

INTRACRANIAL ABSCESS

THE SHAPE OF THINGS TO COME ?

BY

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Antibiotics have decreased the severity and duration, as well as the morbidity and mortality rates, of many infections. However, where complications develop they appear to be more severe than in the pre-antibiotic era; patients who used to suffer mild complications now recover without any trouble, while many patients who would have died now survive, but with severe damage. As with other infections, abscess of the brain has become quite rare among those enjoying a good standard of living and able to attend early for medical treatment. However, I have been struck by the severe nature of the condition when seen in this class of people.

Present Series

This paper reviews all the 29 brain abscesses seen by me between 1952 and early 1962, and an attempt will be made to sustain the impression that where an abscess occurs in a person of low standard of living and health it follows the conventionally described pattern of encephalitic stage with brain-swelling, followed by solitary abscess formation; but in those with a high standard of life and health brain abscesses, while becoming less frequent, are much more severe when they do occur and tend to be multiple. Two cases were seen in the United States of America during 1952-5; the remainder occurred in Africa. Seventeen were in Africans, 12 were in Europeans. Whereas the 17 African cases do not in any way represent the true incidence of the condition in the African population, the 10 European cases seen in Rhodesia probably represent almost all those occurring in a white population of about 300,000 over a period of five and a half years.

The patients fall into two categories more or less divided along economic and racial lines. The average white Rhodesian enjoys a standard of living approximating to the English middle-class and attends early for medical treatment, while the black Rhodesian lives, with few exceptions, at a lower level. Patients from the latter group have a poorer standard of health (parasitic diseases and malnutrition being common) and tend to appear for attention at a much later stage of their diseases.

From the pathological standpoint, this series of 29 cases falls into two groups, group 1 consisting of solitary abscesses (19 cases) and group 2 of multiple abscesses (10 cases). Group 1 consists of 15 black and 4 white patients, and group 2 of 2 black and 8 white patients (Table I).

In group 1 12 patients were cured and 7 died. Six of the latter were Africans, three of whom (Cases 7, 14, and 15) arrived in hospital *in extremis*. One patient (Case 2) died after the elevation of a depressed fracture the fragments of which appeared uninfected but in reality were quite contaminated and infected the underlying brain—one feels that this patient should not have been lost—while in Cases 3 and 13 the actual cause of death was obscure. Better luck in one European case in this group might have produced a good result where a small

abscess of the internal capsule was missed by the aspirating needle. All except one patient (Case 2) had long-standing disease; the majority showed signs of chronically increased intracranial pressure on admission and their courses had corresponded to the oft-repeated description of the condition.

All patients in group 2 died. All but one attended very early for medical treatment and unquestionably survived only as long as they did because of strenuous antibiotic and supportive therapy. In only two cases was the pus adequately loculated so that surgical drainage could have been theoretically possible. In the remainder it was so disposed within the brain and in such small quantities as to make localization by conventional neurodiagnostic measures and surgical drainage quite impracticable. In at least four cases the presence of what were probably multiple septic infarcts serves to illustrate the magnitude and changing nature of the problem.

From the pathological and therapeutic standpoints there are two distinct groups of abscesses in this series—the solitary group with a 36% mortality, and the multiple group with a 100% mortality. The solitary group consists of 15 black and 4 white patients, while the multiple group consists of 8 white and 2 black patients. We therefore see that the two groups fall almost into patterns by race or, more correctly, by standard of living, the lower-standard group presenting the solitary abscesses, the higher-standard group the multiple ones. If we classify the patients in this latter fashion—that is, by race and economic level—the picture is more clearly defined.

On the one hand we have the 17 Africans, all but two of whom had solitary abscesses. These represent the few survivors of many patients living in the township and kraals who, with a poor standard of health and late attendance for treatment, die without ever coming to hospital or from the seriousness of their general disorder if they do, without proceeding as far as brain-abscess formation. Only those patients whose underlying disorder is sufficiently mild to produce a solitary abscess survive to reach the neurosurgical unit. At the same time, a number of those arriving do so when beyond the range of surgical help.

On the other hand, the 12 European cases, with a higher standard of living and early attendance for treatment, showed eight multiple and four solitary abscesses. The eight patients who developed multiple abscesses were those who had had infections serious enough to kill them at a relatively early stage, but who had been kept alive by early administration of antibiotics and energetic treatment long enough to develop the very serious complications from which they finally succumbed. Two of the four solitary abscesses in this group followed relatively mild infections (Cases 16 and 17); all other similar cases but these two having been prevented from developing to the stage of abscess formation by early treatment.

TABLE I.—Summary of Cases

Case No.	Race, Age, Sex	Cause	Site	Comment
<i>Group 1</i>				
1	African. 10. M.	Compound depressed fracture	Parietal	Recovered. Mildly hemiparetic
2	African. Adult. M	" "	Motor-parietal	Abscess followed elevation of fracture. Death from increased pressure
3	African. Adult. M	" "	Frontal	Died from cerebral infarction following drainage. ? cause
4	African. 10. M	Osteomyelitis following scalp abscess	Parietal	Recovered. Hemiplegic
5	African. 8. F	Osteomyelitis	Frontal	Recovered. Asymptomatic
6	African. Adult. M	Mucocele of sinus	"	" "
7	African. 28. M	Otitis media	Temporal	Admitted in coma. Died before treatment
8	African. Adult. M	Unknown	Occipital	Recovered. Asymptomatic
9	African. Child. F	"	Frontal	" "
10	African. Baby. M	Meningitis	Subdural	" "
11	African. Adult. M	Otitis media	Cerebellum	Recovered. Facial paresis
12	African. 23. M	Unknown	Occipito-parietal	Recovered. Asymptomatic
13	African. 25. F	Bronchiectasis	Frontal	Died. Cause of death obscure
14	African. Adult. M	Lung abscess	Cerebral hemisphere posteriorly	Died. Massive destruction of posterior half of hemisphere with basal meningitis
15	African. Adult. M	" "	" "	Died. Massive destruction of posterior half of hemisphere
16	European. 21. M	Meningitis	Internal capsule	Died. Abscess not located by needling
17	European. 45. M	"	Cerebellum, subdural and extradural spaces of posterior fossa	Recovered. Asymptomatic
18	European. 35. M	Compound depressed fracture	Subdural	" "
19	European. 19. M	Fracture through frontal sinus	Frontal	" "
<i>Group 2</i>				
20	European. 20. M	Sepsis complicating elevation of depressed fracture	(1) Frontal subdural. (2) Frontal intracerebral	Died. Only subdural pus detected at operation
21	European. 15. M	Tonsillitis and Sinusitis	(1) Subdural. (2) Hippocampus	Died, despite drainage of subdural space. Hippocampal pus not detected
22	African. 12. M	Meningitis	(1) Fronto-parietal involving subdural, subarachnoid, and cerebrum. (2) Cerebellopontine angle	Died
23	African. Adult. M	Otitis media	(1) Cerebellum. (2) Parietal	Died. Cerebellar abscess drained. Parietal abscess not detected
24	European. 50. M	Lung abscess	(1) Ventricles. (2) Left motor. (3) Right parietal	Died
25	European. 33. F	Acute bacterial endocarditis	Multiple septic lesions throughout brain	Died. Few emboli elsewhere
26	European. 8. F	" "	" "	Died. Few embolic lesions elsewhere
27	European. 28. M	Unknown. ? Meningitis	Many small abscesses throughout brain	Died
28	European. 31. M	Unknown	(1) Many small abscesses throughout brain. (2) Ventriculitis. (3) Meningitis	"
29	European. 31. M	Sinusitis	(1) Parieto-occipital. (2) Paramedian	"

Aetiology

Group 1 (Table II) shows a typical cross-section of the usual local causes of brain abscess, and there are three and possibly up to six metastatic abscesses in this group. There is a strong orientation towards trauma. In group 2, however, although local infections contribute their quota, 5 of the 10 cases appear to be metastatic—two from a septic endocardium, one from bronchopneumonia, and two from undetected sources which may have been cured by the antibiotic, only the metastases remaining. The emphasis in this group seems to be away from the local causes and towards the metastatic, and presumably these latter tend to produce multiple abscesses because of the severity and nature

TABLE II

	Group 1	Group 2
Trauma	6	1
Meningitis	3	1
Osteomyelitis of skull	1	-
Sinusitis	1	2
Otitis media	2	1
Lung abscess or bronchopneumonia	3	1
Cause unknown	3	2
Acute bacterial endocarditis	-	2

of the underlying infection and the many emboli thrown off. The cases in group 2 therefore represent all that remains of brain abscess in groups with a high standard of living and medical attention, cases such as those appearing in group 1 having been prevented by modern antibiotics.

The local pathology of the groups is also different. In the solitary group 18 of the 19 cases were either encapsulated or well loculated; the remaining patient died during the stage of encephalitis. In the multiple group, only two showed good loculation of the pus (Cases 20 and 23), while eight were neither encapsulated nor localized. Of these eight, four showed a diffuse spread of pus throughout the brain and subdural spaces and merited the title of acute diffuse non-encapsulated brain abscess as opposed to the chronic localized or encapsulated type. Botterell and Drake (1952) had five such cases in a series of 23. A feature is that they appear to be destructive and frequently not space-occupying. The remaining four were in fact little more than multiple septic infarcts—none the less the distinction between this and brain abscess is merely one of size. The infarcts were diffusely spread throughout the brain, some with pus formation, others merely with areas of inflammation.

Metastatic lesions occurring in the brain may thus be of three types: (a) acute non-encapsulated brain abscess; (b) one large abscess; (c) diffuse septic infarcts; and the mode of formation of the three different types affords grounds for speculation. The acute non-encapsulated type must be related to the most invasive organisms, and presumably a considerable number of these are embolized at the same time so that a diffuse destructive lesion is rapidly produced. In Case 17 the time between the patient first feeling ill and the detection of pus in his brain was only 10 days. In the large solitary metastatic abscesses the incubation period is longer and presumably the invasiveness of the organisms less. In Case 8 symptoms of increased intracranial pressure had been present for four months before the patient came to hospital. It would seem unlikely even in the solitary metastatic cases that one embolus alone is responsible; it is more probable that a considerable number leave together and more or less fortuitously plug

several vessels in close proximity, so that the body defences have difficulty in approaching the infected area and a nidus of dead tissue is provided on which a foothold is gained. This would certainly seem to be so in Cases 14 and 15, where there were large areas of destruction by apparently solitary abscesses. In the chronic septic embolic type the invasiveness of the organism may be low but the source must have a relatively easy access to the blood-stream, so that the repeated emboli can be fired and diffuse infarction of the brain and other organs takes place.

The organisms responsible are shown in Table III.

TABLE III

	Group 1	Group 2
<i>Staphylococcus aureus</i>	3	5
<i>Staph. aureus</i> and coliforms	2	
<i>Staph. aureus</i> and haemolytic streptococci	1	
Pneumococci	2	
Haemolytic streptococci	1	
Blastomycosis	1	
<i>H. influenzae</i>	1	
Meningococci	1	
Meningococci and streptococci		1
<i>Proteus vulgaris</i>	1	
Pus culture sterile	2	
All tests negative for organisms		2
C.S.F. sterile pus not cultured	1	2
Culture not obtained from pus or C.S.F.	3	1

Group 1 shows a reasonable cross-section of all the organisms responsible for body infections though *Staphylococcus aureus* is the prime offender. In group 2 *Staph. aureus* is even more predominant, accepting 50% of the responsibility, while in a further three cases it was impossible to isolate any organism and sterile pus is a marked feature of both groups.

Four cases from the multiple-abscess group are reported in more detail as they serve to typify the problems posed by this group of cases.

Case 21

A 15-year-old European boy suffered from tonsillitis and sinusitis for 10 days prior to coming under observation. For three days he had been drowsy and vomiting. A right hemiparesis and aphasia had developed. A left fronto-temporal burrhole demonstrated a subdural abscess, which was drained. The patient's condition remained poor and the following day craniotomy and frontal sinusotomy was performed. Large quantities of pus were obtained from both sites, the entire subdural space on the left side being involved. Culture showed the organism to be a *Staph. aureus*. For a short while the patient's condition improved, but once again his temperature started to swing, although pneumo-encephalography and right temporal and parasagittal burrholes, together with re-exploration of the old operative site, showed no other abnormality. His condition deteriorated and he died within a few days. Post-mortem examination showed that the entire left hippocampal gyrus was destroyed, and was filled with abscess material (in addition to the subdural abscess) which could not be detected because it was destructive rather than space-occupying.

Case 24

A 50-year-old European man suffered an attack of influenza during the influenza pandemic of 1957 which progressed to pneumonia. On the day prior to his coming under observation he had suffered from tingling in the right arm, with fever and headache. Some vague right-sided cerebellar signs were present. He was observed closely, but two days later he suddenly became comatose. Burrholes demonstrated pus in both lateral ventricles. A drain was inserted, but his condition deteriorated and he died. Post-mortem examination showed abscesses of both lateral

ventricles and of the left motor area and right parietal regions. A primary abscess was found in the base of the left lung. The infecting organism was a *Staph. aureus*.

Case 25

A 33-year-old European woman had a tubal ligation which was complicated by a brief attack of paralytic ileus, left pulmonary embolism, and left subclavian thrombophlebitis. She became jaundiced for a while but recovered. Two months later she developed generalized aches and pains, headaches, and pyrexia. C.S.F. protein was 50 mg. with 72 polys/c.mm. Urine examination showed casts and squamous and red cells. Urine culture was sterile but blood culture showed *Staph. aureus*. Examination of the heart was normal. The C.S.F. pressure rose to 300 mm. of C.S.F., with 1,030 leucocytes (polys 90%). There were no localizing signs, clinically, by E.E.G., or by cerebral arteriography. A diagnosis of multiple inflammatory intracerebral lesions (aetiology unknown) was made and a bilateral subtemporal decompression performed in an effort to tide her over while the blood could be sterilized. She continued to deteriorate and died. Post-mortem examination showed a bacterial endocarditis with infarctions of the kidney and spleen. There were numerous small metastatic inflammatory lesions throughout the cerebral substance, mostly without pus, though this was found in one larger abscess and in several of the smaller ones. The cerebral lesions completely dominated all others.

Case 28

A 31-year-old European man had developed headache, diplopia, sensory attacks in the left arm, and dysphasia in 1948. Wassermann reaction was negative. In 1952 a subcutaneous gland had been excised which was proved by guinea-pig test to be tuberculous. C.S.F. examination at that time showed a protein of 130 mg./100 ml., and 3 lymphocytes. He was given a course of streptomycin and isoniazid. In November, 1953, he developed headache, vomiting, numbness of the right side, and tingling of the left foot. C.S.F. examination showed a protein of 190 mg./100 ml., with 35 lymphocytes, and a pressure of 290 mm. of C.S.F. E.E.G. showed a right-sided abnormality. Bilateral subtemporal decompression was performed by Professor Norman Dott and the condition settled down.

In August, 1957, he was living in Rhodesia, when he again developed headache, together with a left hemiparesis. His decompression became tense. Cerebral arteriography showed that the anterior cerebral artery was shifted from right to left, and this shift was confirmed but without satisfactory location by ventriculography. The right-sided decompression was greatly enlarged and a yellow arachnoid with three areas of fluid pus was seen. Culture of this fluid was sterile and guinea-pig test was negative. The patient died and post-mortem examination showed numerous small abscesses throughout the cerebral substance with ventriculitis and basal meningitis. These abscesses were sterile and no tubercles were seen on histological section. A primary focus was not found and the aetiology of the infective organism could not be determined.

Discussion

Improvement in the outlook in brain abscess during the bacteriostatic and antibiotic eras has been noted by various authors. Pennybacker (1950) observed that the mortality from this condition fell from 90% to 46% after the advent of sulphonamides and from 46% to 29% after penicillin appeared. Loeser and Scheinberg (1957) divided their series of 88 abscesses into three groups—pre-antibiotic, penicillin, and antibiotic periods—and found that the mortality was 47%, 24%, and 19% respectively. The decline in the mortality of the disease in these series contrasts sharply with the 242 reported

cases collected by Sir William Gowers up to 1888, all of which were fatal.

Nevertheless, the mortality for multiple brain abscesses in almost everyone's hands is 100%. Ramamurthi and Narasimhan (1957) had two multiples in 62 patients—both died; Sperl, MacCarty, and Wellman (1959) lost all three multiples out of 60 cases, though Loeser and Scheinberg (1957) saved two out of eight appearing in a series of 88 cases. In this series all 10 patients died. While we must therefore welcome the undoubted reduction in disease incidence, morbidity, and mortality produced by early antibiotics and adequate treatment of predisposing lesions, the experience of this series would appear to be threefold: firstly, that the overall incidence of brain abscess will be reduced in frequency in those parts of the world where the standard of living is high; secondly, the commonly occurring solitary abscesses will be replaced by the multiple kind, which are virtually untreatable by surgical means; and, thirdly, that by the time almost any abscess comes under observation pus is present and demands release. Therapy directed towards decompression and temporization would not seem to be indicated, though this is partially explained by the fact that in the solitary group many patients did not come to hospital until the symptoms were very serious, while in the multiple group a very invasive organism was undoubtedly present. Ramamurthi and Narasimhan (1957) stress the importance of the preliminary inflammatory phase in their series of 48 patients, who must resemble many of the patients studied here, yet their conclusion is not supported by this evidence.

In this series of 29 brain abscesses, 10 (34%) were multiple, and all 10 patients died. If this is the shape of things to come it would seem that the wheel of mortality may turn a full circle back to Sir William Gowers and his 100% death rate for the condition. If it does so we can only take consolation in the fact that those cases which would previously have been

amenable to surgery have been removed by the antibiotic filter and have never proceeded as far as brain abscess formation.

Summary

A series of 29 cases of brain abscess occurring in a mixed European and African population is reported. The cases are divided into two groups, solitary abscesses (19 cases) and multiple abscesses (10 cases). The solitary group consists almost entirely of Africans and the multiple group of Europeans. The mortality in the solitary group is 36%, in the multiple group 100%. It is my contention that Africans predominated in the solitary group because of their poorer standard of living and health and late attendance for medical treatment, so that any infection severe enough to produce a multiple abscess kills the patient before such a complication sets in. On the other hand, Europeans have a high standard of living and attend early for medical treatment. Mild infections sufficient to produce a solitary abscess are controlled before any complications can occur. Those infections, previously severe enough to kill the patient at an early stage of the illness, produce multiple abscesses because the patient now survives long enough to develop these complications. Thus with increasing standards of living, although the overall incidence of brain abscess will be reduced, the abscesses that will be seen by clinicians will become more severe.

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REFERENCES

- Botterell, E. H., and Drake, C. G. (1952). *J. Neurosurg.*, **9**, 348.
 Loeser, E., and Scheinberg, L. (1957). *Neurology*, **7**, 601.
 Pennybacker, J. (1950). *Ann. roy. Coll. Surg. Engl.*, **7**, 105.
 Ramamurthi, B., and Narasimhan, S. T. (1957). *J. int. Coll. Surg.*, **28**, 589.
 Sperl, M. P., MacCarty, C. S., and Wellman, W. E. (1959). *Arch. Neurol. (Chic.)*, **81**, 439.

RENAL ARTERIOVENOUS FISTULA AFTER PERCUTANEOUS RENAL BIOPSY

BY

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Arteriovenous fistula of the renal vessels is a rare condition. A total of 32 cases have been reported in the literature (Twig, Pradhan, and Perloff, 1962; Boijesen and Kohler, 1962). Of these, 13 were thought to be congenital, three were due to renal carcinoma, and five were of traumatic origin. The remainder followed surgical procedures, usually nephrectomy, with development of fistula in the vascular pedicle. However, arteriovenous fistula can also occur after percutaneous needle biopsy, and the purpose of this communication is to draw attention to the possibility of this complication of a relatively common investigation and to report two cases.

Case 1

A girl aged 19 presented with headache and was found to have a blood-pressure of 170/110. There was no cardiac enlargement or gallop rhythm. The fundi were normal. No evidence of coarctation was found. A faint systolic

bruit was audible in the epigastrium to the right of the midline.

There was no proteinuria; the blood urea was normal. Urine culture was negative, but the white-cell excretion rate was grossly elevated. The 24-hour catechol excretion was in the normal range. Serum potassium was normal. An intravenous pyelogram showed nothing abnormal. Divided renal studies showed a significant increase in creatinine excretion and a diminution in sodium on the right side. Renal angiography showed no definite abnormality, but the proximal part of the right renal artery was overlapping the spine, and was not adequately visualized. Left renal biopsy produced an adequate specimen of cortex together with a tiny portion of medulla. Histological examination showed no abnormality. The patient was discharged on hypotensive therapy. Six months later reinvestigation confirmed the previous results of divided renal studies and white-cell excretion rate. Renal angiography showed a moderate stenosis of the proximal right renal artery. In addition, however, there was a large arteriovenous fistula in the mid-zone of the