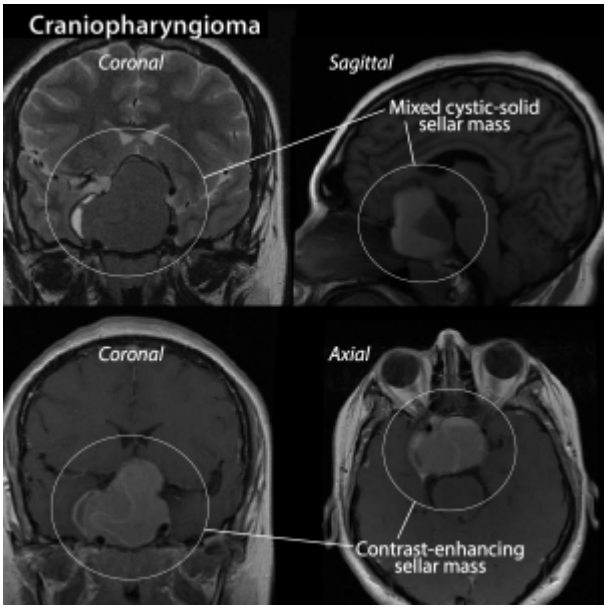
Craniopharyngioma Cyst Fluid



The dense oily fluid content of craniopharyngioma CPs is reported to cause brain tissue damage, demyelination and axonal loss in the hypothalamus; however, its exact effect on different cell types of CNS is still unexplored.

One cause of postoperative morbidity, and indeed mortality, is aseptic meningitis from spill-out of craniopharyngioma cyst contents.

Halliday and Cudlip from the John Radcliffe Hospital, developed a surgical technique for the management of large craniopharygngioma cysts extending into the third ventricle, to reduce this risk.

They described a technique of using an epidural catheter, inserted into the working channel of a neuroendoscope, to decompress the cystic portion of a craniopharyngioma cyst before opening the cyst wall widely, preventing spill-out of large volumes of cyst content into the ventricular system.

They had no cases of aseptic meningitis, nor any complications, from use of the described technique.

They believe that this is a safe and effective technique of decompression and fenestration of large suprasellar craniopharyngioma cysts that reduces rates of aseptic meningitis and the associated morbidity and mortality from this [1)]

In a study, Ghosh et al. from Bangalore, collected CP cyst fluid (CCF) from mostly young patients during surgical removal and exposed it 9-10 days in vitro to the primary cultures derived from rat brain hypothalamus for 48 hours. A gradual decline in cell viability was noted with increasing concentration of CCF. Moreover, a distinct degenerative morphological transformation was observed in neurons and glial cells, including appearance of blebbing and overall reduction of the cell volume. Further, enhanced expression of Caspase-3 in neurons and glial cells exposed to CCF by immunofluorescence imaging, supported by Western blot experiment suggest CCF induced apoptosis of hypothalamic cells in culture.

They demonstrated the deleterious effects of the cyst fluid on various cell types within the tumors

originating region of the brain and its surroundings for the first time. Taken together, this finding could be beneficial towards identifying the region specific toxic effects of the cyst fluid and its underlying mechanism [2)].

Craniopharyngiomas (CPs) are cystic, encapsulated, slow-growing epithelial tumors. CPs can be aggressive forms invading and resorting surrounding structures of adjacent brain tissue, where Rosenthal fibers (RFs) are expressed. The aim of this study was to investigate the ultrastructure of these fibers in human biopsies and compare it with an experimental toxic model produced by the cortical infusion of the oil cyst fluid ("Oil machinery" fluid or OMF) from CPs to rats. For this purpose, the CPs from ten patients were examined by light and electron microscopy. OMF was administered to rats intracortically. Immunohistochemical detection of glial fibrillary acidic protein (GFAP) and vimentin was assessed. In both freshly obtained CPs and rat brain tissue, the presence of abundant cellular debris, lipid-laden macrophages, reactive gliosis, inflammation and extracellular matrix destruction were seen. Ultrastructural results suggest focal pathological disturbances and an altered microenvironment surrounding the tumor-brain junction, with an enhanced presence of RFs in human tumors. In contrast, in the rat brain different degrees of cellular disorganization with aberrant filament-filament interactions and protein aggregation were seen, although RFs were absent. Our immunohistochemical findings in CPs also revealed an enhanced expression of GFAP and vimentin in RFs at the peripheral, but not at the central (body) level. Through these findings we hypothesize that the continuous OMF release at the CPs boundary may cause tissue alterations, including damaging of the extracellular matrix, and possibly contributing to RFs formation, a condition that was not possible to reproduce in the experimental model. The presence of RFs at the CPs boundary might be considered as a major criterion for the degree of CPs invasiveness to normal tissue. The lack of RFs reactivity in the experimental model reveals that the invasive component of CPs is not present in the OMF, although the fluid per se can exert tissue damage [3)].

Fifteen samples of cyst fluid and 14 samples of blood serum were collected from 14 patients with cystic forms of craniopharyngiomas and studied biochemically regarding total protein, albumin, immunoglobulins G and M contents, lactate and pH. Analysis of the data obtained for cyst fluids according to Felgenhauer and comparing them to those obtained for the corresponding blood sera led us to prove the hypothesis of blood-brain barrier impairment in patients with cyst formations in craniopharyngioma.

Arefyeva et al. have also revealed an elevated lactate content and decreased pH in cyst fluids compared with blood sera. Thus the pathogenesis of craniopharyngiomal cyst appears to be much more akin to those described for cysts accompanying other brain tumours than it was believed earlier [4)].

A prospective study of cystic fluid in craniopharyngiomas in 10 patients was performed to correlate signal intensity on T1-weighted magnetic resonance (MR) images and biochemical analysis. Within 2 days before surgery, each patient underwent MR imaging before and after administration of gadopentetate dimeglumine. Five patients had cystic fluid lower in signal intensity than white matter, with protein levels less than 9,000 mg/dL (90.00 g/L) and no free methemoglobin. One of the five patients had the highest triglyceride concentration (84 mg/dL [0.95 mmol/L]) of all 10 patients; another of these five had the highest cholesterol concentration of all (270 mg/dL [6.98 mmol/L]). It is

concluded that the increased signal intensity of cystic fluid in craniopharyngiomas on T1-weighted MR images can be caused by a protein concentration greater than or equal to 9,000 mg/dL (90.00 g/L), the presence of free methemoglobin, or both. In the ranges of concentrations measured in this study, cholesterol and triglyceride did not increase signal intensity [5].

References

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