## ANNALES UNIVERSITATIS MARIAE CURIE-SKŁODOWSKA LUBLIN — POLONIA

**VOL. L, 6** 

SECTIO D

1995

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### Sinusogenous Intracerebral Inflammatory Complications in CT-Imaging

Powikłania zapalne śródmózgowe zatokopochodne w obrazowaniu TK

Purulent sinusogenous, interacerebral changes belong to rare complications in the era of modern antibiotics (3, 15, 18). It should be noted that the spread of infections from sinuses, despite treatment, has not been completely eliminated. Altered and delayed clinical symptoms also reduce the vigilance of diagnosing intracerebral complications (9).

The aim of this report is, therefore, turning attention to the possibility of intracranial complications of sinusitis in cases of exacerbating headaches.

### MATERIAL AND METHODS

The clinically and operatively verified material comprises 5 cases of sinusal abscesses investigated at the Radiology Department of the District Hospital in Kielce, in the years 1990—1995. Patients of both sexes, aged 9—68 years, were treated on sinusitis of different etiology. In 2 patiens inflammation was associated with sustained trauma.

The examinations were performed with a CT apparatus made by Siemens, Somaton H and Q type representing the fourth generation with high spatial and contrast definition. Standard X-rays and coronary scans 3 mm thick revealed opacities of pneumatic cavities of the nose and sphenoidal sinus. Exacerbating headaches, of permanent character, vomitting and neurologic symptoms were indications for repeated CT examinations in the axial projection with 8 mm scan thickness. Empty scans were taken and scans after intravenous administration of 75% Uropolin in the dose not exceeding 2 mg per 1 kg body weight. A repeated examination was performed after the period from 7 weeks to 2 years.

#### RESULTS

In 2 persons abscesses in frontal lobes were observed and in 1 case an abscess in the parieto-temporal region. Empty scans revealed irregular areas of decreased density and blurred contours, surrounded by a zone of oedema (Fig. 2b, 3b). There was also observed lack of a distinct annulus or it was only visible with poor intensity (Fig. 1b).

Contrast scans showed intense circinate enhancement having the character of homogeneous, dense, thin, regular ring (Fig. 1c, 2c). In 1 case the areola of the enhancement had differentiated thickness and density (Fig. 3c), while air filled frontal horns and partly the abscess cavity. A mass effect also occurred in the form of deformation, compression and displacement of the cortical cerebral system through the medial plane, especially of frontal horns of lateral ventricles (Fig. 1b, c, 2b). The abscess was measured in 3 planes since abscesses not exceeding 4 cm in diameter are treated conservatively.

#### DISCUSSION

Nowadays a cerebral abscess is an exceptional complication of sinusitis. All the same sinuses are mentioned as a cause of intracranial abscesses, especially in children (11, 14). An infection can spread through an inflammatorily changed bone or posttraumatic defects (bone route), inflammations of venous and arterial vessels (venous route), osseous canals (anatomic route) — (7, 9). K uś (10) lists the following ways of spreading interacranial infections, by: contact, permanence, preformed canals and metastases. Chun et al. (2) observed in their material 70% of intracerebral abscesses caused by haematogenic spread from sinuses, ear and dental focus.

A special kind of foci are posttraumatic changes (6). Defects of bony walls of sinuses, fissures, displaced bone fragments, denudating of the meninges of the anterio-inferior portion of the skull with secondary opacity of adjacent pneumatic spaces of inflammatory character, can be the starting point of intracranial complications (13).

In Yang's material (18) comprising 400 intracranial abscesses only two cases had nasal origin. In Nielsen's material (11) abscesses of the frontal lobe constituted 24%, out of which one third of cases had the sinusal origin. Complications usually set in in the closest vicinity of the focus (10).

CT scans reveal the stages of abscess formation from diffuse inflammation to the stage of mature encysting (8). Clinical stages of the cerebral abscess were determined by means of serial, contrast CT scans.

The criteria of assessment of abscess formation stages are based on the picture of contrast enhancement (1). There are distinguished periods of early and late "cerebritis" and of late occurrence of the cyst (1, 5).

In the period of early "cerebritis" an irregular area of small density does not get enhanced or reveals a weak, heterogeneous circinate enhancement after 10 min. There was shown lack of any significant enhancement on scans taken after 30—60 min. In the period of late "cerebritis" a thick, diffuse, poorly limited circinate enhancement appears right after contrast injection. The curve of time density of the enhancement increases in 10—20 min. and a constant enhancement level is thus maintained on delayed scans, on which it can have nodular appearance and even get enhanced after 30—60 min. Contrast diffuses to the pus-containing necrotic center causing partial or total filling of the central space. An extensive, low density cerebral oedema surrounding the abscess remains unchanged.

An early period of cyst formation is characterised in an empty scan by a weak areola of higher density than the necrotic center and oedematous brain. The image of the non-contrast areola corresponds to a developing wall cyst and inflammatory infiltrate of the necrotic center margins. After contrast infusion, a well restricted, thin, high density areola can be of unequal thickness. It can be thinner in the medial or ventricular part. A permanent cyst is formed by fibrous bands developing in the abscess wall. In the encysting stage the necrotic center is surrounded by a sac of mature colagen and a gliosis zone. A homogeneous annulus usually correlates with a cystic formation. Encysted formations show fast circinate enhancement (5—10 min.) fading after 30 min. After the initial peak an anhancement reduction occurs. In delayed pictures no contrast diffusion or filling of central transparency is observed. A low density center, not filled with contrast, corresponds to pus-necrotic contents (5).

In the late stage of cyst formation a weakly visible areola appears in an empty scan. It is formed by a colagen sac restricted by low density of the necrotic enter and of the surrounding cerebral oedema. In 70% cases there appears after enhancement a thin, low density areola of uniform thickness with a soft internal contour (5). The peak of intensity occurs after 5—10 min. Delayed scans (30—60 min.) reveal a considerable reduction of enhancement level. In a well encysted abscess the contrast does not spread to the transparent center. The situation is similar after 30—60 min. and this accounts for maturity of the abscess cyst.

A "typical" ring can apper even at an early stage of localised cerebritis. It then corresponds to an inflammatory infiltrate limiting the necrotic center of the developing abscess (12). The ring at an early stage is a marginal inflammatory infiltrate with vascular proliferation giving an enhancement effect. In time, connective tissue develops forming a "real sac" dependent on the amount of connective tissue and age of the abscess (12). Circinate enhancement can vary. Delayed cyst formation in the part in the deep white matter results from its relatively poor vascularisation. Limited encysting suggests a septic, metastatic abscess.

The following zones are distinguished in abscess formation: 1) the necrotic center, 2) inflammatory margins, 3) colagen sac, 4) tenacious "cerebritis" and vascular neogenesis, 5) reactive gliosis and surrounding cerebral oedema (1).

Circinate enhancement sharply internally restricts uniform central areas of reduced density. In big abscesses the annulus is narrow and smooth. In small abscesses it is thick and irregular and requires differentiation with a tumour with central necrosis. Revealing the annulus makes it possible to border the abscess from marginal oedema. Clinical improvement decreases the circinate enhancement and the surounding swelling and mass effect. Operation results in abscesses depend on the form of the sac.

Circinate enhancement does not reflect good development of sac walls (14). They are formed by an areola (the so-called "halo") of contrast in the area of impaired permeability of focus surrounding vessels. The assessment of the specificity of circinate enhancement in differentiating abscesses with other diseases is often difficult (4, 14, 16).

Enhancement is not a reliable indicator of the developmental stage of a focal infection (4). Fallibility of CT circinate enhancement is well known in differentiating "cerebritis" and encysted abscesses (4). Although circinate enhancement is characteristic of encysted abscesses, there are numerous cases showing its non-specifity in focal intracerebral infections (4).

Circinate enhancement, typical of the abscess, was also found at an early stage of "cerebritis" (4). The enhancement can have the character of an irregular or nodular annulus (2).

Y ang et al. (19) showed in a group of 17 abscesses in the "cerebritis" stage a solid enhancement in 9 cases, incomplete one in 2 cases, full thick-walled circinate enhancement in 6 cases. In big abscesses CT can reflect differences in wall development, which on the cortical side is at an early cystic stage, while the cortical part of the wall is at an early "cerebritis" stage. About 40-50% abscesses reveal annulae of differentiated thickness (1). In tumours the enhancement annulae are usually thicker and have more often nodular character, though similar ones can occur in "cerebritis" and at an early stage of cyst formation.

## Conclusions

1. Images of contrast enhancement let determine the stage of focal development of the cerebral nervous tissue inflammatory process.

2. Serial, delayed scans show the degree of abscess encysting, which is of practical value in planning therapy.

3. Enhancement can show different stages of wall development of the same abscess depending on time.

#### REFERENCES

1. Britt R., Enzmann D.: Clinical stages of human brain abscesses on serial CT scans after contrast infusion. J. Neurosurg. 59, 972, 1983.

- 2. Chun C. et al.: Brain abscess. A study of 45 consecutive cases. Medicine Baltimore 65, 415, 1986.
- 3. Dietrich U. et al.: Epidural abscess following frontal sinusitis demonstration of communication by epidural contrast medium and coronal computerized tomography. Comput. Med. Imaging-Graph. 13, 351, 1989.
- 4. Dobkin J. et al.: Nonspecificity of ring enhancement in "medically cured" brain abscess. Neurology 34, 139, 1984.
- 5. Enzmann D. et al.: Staging of human brain abscess by computed tomography. Radiology 146, 703, 1983.
- Farrell V., Emby D.: Meningitis following fractures of the paranasal sinuses: accurate, non-invasive localization of the dural defect by direct coronal computed tomography. Surg. Neurol. 37, 378, 1993.
- 7. Goździk-Żołnierkiewicz T.: Uszne i zatokopochodne powikłania wewnątrzczaszkowe o charakterze ropnym, [in:] Brzozowski R., Januszkiewicz J.: Ropne zakażenia ośrodkowego układu nerwowego. Warszawa 1987, 88.
- 8. Henze T. et al.: Difficulties in the diagnosis of brain abscesses. Neurosurg. Rev. 10, 321, 1987.
- 9. Kaplan K.: Brain abscess. Medical Clinics North America 69, 345, 1985.
- K uś J.: Usznopochodne i zatokopochodne powikłania zapalne wewnątrzczaszkowe. Choroby infekcyjne układu nerwowego. Warszawa 1981, 73.
- 11. Nielsen H. et al.: Cerebral abscess. Acta Neurol. Scandinav. 65, 609, 1982.
- 12. Niezabitowski K.: Współczesne możliwości diagnostyki radiologicznej ropnych schorzeń ośrodkowego układu nerwowego [in:] Brzozowski R., Januszkiewicz J.: Ropne zakażenia ośrodkowego układu nerwowego. Warszawa 1987, 24.
- 13. Pietilä T. et al.: Subdural effusions: determination of contrast medium influx from CSF to the fluid accumulation by computed tomography as an aid to the indications for management. Acta Neurochir. (Wien.) 118, 103, 1992.
- 14. Piszczor M. et al.: The evaluation of contrast-enhancing brain lesions: pitfalls in current practice. Yale J. Biol. Med. 58, 19, 1985.
- Remmler D., Bales R.: Intracranial complications of frontal sinusitis. Laryngoscope 90, 1814, 1980.
- 16. Salzman C., Tuazon C.: Value of the ring-enhancing sign in differentiating intracerebral hematoma and brain abscesses. Arch. Intern. Med. 147, 951, 1987.
- 17. Weisberg L.: Central computerized tomography in intracranial inflammatory disorders. Arch. Neurol. 37, 137, 1980.
- 18. Yang S.: Brain abscess: a review of 400 cases. J. Neurosurg. 55, 794, 1981.
- 19. Yamg S., Zhao Ch.: Review of 140 patients with brain abscess. Surg. Neurol. 39, 290, 1993.

Otrzymano 1995.03.20

### **EXPLANATIONS TO FIGURES**

Fig. 1: a — coronary scan — inflammatory opacity of maxillary sinuses, nasal cavities and ethmoidal cells; b — axial plain scan — less than moderately visualised low-density ring in the right frontal lobe, surrounded by extensive cerebral oedema; c — contrast scan-shows uniform, hyperdensic, circinate enhancement with thin, smooth wall (abscess cyst); brain stem slightly twisted, stem containers constricted; displacement of frontal horns.

Fig. 2: a — coronary scan shows opacity of ethmoidal cells, sphenoidal sinus and maxillary sinuses; rhinorrhea occurs after trauma sustained 2 years before; b — axial plain scan; in peribasal layers of the antero-medial part of the left frontal lobe visible an oval hypodensic focus with a trace

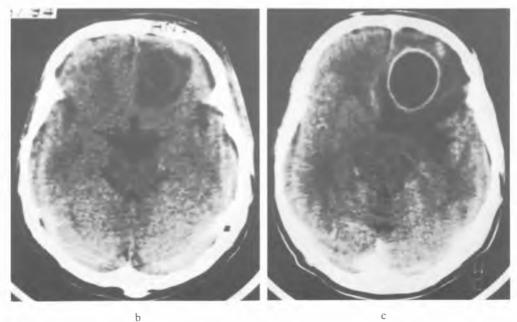
circinate ring; it is accompanied by an extensive, hypodensic CT imaging; zone of oedema within the frontal lobe; c — contrast scan shows intense circinate enhancement, regular, sharply contoured; a fragment of the frontal horn of the left cerebral ventricle compressed and displaced through the medial plane toward the opposite side; structures of the cerebral falx in the anterior part less than moderately brought into prominence to the right; marked constriction of the left temporal horn and containers surrounding the brain stem.

Fig. 3: a — CT imaging; the scan visualises posttraumatic changes of the lateral and posterior wall of the right eye-socket and fractures within the anterior part of the ethmoid as well as bilateral fractures of lateral walls of sphenoidal sinuses with sinusal opacity; free bone fragments in the posterior part of the eye-socket intussuscepted into the infratemporal fossa; b — axial plain scan shows extensive, heterogeneous, hypodensic oedematous area and air within widened frontal horns (examination 7 weeks after trauma); c — contrast scan visualises in the left parieto-temporal region a polycavernous formation with a circinate ring of irregular thickness and poor intensity.

#### STRESZCZENIE

Zweryfikowany klinicznie i operacyjnie materiał 5 przypadków ropni zatokopochodnych analizowano w obrazie TK, określając przydatność seryjnych kontrastowych przekrojów w ocenie stadium rozwoju ropnia. W okresie wczesnego cerebritis pole zmniejszonej gęstości wykazywało brak lub słabe niejednorodne wzmocnienie obrączkowate. W okresie późnego cerebritis grube rozlane wzmocnienie obrączkowate pojawia się bezpośrednio po iniekcji kontrastu, utrzymując, a nawet zwiększając intensywność na przekrojach opóźnionych. Wczesny i późny okres tworzenia torebki charakteryzuje na przekroju miejsce puste z miernie uwidocznioną obwódką. Ulega ona szybkiemu wzmocnieniu kontrastowemu, zwiększając gęstość i ujawniając regularne zarysy. Zróżnicowane wzmocnienie obrączki może odzwierciedlać różne okresy rozwoju ściany ropnia.



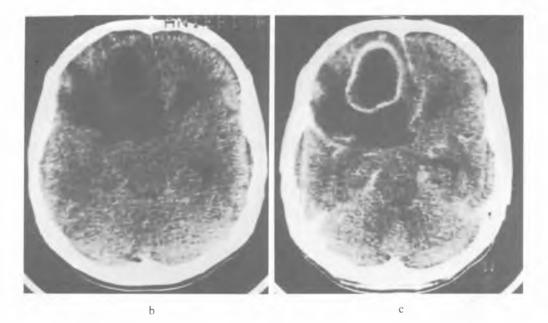


b



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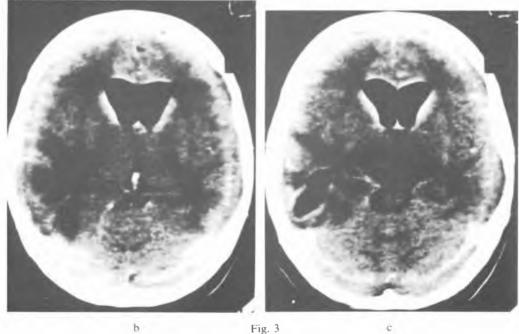


Fig. 3

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