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The Case of Clinically Benign Spino-Cerebral Meningitis in the Development of Cranial Basis Neoplasm

Przypadek łagodnego klinicznie zapalenia opon mózgowo-rdzeniowych w rozwoju nowotworu podstawy czaszki

Primary and secondary neoplasms of the cranial basis cause, according to their location, the formation of different neurological syndromes. The damage of cranial nerves, mainly of oculomotor, abducent, trigeminal, facial and acoustic nerves occurs (2, 5, 8). There is often found unilateral or bilateral damage of oculomotor nerves, sometimes with exophthalamus, as well as facial dysaesthesia with untypical trigeminal neuralgia or vegetative disturbances. The consequences of damage of long nerve tracts, pyramidal and sensory, as well as cerebellar symptoms occur very seldom. Of the neoplastic process covers the hypothalamus and hypophysis, it may be the cause different endocrinological disorders (1, 3). The extension of growth of these neoplasms into intracranial space causes epileptic seizures. The symptoms of intensified intracranial pressure occur seldom (1—3, 6, 8). Sometimes complications occur, such as corneal ulcerations, inflammation of all the tissues and structures of the eye, primary atrophy of nerve II, presence of air in cerebral ventricles, meningitis, encephalitis and brain abscesses (2, 9).

CASE DESCRIPTION

The patient L. S., aged 65, a pensioner. At the age of 35 she stopped having menstruation and for this reason was treated in Gynecological Outpatient Clinic. In March 1970 the patient was hospitalized in Neurology Clinic due to occurrence of transient damage of the left oculomotor nerve. A roentgenogram of the skull visualized blurring of the fundus and of the dorsum of the sella turcica, an open sphenoid sinus and blurring of the left pyramid apex (destruction). The clinical image spoke of intracranial expanding peri- and suprasellar lesions.

In November 1982 strong pains in the left part of the head and bleeding from the left nasal meatus occurred in the patient. In neurological examination the

narrowing of the left palpebral fissure, limitation of the upward movement of the left eyeball at the correct visual area and slightly paler disc of nerve II were found. A roentgenogram of the skull showed destruction of the sella on the left side and of the left pyramid apex. Angiographic examination was suggested, but the patient refused to go through them.

In 1986 coronary arterial disease occurred as well as hypertension and diet-controlled diabetes treated with oral drugs.

In October 1988 the patient was admitted to Neurology Clinic due to acute headaches persisting for three days, chills, nausea and vomiting. The fever was up to 39°C.

General examination of the patient did not reveal any perceptible deviations from the normal state. Neurological examination showed distinct meningeal signs, exophthalmus of the left eye, narrowing of the left palpebral fissure, limitation of the upward movement of the left eyeball at normally preserved visual area. The discs of nerve II bilaterally pale. No symptoms were found as regards long sensory and pyramidal tracts, cerebellar signs or anomalies testifying to an intensified intracranial pressure.

OB — 100/160 mm. RR — 120/80 mm Hg, leucocytosis — 21 500, glucose in blood — 160 mg% and trace amount of sugar in urea. Other laboratory examinations of blood and urea did not show any deviation from the norm. Cerebrospinal fluid was incompletely clear, cytosis — 4608/3, mostly polynuclears, protein 1.45 g/l, glucose — 2.28 mmol/l. The result of cerebrospinal fluid culture was negative.

The result of X-ray examination was normal. A roentgenogram of the skull revealed porosis of the skull-covering bones, extensive calcification in the cerebral falx, descruction of the sella with visible calcifications of this area. The sphenoid sinus opacified, with no contours. Destruction of the clivus. Anterior and posterior sloping processes invisible. Destruction of pyramids apices. Foramen: ovale, rotundum and lacerum invisible. CT examination of the head showed an extensive expanding process looking like sellar-suprasellar tumour in the form of extensive pathological tissue of hyperdense structure. Downwards tumour filled the sieve bilaterally, it was within the pterygopallatine fossa, destroying bony frame; it filled subtemporal fossa and caused the complete destruction of the sella turcica. Upwards it reached higher than suprasellar cisterns, nearly up to the height of ventricle III. The wings of the sphenoid bones were also subject to bilateral destruction. The tumour underwent a distinct contrast intensification.

Gynecological examination was within normal range. Laryngological examination — auriculoscopy, rhinoscopy, endoscopy of mouth cavity and pharyngoscopy did not reveal any visible pathological changes. X-ray examination of the nasal collateral sinuses showed opacity of the left maxillarly sinus with blurring of the superior wall bone structure (orbital and medial). Carrying out of the puncture of the sinus and segment sampling were renounced.

The examination of cerebrospinal fluid showed that it was translucid and water-clear, cytosis — 48/3, mostly mononuclears, protein — 0.99 g/l, glucose — 2.8 mmol/l.

The patient was not classified for neurosurgical intervention, however she was transferred to oncological hospital with the aim of carrying out palliative irradiation.

DISCUSSION

The tumor developing on the cranial base in the above patient looks like a primary tumour. The long lasting development of the tumour and large destructive lesions on the cranial base at the minimum neurological symptoms are very interesting. On the basis of the interview it may be presumed that the neoplastic process has been developing in the patient for about 30 years, if endocrinological disorders are to be considered the first symptom of the tumour, or, for at least 18 years, taking into account the time when radiological symptoms of the cranial bones destruction were found. Long lasting development of the neoplasm testifies to its benign nature. Long lasting and slow development makes the adaptation to the changes taking place easier and damage of the nervous system smaller. It is well-known from literature that occasionally a difuse destruction of the cranial base may develop without any particular neurological symptoms (1—5).

The clinical image of the cranial base neoplasm in the patient was oligosymptomatic, however distressing were the symptoms of acute meningitis. The reactive process in the patient might have been the cause of inflammatory changes in the spinocerebral meninges. The possibility of purulent meningitis as the consequence of pathological junction of submeningeal spaces with nasal sinuses or nasopharynx has also been considered. The quick regression of inflammatory changes in the spino-cerebral fluid testified to the reactive nature of meningitis. The occurrence of such a form of meningitis in the course of neoplastic process of the cranial base depends on the junction between the tumour and the meninx. For a considerable period of time the meninx functions as a protective barrier and is only displaced by the tumour. Martin contends that the meninx is the barrier that protects against spreading of neoplasms (2). Sometimes, however, tumours spread along the submeningeal space, causing the characteristic symptoms of brain damage. The resistance of the meninx is also important in prevention from the spreading of infection out of potentially highly infected nasopharynx. If the infection passes pachymeninx, it may cause meningitis, encephalitis or brain abscess (2, 7, 9).

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STRESZCZENIE

Opisano przypadek prawdopodobnie reaktywnego zapalenia opon mózgowo-rdzeniowych w przebiegu nowotworu podstawy czaszki. Zwrócono uwagę na długi okres rozwoju nowotworu, bardzo duże zmiany destrukcyjne na podstawie czaszki przy minimalnych objawach neurologicznych.