the period of onset (the time between the first onset of trismus and the onset of reflex spasms) and the duration of life. Observations on the 16 fatal cases in a previous series treated only with intravenous antitoxin and sedatives indicated that a period of onset of thirty hours would give a duration of life of between three and five days from the first onset of trismus (Cole, 1940). In the above case death took place eighty hours from the onset. The chances of recovery, however, seemed sufficiently borderline to suggest that an additional effective therapeutic measure might tip the scales in favour of recovery. The fact that sulphapyridine not only failed to do this but did not seem to have any effect whatever on the course of the disease or the duration of life suggests that it is not of much value in tetanus.

It is unwise to draw conclusions from a single case. Further observations are needed, but a small number of cases carefully observed on the above lines would give reliable information. It is important to remember that lobar pneumonia is an occasional complication of tetanus and that sulphapyridine would then be of great value. This case also illustrated the fact that, with present treatment, once a lethal dose of toxin has been absorbed death will take place at the appointed hour, whether or not the reflex spasms are controlled, probably from the effect of toxin on the medullary centres.

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Hemiplegia as a Complication of Diphtheria

The following case records the sudden onset of a complete hemiplegia in a child suffering from nasopharyngeal diphtheria, with subsequent recovery.

CASE HISTORY

A girl aged 9, with no previous illnesses of any significance, was admitted to Kendray Isolation Hospital on April 21, 1941, with thick membrane extending from the swollen tonsils on to the soft palate and posterior pharyngeal wall, membrane inside the excoriated nares, sero-sanguineous rhinorrhoea, typical bull-neck, and the usual features of a third-day case of severe nasopharyngeal diphtheria. The heart sounds, though poor, were closed, and the temperature remained below 99° throughout the illness. She received 80,000 units of antitoxin intravenously, cardiac stimulants, and intravenous drips, and, apart from transient palatal paresis from the seventh to the eleventh day in

hospital and gallop rhythm from the eleventh to the fourteenth day, progress was uneventful for seventeen days.

The eighteenth day witnessed the sudden onset of a flaccid paralysis which affected the right side of the face and right limbs, and which was accompanied by double incontinence. There was no coma, but the child was dazed and very weak. Auscultation revealed a loud, blowing systolic murmur, maximal at the pulmonary area. Detailed clinical examination, deferred for thirty-six hours because the girl was so desperately ill, showed that the paralysis was becoming spastic: brisk ankle-jerks, biceps-jerks, and triceps-jerks, ankle clonus, and Babinski's sign; motor function and fine sensation absent, but gross sensation present in the hand; no athetosis; no aphasia, although the child was right-handed. Both knee-jerks were absent, presumably indicating diphtheritic neuritis.

Treatment consisted in nursing care, strapping the side of the face, the provision, later, of a cock-up splint and an adjustable Thomas splint with a foot-rest to combat wrist-drop and drop-foot, and, finally, exercises. Sensation returned speedily; organic reflexes were restored in nine days; the motor nerves recovered slowly, the leg movements, which did not begin until the nineteenth day, being the last to return. The pulmonary murmur soon disappeared. During the period of gradual restoration of function, which was complicated by the recurrence of palatal paresis and by right internal strabismus (both transient), radiography and electrocardiography revealed no cardiac abnormality. After 125 days in hospital the patient was discharged, with all movements present, though weaker than on the unaffected side, and the heart normal to clinical examination.

DISCUSSION

Hemiplegia is usually a sequel to cardiac disease; therefore the pulmonary murmur must be considered first. As this was absent before and after the illness, and as the subsequent normality of the heart was confirmed by radiograph and electrocardiogram, it is necessary to exclude all organic causes, congenital and acquired. It might perhaps be suggested that an acute simple endocarditis, such as occurs after streptococcal infections, had developed at the pulmonary valve, and that subsequent absorption of vegetations had left the heart apparently normal. But to postulate an endocarditis which is completely apyrexial and which clears up in a matter of days carries us beyond the limits of human credulity. Clearly, despite its loudness, the murmur was merely one of those functional murmurs about which much has been written and little is really known. Possibly it was akin to the soft apical systolic bruits often audible in febrile illnesses, or, more probably, since in diphtheria the blood volume is reduced by the passage of plasma into the tissue spaces, it was akin to the haemic murmurs of anaemia. Anyway, it lacked pathological significance.

We must now find a connexion between diphtheritic toxæmia and a lesion of the internal capsule. Hemiplegia can develop after encephalitis (which disease has been described in association with diphtheria and other infections), but there is no evidence on which to base such a diagnosis. Again, it can occur as a complication of meningitis, but meningeal symptoms could hardly pass unnoticed in a hospital for infectious diseases. Moreover, encephalitic and meningitic hemiplegias are typically slow in onset.

Cerebral haemorrhage and tumour being excluded by the child's recovery, the diagnosis seems to lie between embolism and thrombosis, with absence of coma perhaps favouring thrombosis. Since the left side of the heart was not diseased, and since, even if the pulmonary valve had been diseased, an embolus from that valve would have had to traverse the entire pulmonary circulation before entering the systemic blood stream, the most feasible explanation of embolism would seem to be that a minute fragment of aspirated membrane in some way gained entrance to the lung capillaries and travelled onwards with the blood: not impossible, but, surely, most unlikely. The grossly enfeebled circulation and the increased viscosity of the blood as a result of the extrusion of plasma to the tissue spaces make cerebral thrombosis a much more probable diagnosis.

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