A CASE OF LYMPHADENOMA INVOLVING THE STOMACH IN A CHILD AGED EIGHTEEN MONTHS COMPLICATED BY RICKETS AND CLOSELY SIMULATING LEUKÆMIA.

BY H. D. ROLLESTON, M.D. CANTAB., F.R.C.P. LOND.,
SENIOR ASSISTANT PHYSICIAN TO ST. GEORGE'S HOSPITAL AND TO THE
VICTORIA HOSPITAL FOR CHILDREN;

AND

A. C. LATHAM, M.B. OXON., M.R.C.P. LOND.,
CURATOR OF THE MUSEUM, ST. GEORGE'S HOSPITAL; ASSISTANT
PHYSICIAN TO THE VICTORIA HOSPITAL FOR CHILDREN;
LATE RADCLIFFE TRAVELLING FELLOW IN
THE UNIVERSITY OF OXFORD.

THE patient, a male infant, aged one and a half years, was admitted to St. George's Hospital on Feb. 14th, 1898, and died two days later. He was under the care of Dr. Cavafy, who has kindly allowed us to make use of the case. Two months previously the child had been operated upon for a (granulation?) polypus in the right ear and since then had been losing strength and colour. The mother said that the child had brought up blood. A week before admission spots were noticed on the skin which at the post-mortem examination were seen as scattered petechiæ. There was a tender swelling behind the right parotid, and the spleen was found to be much enlarged, but no abnormal signs were discovered in the lungs. The child was very anæmic, but did not complain or appear to suffer any pain except when the right side of his neck was examined, and died quietly from increasing asthenia two days after coming into the hospital.

Owing to the fact that we did not see the patient during life we were unable to make a complete examination of the blood—for instance, we have no data with regard to the absolute number of white and red cells. We were fortunate, however, in having several blood films prepared by Mr. S. Smith, the house physician of the case. An examination of these films showed that there was very considerable degeneration of the red cells and that this was accompanied by a large increase in the number of white cells. A differential count of 500 white cells gave the following figures: lymphocytes, 61.1 per cent.; polynuclear cells, 16.5 per cent.; eosinophile cells, 1.6 per cent.; and myelocytes, 20.8 per cent. During this count 94 normoblasts and 6 megalo-blasts were noticed; many of these showed mitotic changes. There were no Charcot-Leyden crystals. The blood thus showed very marked anæmia, together with a marked increase in the number of lymphocytes—many of which were of the larger form—and a very large percentage of cells foreign to normal blood—namely, myelocytes.

At the necropsy the body was found to be markedly anæmic with scattered but not very numerous petechiæ. There was slight œdema of the legs. There were marked rickety curves of the tibiæ and enlargement of the wrists. There was a hard swelling in front of the sterno-mastoid muscle on the right side reaching up into the parotid region. On dissection this was found to be a packet of glands passing underneath the sterno-mastoid muscle. The glands appeared gelatinous from œdema and the fibrous tissue separating them seemed prominent and increased in amount. Microscopically, the packet of glands on the right side of the neck showed thickening and increase of their fibrous capsules; the normal arrangement of the glands was obscured and lost. There was a general slight increase of the fibrous tissue throughout the tissue of the gland with a number of oval connective tissue cells. The number of small lymphocytes was diminished but large lymphocytes were present in considerable quantities. These cells appeared to be the same as those described by many writers as endothelioid cells. The other accessible lymphatic glands in the body were examined but were not found to be enlarged. The anterior fontanelle was open, but not widely; the diploe and bones of the skull-cap were healthy. The membranes and sinuses were normal. The brain weighed 36 oz. and was healthy. Both membranæ tympana were perforated and the middle ears contained soft granulation tissue polypi and muco-pus. The sternum appeared to be normal on

section. The ribs showed very marked rickety enlargement at their costo-chondral junctions, there being in several instances some backward displacement of the ribs on the costal cartilage. At a distance of from 1½ in. to 2 in. from the costo-chondral junction several of the ribs showed periosteal nodes which on section invaded the medullary cavity and rather suggested a united fracture. But as the node was only on one side, the fracture, if there had been one, must have been a "greenstick." The rickety enlargement was very vascular and was in places excavated into loculi; reddish marrow extended some distance into the shafts of the ribs, in some as far as the periosteal nodes already referred to. There was no hæmorrhage under the periosteum or sign of scurvy rickets. Microscopically, besides marked evidence of rickets, the costo-chondral junction showed an excess of lymphocytic infiltration as compared with normal bones from a child of the same age. This lymphocytic infiltration was also present in the shafts of the ribs: this condition might be thought to resemble, but only in a very minor degree, the lymphadenomatosis of bone described by Weber.¹ The nodes on the ribs were composed of ossifying cartilage; under the node there was a transverse break on the underlying lamina of bone strongly suggesting that a partial fracture had occurred some time before death. The bones of the limbs were not examined. The thymus and thyroid glands were healthy. The pleuræ were normal. The lungs showed early broncho-pneumonia with small hæmorrhages into their substance. The bronchial and tracheal glands were healthy. There were petechiæ on the visceral pericardium. The cardiac valves were healthy. The myocardium was pale but there was no manifest fatty degeneration. The abdomen was distended from the presence of flatus in the intestines; but there was no ascites. The œsophagus was perfectly normal. The outside of the stomach near the lesser curvature showed a few plaques of white growth. The stomach contained some grumous fluid. Its mucous membrane presented a remarkable appearance (vide Fig. 1) from the presence of a number

FIG. 1.

Lymphadenomatous growths arising from the mucous membrane of the stomach. The only part not affected is the region around the pylorus.

of polypoid growths that were scattered all over the lining mucous membrane except for a distance of from 1 in. to 1½ in. around the pylorus. The growths were smooth, white, and not ulcerated. These were of varying size, the largest being equivalent to a small walnut. They were largest and most

¹ F. P. Weber: General Lymphadenomatosis of Bone, one form of Multiple Myeloma. *Journal of Pathology and Bacteriology*, January, 1898.

closely packed near the cardiac orifice, gradually diminishing as the pyloric region was approached. Microscopically the polypoid growths were composed of the mucous membrane thrown into folds and extensively infiltrated with large and small lymphocytes (vide Fig. 2). The submucous coat did not

FIG. 2.

Microphotograph of lymphadenoma of the stomach $\times 12$ showing infiltration limited to the gastric mucosa and not invading submucosa.

contain any lymphocytes at all. This growth, which also contained some spindle-shaped connective tissue, was apparently derived by proliferation of the lymphoid tissue that is normally found on the mucosa. The tubular glands of the stomach were somewhat displaced by the growth but showed no proliferation or degeneration and were well preserved. The duodenum showed some vascular streaks from which the stomach was quite free. In the second and third parts of the duodenum there were some small white nodules on the mucous membrane. Close to the entrance of the ileum into the cæcum there were a few enlarged and superficially eroded Peyer's patches, but the rest of the small intestine was free. The vermiform appendix appeared to be natural and except for a single sessile growth in the rectum the large intestine was free from growth. There was no infiltration of the peritoneal coat of the intestines and the mesenteric glands were not larger than they normally are in a child of this age. The aortic lymphatic glands were normal, but there were some pigmented glands along the iliac arteries and accompanying the splenic artery at its entrance to the hilum of the spleen. The spleen weighed 8 oz. and was much enlarged; it rested on an extremely well-developed costo-colic ligament and the adjacent splenic flexure of the colon was collapsed as if from pressure. There was no capsulitis and on section the spleen appeared to be tough, reddish, and uniform. There were no manifest pigmentation, no enlargement of the Malpighian bodies, and no lardaceous reaction. There were no infarcts or hæmorrhages. Microscopically the pulp was inundated with large and small lymphocytes; the Malpighian bodies were small but had not undergone any fibrotic change. There was some pigmentation around the walls of the sinuses. There was no fibrosis of the spleen. The suprarenals and the pancreas were normal. The kidneys (each weighed 1 oz.) were pale but otherwise normal. Microscopically there were a few small foci of small and large lymphocytes, small lymphadenomatous growths, and some blocking of the capillaries with lymphocytes. The tubal epithelium showed a little proliferation. The bladder was normal. The testes were large but healthy. The liver was enlarged to a slight extent and on section appeared to be swollen and pale. There was a large white gland in the portal fissure, but there were no growths on the substance of the organ. The bile-ducts were pervious. The gall-bladder was almost empty. Microscopically there was a general excess of lymphocytes in the capillaries, so that the liver appeared to be in a state of early leukæmia. In addition there was in the portal spaces and outside the capillary vessels early lymphadenomatous growth composed of lymphocytes and young connective tissue-cells. There was no cirrhosis or fatty change.

Remarks.—Viewing the case in the first place from the standpoint of the blood examination alone the case would plainly have to be described as one of lieno-myelogenous leukæmia. The small percentage of eosinophile cells is unusual, but the majority of observers are agreed that the part played by eosinophile cells in leukæmia is unimportant. Some of the blood changes are undoubtedly due to the fact that the child was suffering from marked rickets. In this disease there is considerable anæmia, which is often accompanied by a lymphocytosis and a small (from 2 to 3 or less) percentage of myelocytes. In this patient, however, the large percentage of myelocytes was far in excess of anything that has been found in any other disease than leukæmia. Again, a part of the lymphocytosis and the small number of polynuclear cells might readily be accounted for by the age of the child, so that it is fair to regard the excess of small lymphocytes as due to some other cause than leukæmia. In fact, our knowledge of the changes which occur in the blood in children is not sufficiently accurate for us to form any definite conclusions on a blood examination alone in such a case as this. It might be suggested that the case should be classified under what von Jaksch² and others have called "anæmia infantum pseudoleukæmica." It is very doubtful, however, whether this name can be fairly given to any class of disease. The vast majority of cases recorded under this heading have not been examined in a sufficiently thorough manner in the matter of a differential count of the white cells for us to say under what classification they should be put. Further, very different types are given by different observers. We agree with von Limbeck³ that no such disease exists and we believe that Cabot⁴ is right in suggesting that the cases which have been recorded as cases of "anæmia infantum pseudoleukæmica" are in reality either cases of pernicious anæmia, secondary anæmia without leucocytosis, Hodgkin's disease, lymphatic leukæmia, or possibly cases of spleno-myelogenous leukæmia. It is further possible, as Cabot suggests, that there may be cases which are intermediate between leukæmia and pernicious anæmia. Turning to the point of view of the morbid anatomy the case appears to be a remarkable example of lymphadenoma. The growth, as is so often the case, apparently began in the cervical glands, and it is probable that the poison, whatever it may be, which gives rise to lymphadenoma gained entrance to the lymphatic system through the abraded surface in the right tympanum. Starting here, the growth had become general and, a noteworthy feature in the case, chiefly in the alimentary canal and especially in the stomach. The spleen was much enlarged and there was some infiltration of the ribs. These latter lesions, as will be seen, are of great interest. Now though lymphadenoma, unlike leukæmia, is far from rare in young children, it appears that lymphadenomatous growths from the gastric mucous membrane have not been previously described at so young an age. The youngest case which Pitt⁵ recorded was twenty-four years old and the average of his 5 cases was 44.8 years. The morbid appearances in our case closely resemble those in Pitt's own patient, who was forty-eight years old. In both the mucous membrane of the stomach around the pylorus, in the antrum pylori, was free from lymphadenomatous growths. This is very striking, especially in Pitt's case, when it is considered in the light of the anatomical distribution of lymphoid tissue in the mucous membrane of the stomach. From a number of observations Soltau Fenwick⁶ concluded that in "infancy and childhood these collections of lymphoid tissue are fairly developed and profusely scattered over the whole surface of the organ, but are always particularly large and distinct in the pyloric region near the lesser curvature. After the age of forty it is often impossible to demonstrate their presence in the cardiac two-thirds of the stomach without the use of the microscope, and in the pyloric region they are much reduced in numbers and appear small and shrunken." The growths have therefore in both cases avoided the parts where the lymphoid follicles are normally best developed, and in Pitt's case were present where the lymphoid tissue is usually atrophied at that age. It is plain, then, that although the disease appears from the morbid anatomy

² Von Jaksch: Wiener Klinische Wochenschrift, 1889, Nos. 22 and 23.

³ Von Limbeck: Grundriss einer Klinischen Pathologie des Blutes, p. 368.

⁴ Cabot: Clinical Examination of the Blood.

⁵ Pitt: Transactions of the Pathological Society of London, vol. xl, p. 80.

⁶ Soltau Fenwick: Journal of Pathology and Bacteriology, vol. i, p. 424.

standpoint to be lymphadenoma, yet it presents some unusual features in such points as the distribution of the disease. So far we find that the blood examination points to a diagnosis of lieno-myelogenous leukæmia and that post-mortem evidence gives support to the diagnosis of lymphadenoma. If the disease were leukæmia throughout there ought to have been much more marked leukæmic infiltration of the liver and the kidneys than was present, for it is probable that if the large polypoid growths in the stomach were due to lymphocytic infiltration occurring in lymphatic leukæmia the other organs should be affected to a corresponding degree. On the other hand, the examination of the sections of the liver and kidney showed that the capillaries were in places filled with lymphocytes, thus supporting the diagnosis of early leukæmia. Against leukæmia, however, we have to face the fact that only two cases of genuine leukæmia in infancy have been recorded,⁷ and that of these only one was of the lieno-myelogenous type. In this case there was a percentage of 21 myelocytes and this was associated with a large increase in the number of white cells and an abundance of nucleated red cells. It is possible to regard the case as primarily one of Hodgkin's disease which in its later stages had taken on the characteristics of leukæmia. There is no doubt that cases which clinically are primarily cases of Hodgkin's disease do before death develop the blood characteristics of leukæmia. It may be that towards the end of a case of lymphadenoma we get in some instances what may be called a lymphadenoma of the blood—that is to say, the process in the glands becomes acute and there is secondary invasion into the blood, getting in this way the appearance of lymphatic leukæmia, just as in sarcoma a secondary invasion occurs by means of the blood. This secondary invasion in sarcoma is, it may be noted in passing, often associated with what appears to be a lymphocytosis. This occurrence thus becomes very interesting, not only from the point of view of the relationship between lymphadenoma and leukæmia, in which the lymphatic glands and the leucocytes are respectively affected, but as bearing on the relationship of lymphadenoma to sarcomatous growth. The close connexion of these diseases is made more evident by the result of Muir's⁸ careful study of a series of leukæmic cases; he comes to the conclusion that the changes in both forms of leukæmia are due to the proliferation of a certain cell—possibly owing to the action of some irritant—in a manner analogous to what is found in certain tumours, such as sarcomata. In this particular case the morbid appearances suggest Hodgkin's disease, the blood examination points to a mixed leukæmia, and in view of the above it might be held that the case was primarily Hodgkin's disease and secondarily had taken on the blood characteristics of lieno-myelogenous leukæmia. Against this, however, we know in the first place that strange variations occur in the blood of children without any very apparent cause, and further although the blood examination is in itself very much in favour of lieno-myelogenous leukæmia, yet considering the age of the child it does not justify the conclusion that the case was leukæmia. This conclusion is very much strengthened by the fact that there appears to be no record of any case of lymphadenoma which went on to lieno-myelogenous leukæmia, although cases of Hodgkin's disease indistinguishable in the late stages from cases of lymphatic leukæmia are common. It would seem best to regard the case as one of aberrant lymphadenoma in which changes simulating lieno-myelogenous leukæmia had been induced by a marked condition of rickets. As has already been pointed out, the large amount of lymphocytosis and the marked anæmia with the presence of nucleated red cells is what is found in rickets. Further, in rickets there is apparently no increase in the number of eosinophile cells and this would be in keeping with the small number—practically normal—of eosinophile cells present in this particular case. Again, the age of the child might account for the relatively small number of polynuclear cells present. So far then we have accounted for all the blood changes with the exception of the presence of the abnormal cells or myelocytes. A very small percentage of these cells is common in rickets. It is probable that the infiltration of the marrow of the ribs by lymphadenomatous material which occurred in this case may have acted in some way in conjunction with the marked rickety condition, either as an irritant or otherwise, so that it caused a more or less

intense proliferation of the cells which are found in bone marrow but are foreign to the blood—namely, the myelocytes. Viewed in this light it seems best from post-mortem evidence to regard the case as one of lymphadenoma complicated by rickets, especially as the blood changes can be satisfactorily explained by this combination of diseases.

For the drawing of the stomach and the micro-photograph we are indebted to Mr. P. L. Mummary, B.A., and Mr. Harold Spitta, senior students at St. George's Hospital.

A METHOD OF EXPOSING AND OPERATING ON THE KIDNEY WITHOUT DIVISION OF MUSCLES, VESSELS, OR NERVES.¹

By A. W. MAYO ROBSON, F.R.C.S. ENG.,
SENIOR SURGEON TO THE GENERAL INFIRMARY AT LEEDS AND PROFESSOR OF SURGERY IN THE YORKSHIRE COLLEGE.

My much lamented friend, the late Greig Smith, used to insist on the importance of opening the abdomen in the course of the muscular fibres as much as possible so as to avoid the weakness that is apt to follow on division of muscle, but I can find no evidence in his writings, nor did I gather in my many conversations with him, that he had conceived the idea of exploring the kidney on this principle. Dr. McBurney's method of exposing the vermiform appendix, which I have employed on numerous occasions with great satisfaction, suggested to my mind the possibility of exposing the kidney in a similar manner, and it was not until I had performed a number of operations by the method I am about to describe that my attention was drawn to a paper by Dr. Abbé in the Transactions of the New York Surgical Society, in which he describes a case of nephrectomy performed on the same principle. Seeing that the operation is one which from ample experience I have found of great utility in all operations on the kidney I have no hesitation in describing it and recommending it to the notice of other surgeons. The accompanying diagram, which was drawn for me by my friend

Diagram of incision, showing splitting and retraction of fibres of the external oblique muscle and also of internal oblique and transversalis.

Dr. F. Gairdner, who has assisted me in several of these operations, shows at a glance the principles on which the operation is based. The incision beginning to the inner side of the anterior superior spine of the ilium is carried backwards obliquely towards the tip of the last rib. The fibres of the external oblique and its aponeurosis are then split and retracted, exposing the internal oblique muscle, the muscular fasciculi of which are split in a line between the ninth costal cartilage and the posterior superior spine of the ilium, in which position they are longer than in front of or behind that line. When the fingers are pushed through the internal oblique to split it the fibres of the transversalis are pierced and can be retracted along with the oblique muscle. A

⁷ Morse: Boston Medical and Surgical Journal, 1894.
Muir: Journal of Pathology and Bacteriology, vol. i., p. 123.

¹ A paper read before the Leeds and West Riding Medico-Chirurgical Society on April 22nd, 1898.