Clinicopathological Characteristics and Immunohistochemistry of Intracranial Hemangiopericytoma: Case Report and Literature Review

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Case Report

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Abstract

Background

Hemangiopericytoma was first named as a soft tissue tumor in 1942. It is a group of tumors composed of short spindle cells with pericyte growth. It is rare in clinical practice and accounts for 0.4 of all tumors originating in the central nervous system 0.4%, with a high degree of recurrence and the potential for metastasis outside the central nervous system.

Case presentation

We report 4 patients, including 1 male and 3 females. All patients had headache, dizziness, and fatigue. The first and fourth patients showed acute exacerbation, paroxysmal limb twitching, flexion of both upper limbs, straightening of both lower limbs, unconsciousness, unresponsiveness, and upper eyeballs. Turning and closing of the teeth, 1 out of 2 patients was accompanied by a tongue bite. All 4 patients underwent imaging examinations. Considering the possibility of meningiomas and gliomas, all 4 patients underwent surgical resection and were followed up many times after the operation. The first case was followed up for 68 months and passed away due to recurrence without treatment. The second case was followed up for 50 months and relapsed at 42 months after the operation. It relapsed again 18 months after the operation and passed away with multiple metastases throughout the body; the third patient died in time The 4 patients were followed up for 32 months and 9 months respectively, and there is no recurrence at present.

Conclusions

Meningeal HPC is a rare clinical tumor. 4 cases were followed up for 9-68 months, 2 cases recurred after surgery, and 1 case had multiple metastases throughout the body. It is not easy to distinguish HPC from meningioma on imaging. The diagnosis still depends on pathological examination, combined with the combined diagnosis of STAT6, CD34 and ALDHIA1 immunohistochemical markers, which can effectively improve the diagnosis and differential diagnosis of the disease. Because HPC is prone to recurrence and metastasis, Long-term follow-up and timely follow-up are still needed after operation.

Background

Hemangiopericytoma was first named as a soft tissue tumor in 1942. It is a group of tumors composed of short spindle cells with pericyte growth, and it is speculated that the tumor originated from a smooth muscle-like cell located around the vessel. That is, perivascular cells. Almost all intracranial hemangiopericytoma is attached to the dura mater and is rare in clinical practice. It accounts for 0.4% of all tumors that originate in the central nervous system. It has the potential for high recurrence and metastasis outside the central nervous system [1]. It was suggested that hemangiopericytoma and solitary fibroma belong to the same disease spectrum [2]. Although hemangiopericytoma is a non-meningeal epithelioma, it is highly similar in morphology and histology to meningiomas and synovial sarcoma, and has different clinical pathological malignancies. Its diagnosis depends on a combination of pathological diagnosis and imaging. Here we report 4 cases of hemangiopericytoma originating in the central nervous system, and review the literature to summarize their pathological and imaging characteristics, in order to make a better diagnosis and differential diagnosis.

Case Presentation

Case 1

50 years old, male, right cerebellopontine angle area, complaining of headache, dizziness, and unconsciousness. MRI prompts: Irregular abnormal signals can be seen in the right cerebellopontine angle area-prepontic cistern-cavernous sinus area on both sides, with equal/slightly low signal on T1WI, slightly high signal on T2WI, slightly high signal on T2FLAIR, and obvious enhancement on enhanced scan The size of the lesion is about 4.6cm×2.7cm×5.6cm, and the boundary between the

cavernous sinus segment and basilar artery of the internal carotid artery on both sides is not clear. The brainstem is compressed and shifted to the left. Considering the possibility of meningioma.

Case 2

58 years old, female, left temporal and occipital, headache, dizziness, fatigue, acute exacerbation, paroxysmal limb twitching, flexion of both upper limbs, straightening of both lower limbs, unconsciousness, uncomfortable response, upturned eyes, closed jaw, With tongue bite. MRI showed that the white matter under the frontal cortex on both sides and the left basal ganglia showed spotty T2 high signal and T1 low signal shadow, and no abnormal signal was seen in the brainstem and cerebellum. An irregular shape of T1, T2, and other signals can be seen in the left temporal occipital area. T2FLAIR is slightly higher signal, with a maximum diameter of about 1.8cm. The lesion compresses the adjacent skull and sags outwards. The enhanced scan showed uniform and obvious enhancement.

Case 3

44 years old, female, right tentorium cerebellum, the patient presented with paroxysmal headache, dizziness with nausea and vomiting. MRI prompts: Irregular T2 and T1 signal shadows can be seen beside the transverse sinus on the right side of the posterior fossa. T2FLAIR is equal/slightly high signal, lobulated, with intact capsule, and linear low signal shadows can be seen inside, adjacent the brainstem and cerebellar hemispheres were compressed and shifted to the left, with a size of about 4.6cm×4.2cm×3.2cm. The enhanced scan showed uniform and obvious enhancement, and the lesion was closely attached to the left transverse sinus. There are flaky FLAIR hyperintensity shadows in the white matter of the lateral ventricles on both sides, and the fourth ventricle is compressed and narrowed. Consider the possibility of meningioma.

Case 4

A 19-year-old female, on the right frontotemporal area, complains of headache, dizziness, fatigue, and acute exacerbation. The mRI showed a kind of circular abnormal signal in the right temporal lobe, T1W1 showed equal/slightly low signal, T2W1 showed equal/slightly high signal, T2FLATR showed equal/slightly low signal, the cross-sectional size was about 1.4cm~.3cm, and it was close to the temporal. The leaf is slightly compressed, and the right frontotemporal area is occupied. Consider low-grade glioma.

Results

Imaging findings

Figure 1A The lesion is located in the right cerebellopontine angle area, with slightly high signal on T2FLAIR, and the enhanced scan shows obvious and uniform enhancement, and the dural tail sign is seen near the meninges; Figure 1B The left temporo-occipital lesion showed a slightly high signal on T2FLAIR, and the adjacent skull was compressed and depressed; Figure 1C The tentorium cerebellar lesion on the right is iso-signal on T2WI, with lobes visible, and line-like low signal is seen in it. The brainstem and cerebellar hemisphere is compressed and shifted to the left, and the enhanced scan shows obvious uniform enhancement; Figure 1D the right forehead occupies the T2WI with equal/slightly high signal, and the enhanced scan shows obvious uneven enhancement.

Pathological findings

Under the microscope, the tumor cells are dense, uniform in size, rich in blood vessels, and the cells are arranged in sheets and grow diffusely. The nucleus is round and elliptical; the interstitium is rich in fissure-like blood vessels, and some areas of the blood vessels expand into sinusoids or are compressed into fissure-like shapes, which anastomose each other to form a "stag horn-like"; under high magnification, the nuclear chromatin has a medium density and small nucleoli can be seen (Picture a, e); where anaplastic cells grow actively, mitosis is about 5/10 HPF, and there is hemorrhage.

Immunohistochemical findings

Figure 2a: The cells are dense and uniform in size under low magnification, rich in staghorn and slit-like blood vessels, HE×100; Figure 2b: Tumor cells express STAT6, SP×400; Figure 2c: Tumor cells express FLI, SP×400; Figure 2d: Tumor Cells are negative for GFAP, SP×400; Figure 2e: high-powered nuclei are round, oval, with moderate nuclear chromatin density, mitotic images are visible, HE×400; Figure 2f: tumor cells express CD34, SP×400; Figure 2g: tumor cells desmin Negative, SP×400; Figure 2h: Ki-67 index 20%.

Discussion And Conclusion

Hemangiopericytoma, also known as hemangiopericytoma, is an extremely rare mesenchymal tumor. HPC originating in the central nervous system accounts for about 1% of brain tumors and was first proposed by Stout and Murray in 1942 [3]. In 1995, Joseph et al. [4] confirmed that HPC originated from Zimmerman epithelial cells on capillaries from the molecular and genetic level. It is a kind of contractile spindle cells distributed around the capillaries and venules behind the capillaries, regulating blood flow and permeability, and distributed in any part of the human body [5, 6]. In the 2000 edition of the WHO classification of nervous system tumors, hemangiopericytoma was classified as "mesenchymal tumors, non-meningeal epithelial tumors" in meningeal tumors, and intracranial HPC was classified as anaplastic HPC in the 2007 edition of the WHO classification of central nervous system. (AHPC, WHO level III) and HPC (level II), were recognized as a new entity in the 2016 edition of the WHO classification of the central nervous system. The recurrence rate of HPC and the rate of metastasis outside the nervous system are lower than AHPC [1, 2]. Although the tumor is a non-meningeal epithelial cell tumor, it is highly similar to meningioma and synovial sarcoma in morphology and histology. Because of the non-specific clinical manifestations, most of the imaging examinations are diagnosed as meningioma, so it is easy to be misdiagnosed in the clinic. The diagnosis still depends on the combination of pathological diagnosis and imaging.

HPC can occur in any part of the body, especially the lower limbs, pelvis and retroperitoneum. Intracranial HPC is more common in men. The age of onset is mostly between 38-50 years old. The median age at diagnosis is about 40-50 years old [7, 8]. Its clinical manifestations are non-specific, mainly depending on the location of the tumor, the invasion and compression of the peripheral nerves by the tumor, and can be manifested as headache, dizziness, visual and hearing impairment, numbness and weakness of the limbs and seizures caused by increased intracranial pressure [9, 10]. Intracranial HPC is a tumor with aggressive behavior, including local recurrence and distant metastasis such as liver, bone, lung, kidney, abdominal cavity, lymph nodes, skeletal muscle, pancreas, skin and subcutaneous tissue, etc., and its recurrence within 12 years. The rate is as high as 90% [11]. Of the 4 patients in this article, 2 died of tumor recurrence and metastasis after surgery, and the survival time was 60 months. The clinical follow-up of 2 cases was 5 months and 32 months without special. On the image, intracranial hemangiopericytoma is mostly irregular or lobulated, meningiomas have smooth edges, lobes are rare, and mostly round or elliptical. This irregular shape is mainly due to the rapid growth of the tumor, Caused by violations [12, 13]. On T1WI and T2WI signals and DWI signals, meningioma generally have more uniform signals, while intracranial hemangiopericytoma cysts and necrosis are more common than meningioma, so intracranial hemangiopericytoma T1WI and T2WI signals and DWI signals are usually More mixed; intracranial hemangiopericytoma is more invasive, mostly connected to the meninges, with high signal changes on DWI, meningioma with iso-signal or slightly high signal changes [12]. In addition, hemangiopericytoma usually has Wide base dura mater is attached, and there may be a dural tail sign [8, 14]. A retrospective analysis of 22 cases found that SFT showed high attenuation signal on CT, and showed equal intensity signal and low signal intensity on MR image, as well as obvious enhancement. These features may have certain suggestive significance for SFT [14, 15].

The final diagnosis of intracranial SFT/HPC depends on pathological examination. In general, HPC has a clear boundary, soft texture, brown, dark red, or off-white leaf-like structure, which is wrapped into a leaflet or nodular mass by a covered pseudocapsule with abundant blood supply [16, 17]. Microscopically, the tumor tissue cells are abundant and dense, uniform in size, and composed of short spindle, oval or polygonal cells. The tumor cells are unclear, and the cytoplasm is slightly eosinophilic; the nucleus is oval, short spindle, and chromatin. Medium-density diffuse distribution, tumor cells generally have inconspicuous nucleoli, lack of nuclear inclusion bodies, and can see nuclear atypia and mitotic figures; the stroma is rich in blood vessels, showing fissures, staghorn-shaped blood vessels, lined with flat endothelial cells, tumors Cells surround the

abundant thin-walled blood vessels, the size of the blood vessels varies, and a large number of branches are seen. These branches are attached and wrapped by collagen and run through the cells and the interstitium, showing a stag horn-like structure; some tumors have coagulative necrosis and hemorrhage, Can also infiltrate adjacent brain tissue and skull. One patient showed hemorrhage and necrosis under the microscope, and one case was diagnosed as a recurrence of meningeal hemangiopericytoma (low-grade sarcoma), and the mitosis was about 5/10 HPF where the cells were actively growing. The other two patients were consistent with the literature report under the microscope, the Ki-67 index was relatively low, and there was no recurrence temporarily. In the past, SFT/HPC was considered to be different pathological solid tumors, but the latest research confirmed by immunohistochemistry and gene sequencing that they are a common solid tumor with many similar histological features [18]. Some scholars performed whole-genome sequencing by isolating the DNA of SFTs, and found that the inversion in chromosome 12 caused the NAB2 and STAT6 genes to be juxtaposed, and thus showed that NAB2-STAT6 fusion is a unique molecular feature of SFT [19, 20], related literature It is believed that the presence of NAB2-STAT6 fusion protein was strongly positive in STAT6 immunohistochemistry, and the tissues without NAB2-STAT6 fusion protein showed the nuclear expression of NAB2 and the cytoplasmic expression of STAT6 protein [18]. Therefore, the 2016 WHO classification of the central nervous system proposed that the nuclear expression of STAT6 protein can be used to diagnose HPC/SFT and other mesenchymal tumors [2]. Relevant experiments have shown that the specificity and sensitivity of STAT6 to SFT/HPC are 100% and 96.6%, respectively [21]. Karen et al. [22] used 30 cases of tumors that were initially diagnosed as SFT/HPC meninges and found that all tumors expressed expression NAB2-STAT6 fusion, and it is believed that the fusion of NAB2 exon 4-STAT6 exon 3 is related to the classic SFT morphology and higher age, and shows a trend of decreased mitotic activity. Among the abnormal genes detected, NAB2 exon 6-STAT6 exon 16/17/18 are mostly located in the meninges, soft tissue and SFT of the head and neck, while NAB2 exon 4-STAT6 exon 2/3 It is mostly located in the SFT of the pleura and lung. The recurrence frequency of SFT with NAB2 exon 6-STAT6 exon 16/17/18 is compared with SFT with NAB2 exon 4-STAT6 exon 2/3 The frequency of recurrence is higher [23].

For the treatment of intracranial HPC, it is recognized that the most effective is surgical resection supplemented by radiotherapy. The combination of the two strategies can hinder the progression of the tumor, but has no effect on the median survival rate and the occurrence of metastasis [5, 6], and Total surgical resection has a better therapeutic effect than subtotal resection [7–9]. According to the results of pathological diagnosis, Kim et al. [24] conducted a statistical analysis on the overall survival rate. The results showed that the median time to local recurrence in patients with WHO II HPC and WHO III HPC was 66.2 months and 38.1 months, respectively [9]. Among the 4 patients diagnosed by us, 2 cases had recurrence time consistent with those reported in the literature. In terms of differential diagnosis, it is mainly differentiated from meningioma and synovial sarcoma. First, SFT/HPC has now been confirmed as a new solid tumor, which has a patternless structure with alternating bottom cell and high cell area in the omics. The cell area usually appears as a thicker collagen band, and the high cell area shows the presence of staghorn-shaped blood vessels. Both have similar structure and immunohistological characteristics in omics because they have the same spectrum system. Interestingly, someone compared the ALDHIA1 gene with soft tissue sarcoma and found that ALDHIA1 gene is highly expressed in SFT/HPC, and its sensitivity and specificity in SFT are 84% and 98.8%, respectively, and in HPC it is 84.5% and 98.7%. In meningioma and synovial sarcoma, only 1.2% and 7.1%. In addition, the analysis of the immunohistochemical expression pattern of SFT/HPC and meningioma in the literature shows that the nuclear expression of STAT6 has the highest specificity for SFT/HPC (100%), followed by CD34 (93.6%), and CD34 in meningiomas The positive expression is only 6.4% [25]. If CD34 and ALDHIA1 are diagnosed in combination, the positive predictive value and negative predictive value are both 100%. Therefore, imaging and microscopic performance combined with the current new immunohistochemical markers can distinguish meningiomas from synovial sarcoma.

List Of Abbreviations

HPC: Solitary fibrous tumor/hemangiopericytoma; WHO: World Health Organization; AHPC: anaplastic hemangiopericytoma; SFTs: Solitary fibrous tumors; MRI: Magnetic Resonance Imaging

Declarations

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Not applicable.

Authors' contributions

SK Y, LR D conceived and designed the study, and were the major contributors Including drafted the manuscript and revised it. LR D provided critical contribution to the processing and interpretation of the pathological findings. SJ H, JB L, and SK Y contributed to data acquisition, SK Y, JB L, and YL take responsibility for data analysis, WX is mainly responsible for imaging data diagnosis and image processing of imaging. All authors read and approved the final manuscript.

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Ethics approval and consent to participate

Ethical approval for the study was obtained from the Affiliated Hospital of North China University of Science and Technology after complete description of the study and the expected benefits from the study.

Consent for publication

Written informed consent was obtained from the patient for publication of this Case report and any accompanying images. A copy can be provided at any time.

Competing interests

The authors declare that they have no competing interests.

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Tables

Table 1 Clinical and imaging characteristics of 4 meningeal HPC patients

Case	Sex	Age	Location	Mass	Clinical diagnosis	WHO Grade	Surgical method	Follow-up (months)
1	male	50	Right cerebellopontine angle	Two pieces of gray unplastitized tissue, 2cm×2cm×1cm, the cut surface is gray and white and tough	Postoperative epilepsy after meningioma	Level III	Surgical resection	Passed away in 68 months due to relapse without treatment
2	female	58	Left temporal occipital	A pile of gray and white pieces of tissue, 3cm×2cm×1cm in size, the cut surface is off-white, solid, and medium in texture.	Intracranial space	Level III	Surgical resection	50 months, 42 months after surgery, recurrence, 18 months after surgery again, recurrence again, multiple metastases throughout the body died
3	female	44	Left Tentorium	A pile of unshaped gray-white broken tissues 5.5cm×4.5cm ×1cm.	Intracranial space	Level II	Surgical resection	32 months, no recurrence at present
4	female	19	Right frontotemporal	A piece of gray-yellow unplastitized tissue, the size is 1.5cm×1.5cm×1.5cm, and the cut surface is gray-yellow and grayish-white and slightly tough.	Right temporal space occupying lesion with symptomatic epilepsy	Level II	Surgical resection	September, no recurrence

Table 2 HPC immunohistochemical marker staining results

Case	CFAP	STAT6	FLI	Bcl2	CD34	PR	desmin	Ki67
1	-	+	+	+	+	-	+	10%
2	+	Localized +	+	-	+	-	-	5%
3	-	+	+	Faintly +	+	-	-	20%
4	Individual +	+	+	-	+	-	-	3%

Figures

Figure 1

Clinical imaging data

Figure 2

HE staining and immunohistochemistry

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