Thrombosis of the cerebral sinuses extending into the choroid plexuses and cerebral veins, with clinical notes of a case of the disease / by G. A. Gibson and J. Purves Stewart.

THROMBOSIS OF THE CEREBRAL SINUSES EXTENDING INTO THE CHOROID PLEXUSES AND CEREBRAL VEINS, WITH CLINICAL NOTES OF A CASE OF THE DISEASE.

By G. A. GIBSON, M.D., D.Sc., F.R.C.P.Ed.,

AND

J. PURVES STEWART, M.A., M.B.;

Including the Pathological Investigation of the Case by J. Lorrain Smith, M.D., and D. A. Welsh, M.A., M.B., B.Sc.

Reprinted from Volume Third of the Edinburgh Hospital Reports.

Edinburgh and London, Young J. Pentland, 1895.

THROMBOSIS OF THE CEREBRAL SINUSES EXTENDING INTO THE CHOROID PLEXUSES AND CEREBRAL VEINS, WITH CLINICAL NOTES OF A CASE OF THE DISEASE.

Thrombosis of the Cerebral Sinuses extending into the Choroid Plexuses and Cerebral Veins, with Clinical Notes of a Case of the Disease.

By G. A. Gibson, M.D., D.Sc., F.R.C.P.Ed., and J. Purves Stewart, M.A., M.B.; including the Pathological Investigation of the Case by J. Lorrain Smith, M.D., and D. A. Welsh, M.A., M.B., B.Sc.

A very considerable number of cases of thrombosis of the cerebral sinuses is to be found in the medical literature of the last forty years, from a survey of which a classification, based on etiological considerations, has been generally adopted. It is easy to understand that, in consequence of their wide lumen and intricate arrangement, the cerebral sinuses are especially liable to become the seat of thrombotic processes, and the sluggish flow of the blood, as well as its tortuous course, must increase this tendency. The causes leading to the development of the affection appear to be threefold. Direct obstruction to the current of the blood, by a tumour, for instance, may, in the first place, produce such a degree of stagnation as to give rise to coagulation. Extension of inflammation from a neighbouring part, as the ear, may, in the second place, cause such changes in the sinus wall as to lead to thrombosis. And, in the third place, conditions of debility may, particularly in children, lead to the clotting of the blood.

A very striking example of sinus thrombosis has recently been under our care, and has brought before us in an especial manner the prominent clinical and pathological features of the disease. Without further prelude we shall proceed to the description of the case.

Clinical Report.

A housemaid, æt. 25, was admitted to Ward 25 of the Royal Infirmary at midnight on 15th April 1895, under the care of Dr. Gibson. The patient being unconscious, the following history was obtained from a fellow-servant who accompanied her.

The patient was a quiet, steady girl, not naturally excitable. She had suffered for a long time from indigestion, and occasionally also from feelings of lightness in the head. During the last fortnight she had complained of occipital headache.

Four days prior to admission the headache became more severe, and she began to have attacks of vomiting, apparently quite unconnected with the ingestion of food. These attacks recurred at frequent intervals, and she had last vomited at 5 A.M. on the morning of the day of admission. She became gradually more and more dazed and stupid, and about 6 P.M. on the evening of admission she was quite unconscious. She had been tossing her arms and legs about ever since. The bowels had moved on the night before admission, but had been confined for three days prior to that date.

The family history revealed nothing of interest beyond the fact that patient's father had died two years ago of "inflammation of the brain."

The patient, on admission, was seen to be a well-developed and well-nourished young woman. The temperature was 102° Fahr.; the pulse, 108; respirations, 40. She lay in a supine position, with flushed face, grinding her teeth occasionally, and moving all her limbs in a restless, aimless, uneasy fashion. She could not be roused by shouting or pinching. She resisted the placing of a thermometer in the axilla. Her eyes were almost closed, the pupils were semi-contracted, and reacted to light. Ophthalmoscopic examination showed both discs to be normal, there being no trace of optic neuritis.

Patient was put into bed, and 2 minims of croton oil were administered. After the usual routine sponging of the whole body, the temperature fell somewhat, to 99°·6 Fahr., but soon

rose again, until at 4 A.M. it stood at 102°·8 Fahr., with pulse 120 and respirations 36 per minute. A sample of urine, procured at 1 A.M. by the passage of a catheter, proved highly concentrated, acid in reaction, and with a copious deposit of urates. There was no albuminuria.

At 3 A.M. patient took her first "fit," which lasted some eight minutes. Another attack occurred at 5.5 A.M., and thenceforward others at frequent intervals, each fit lasting from four to eight minutes, with intervals of from five to fifteen minutes, until 9 A.M. The fits diminished in severity after 8 o'clock. Patient never regained consciousness between the fits.

During the fit (which was not preceded by any cry) both arms became stretched in front of and parallel to the trunk, extended at the elbows, strongly pronated, and quite rigid, with the thumbs turned inwards towards the palms. The legs were extended and rigid, the teeth were clenched, and the back was arched in the opisthotonic posture. There were no jerking movements. No urine was passed during the fit. The breathing during the fit was loud and stertorous, and the saliva was blown into a froth at the lips. The face was flushed, the pupils were contracted, and the eyes were directed downwards and slightly to the left side. The pulse became very frequent during the fit, 160 per minute. Blowing on the conjunctiva caused slight winking; whilst touching the cornea with the finger did not.

As these attacks resembled the attitudinising postures of hysteria to some extent, a strong faradic current was passed through the ovarian regions, but without exercising any effect either on the frequency or severity of the fits. The current was tried both during and between the fits.

At 9.45 A.M., the temperature, which at 3 A.M. was 104°, had now risen to 104°·4 in the left axilla. In the right axilla it stood at 104°. Saliva was now running very freely from the mouth, and there was marked epiphora of the left eye. Urine had been passed in bed. There was a slight blood stain on the sheet, due to the catamenia. The pulse was 93 per minute, regular in time and force and of moderate tension. The respirations, 24 per minute, were snoring in

character. The pupils were moderately dilated, equal, and insensitive to light.

At 10.20 A.M. the pulse rate suddenly rose to 198 per minute. The snoring respirations remained unaltered in rate, but at every inspiration there were two or three jerking sobs.

At 10.30 A.M. the respiration suddenly stopped, and the patient became very cyanosed. Prompt bleeding relieved this, and artificial respiration by Howard's method was at once started. This produced a temporary improvement, and the pulse, which was very frequent—too much so to count—steadied down somewhat.

At 11.10 the temperature in the groin was 103°·4. Artificial respiration was still kept up, as the patient made no effort to breathe. The pulse was very rapid and feeble. Ether, strophanthus, and atropine were injected. The faradic battery was applied at each artificial inspiratory movement, one pole being at the back of the neck, the other over the diaphragm.

At 12.45 P.M. patient was looking paler, and the temperature had fallen to 101°. At 1.30 P.M. the temperature was 99°·2, and frequent stimulation with ether and strophanthus was necessary to keep the heart going.

At 1.30 the patient was evidently moribund; the pulse was practically imperceptible, and she died at 1.40 P.M., three hours and ten minutes after natural respiration had ceased.

The diagnosis in this striking case was surrounded by difficulties. The only disease that seemed to us, as in the least degree a possible explanation of the symptoms, was tubercular meningitis, but when making the return for the pathological department we left the diagnosis an open question.

Pathological Report.

J. S., æt. 25 years; died 16th April; sectio, 17th April 1895.

Externally the body appeared well nourished. Cadaveric rigidity was present in all the limbs. There was a recent slight ecchymosis below each mamma.

Head.—The calvarium showed no abnormality. The dura

mater was somewhat thickened over the vertex, where it was markedly adherent to the skull and to the pia-arachnoid. On the under surface of the dura there were two stellate groups of bony spicules about $\frac{3}{4}$ in. in diameter, situate one on each side of the great longitudinal fissure—the one being over the upper end of the right ascending parietal convolution, the other being immediately behind the corresponding convolution on the left side. The inner surface of the dura mater was everywhere considerably injected, but most especially in the middle fossa in either side. The superior longitudinal sinus and each lateral sinus were occupied by a mixed blood clot, the greater part of which was dark red in colour, with a slight admixture of scattered whitish flakes, the whole being soft and only very slightly stringy. The bones at the base of the skull were apparently quite healthy.

The brain weighed 46 oz. The convolutions were markedly flattened, and were closely opposed along the lines of the sulci, which were partially obliterated. The surface of the brain was dry and sticky over the vertex and sides. There were one or two oz. of clear fluid in the posterior fossa at the base. The arteries at the base and the cortical arterial branches were free from thrombosis. There was no indication of any change in the appearance of the base of the brain.

On section, the grey matter appeared fairly healthy. In the white matter the *puncta cruenta* were very prominent. On exposing the lateral ventricles, the choroid plexus on each side was found to be occupied by a mixed thrombus. On the left the thrombus was of almost uniform thickness from the anterior horn to the foot of the descending horn, and nearly $\frac{1}{4}$ in. in diameter; on the right the thrombus was more tortuous and irregularly nodular, varying from a thin cord to nodules $\frac{1}{2}$ in. in thickness. The thrombus extended backwards along the veins of Galen and other vessels of the velum interpositum to the straight sinus, occupying all these vessels completely, (Plate 1). The choroid plexus of the fourth ventricle was deeply congested, but showed no obvious thrombosis. The thrombus in the choroid plexuses, veins of Galen, and straight sinus was paler in colour and firmer in consistence than that in either lateral sinus or in the superior longitudinal sinus.

The lateral ventricles were not dilated, and did not contain any free blood or other fluid.

The basal ganglia showed very extensive changes. On the left side the optic thalamus and caudate nucleus—on the right the optic thalamus alone—were completely replaced by a tissue of a dark brownish-red colour, and of exceedingly soft and friable consistence. The central parts of the ganglia affected were much darker in colour than their more superficial parts, as if hæmorrhage had occurred into their substance. The right caudate nucleus showed no naked-eye change. At the posterior part of each lenticular nucleus there was a slightly reddened triangular area; the condition was more marked in the left side. The internal capsule showed no obvious lesion on either side. No abnormality could be detected with the naked eye in the substance of the cerebellum, crura, pons, or medulla.

Thorax.—The pericardium was healthy. The right pleural sac was obliterated by fine fibrous adhesions over the whole anterior surface of the lung. The left pleura was healthy.

Heart.—Both arterial and auriculo-ventricular valves were healthy. The left ventricle contained some post-mortem clot. The left auricle was empty. The right auricle was filled with dark post-mortem clot, but its appendix contained a pale ante-mortem clot, which passed through the tricuspid orifice and occupied the right ventricle. The thrombosis did not extend into the pulmonary artery. The muscle of the left ventricle was firmly contracted, and the wall thick but apparently healthy. The right ventricle showed a zone of fatty infiltration towards the apex.

Lungs.—Both showed marked emphysema along their anterior margins; the pulmonary substance was congested and œdematous, especially posteriorly. The larger bronchia were filled with a frothy serous fluid; their mucous membranes were healthy. There was no thrombosis of the pulmonary vessels within the lung.

Abdomen.—The peritoneum was healthy. The inferior vena cava was occupied by dark fluid blood; the vena portæ contained a clot which was uniformly dark and soft. No evidence of ante-mortem thrombosis was anywhere seen.

Stomach.—The mucous membrane of the stomach was puckered around a small healed ulcer, about the middle of the lesser curvature high up on the posterior wall. The ulcer was circular in outline, and about ¼ in. in diameter. Its floor was smooth, and was composed of exceedingly thin fibrous tissue. There were no peritoneal adhesions around. A small branch of the coronary artery traversed the floor of the ulcer externally. In all other respects the stomach appeared healthy.

The intestines, liver, and spleen presented no abnormality.

Both kidneys were slightly congested, but otherwise healthy.

The uterus was small, nulliparous, and non-pregnant. There were small cysts with semi-solid, yellowish contents in each ovary.

Microscopic Examination.

The portions of tissue examined were fixed in corrosive sublimate solution, and cut in paraffin. They comprised pieces from each of the following structures—(1) The left optic thalamus and caudate nucleus; (2) The right choroid plexus; (3) The medulla.

1. The tissues of the left optic thalamus and caudate nucleus were the seat of very numerous, recent, capillary hæmorrhages; and so abundant was the total hæmorrhage in any one section, that the greater part of the field appeared to consist of red blood corpuscles, leucocytes, and some fibrin in place of the proper nervous structures. Some of the hæmorrhages were distinctly limited to the perivascular spaces, which then became distended with blood; the greater number, however, had ruptured into the adjacent tissues, but still remained discrete; while here and there a more diffuse extravasation had occurred, ploughing up a greater area. The hæmorrhages were all so recent that the red blood cells were practically unaltered. In the centre of some of the larger masses strands of fibrin had appeared. No altered blood pigment was anywhere visible. The smaller vessels

that had not ruptured were intensely congested and distended, and many of them showed an accumulation of leucocytes at the periphery and in the perivascular space. A medium-sized vessel in the substance of the left caudate nucleus was the seat of recent ante-mortem thrombosis, being completely plugged by a mass of leucocytes, fibrin, and red blood cells. The nervous elements were partly lacerated, partly compressed by the extravasated blood.

2. The right choroid plexus was so altered, partly by recent thrombosis of its vessels, partly by recent hæmorrhage into its substance, that it was almost impossible to identify its structure. Nearly every vessel appeared to be occupied by a thrombus, in which filaments of fibrin could be seen along with leucocytes and red corpuscles, the latter everywhere retaining their outlines, but at parts showing loss of staining power. Granules of golden-brown pigment, apparently altered hæmoglobin, were present in the mass of the thrombus, but more especially in the walls of the perivascular spaces. The rest of the structures were more or less completely obliterated by diffuse extravasation of blood, in which also fibrin and altered blood pigment were seen. The fringes with their endothelial covering were for the most part quite unrecognisable, only at rare intervals could they be distinguished.

3. Both in the substance and on the surface of the medulla the vessels were greatly enlarged with blood, but apparently free from thrombosis. In a cross-section through the middle of the medulla, one of the distended capillaries had ruptured and led to a small extravasation of blood. Although very limited in its extent, the hæmorrhage had occurred in the centre of a group of nerve cells occupying the position of the vagus nucleus on the right side, and was probably quite sufficient to account for the sudden respiratory failure which preceded the death of the patient.

There were one or two minute scattered patches of softening in the white matter of the medulla at this level also, otherwise no abnormality was detected.

Note.—Owing to the short time available for the microscopic examination, this report must be regarded only as preliminary

to a more complete investigation. But, so far as we have gone, we are enabled to state the following conclusions :—

1. There was no condition discoverable either in the bones of the skull or in the walls of the sinuses to account for the thrombosis, which must therefore be regarded as essentially primary in its origin.

2. The exact site at which the thrombosis commenced is uncertain. It was not possible definitely to decide whether it had begun in the choroid plexus and velum interpositum or in the larger sinuses beyond.

3. The acute hæmorrhagic infiltration of the basal ganglia affected was undoubtedly secondary to the thrombosis, more especially of the veins of Galen, and was due to the intense congestion of their venules and capillaries, produced by the stoppage of their venous return.

General Remarks.

In this most interesting case the profound unconsciousness pointed to some grave central disturbance, and the rigidity of the muscles, with the firm contraction of certain groups, gave evidence of irritation in some part of the motor tract. It was quite evident that this could not be in the cortical region, as there were no Jacksonian symptoms. The history of the illness and the absence of any optic neuritis negatived the possibility of any tumour, and, as above mentioned, the only disease which seemed to us as in the least degree likely to explain the symptoms was tubercular meningitis.

One of the most striking clinical features was the sudden and absolute failure of the respiration, and it is worthy of note that the post-mortem examination has given a complete explanation of this symptom, by revealing a hæmorrhage in the region of the medulla occupied by the nucleus of the vagus nerve. In this connection we may refer to the case published in this volume of the Reports by Dr. Gibson and Dr. Fleming, in which failure of the respiration was produced by lesions of the phrenic and intercostal nerves. These two cases afford a most interesting contrast in the development of the same clinical phenomenon.