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Central neurocytoma presenting with intraventricular hemorrhage : case report and review of literature

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Abstract

We report a case of a 25 year old man presenting with acute headache, vomiting and nuchal rigidity. Computed Tomography (CT) scan and MRI without contrast showed a right ventricular hemorrhage surrounding a mass lesion. The tumor and hematoma were completely removed by neurosurgical transcortical-transventricular approach. Anatomopathological analysis revealed a central neurocytoma. Central neurocytoma seldom present with hemorrhage. We review 16 cases of neurocytoma with hemorrhage. It is important to recognize central neurocytoma as a cause of intraventricular hemorrhage, especially in adolescents and young adults. Outcome is often favorable when the tumor is completely removed. In some patients the clinical course is more aggressive and additional treatment such as radiotherapy, radiosurgery or chemotherapy is needed.

Key words : intraventricular tumor, adolescents, young adults, lateral or third ventricle, apoplexy.

Introduction

Central neurocytoma was first described by Hassoun *et al*. in 1982 (Hassoun *et al.*, 1982) as a well differentiated tumor of neuronal origin. The tumor was classified as grade II according to the WHO classification in 1999. Tumor cells have the ultrastructural features of neurons and are immunoreactive for immunohistochemical markers of neuronal differentiation, in particular synaptophysin. (von Deimling *et al*., 1990 ; Barbarossa *et al*., 1990 ; von Deimling *et al*., 1991 ; Kubota *et al*., 1991). Usually located in the lateral or third ventricles, they represent 0,25-0,50% of all intracranial tumors (Schmidt *et al.*, 2004). The mean age in the series of Hassoun *et al*. (Hassoun et al., 1993) was 29 years. Incidence is similar in both females and males (Hassoun *et al*., 1993). Although several types of neuroepithelial tumors are well recognized to present with intracranial hemorrhage (Wakai *et al*., 1982), central neurocytoma is seldom associated with hemorrhage.

We present a patient with a central neurocytoma that caused an intraventricular hemorrhage and review the literature of 16 similar patients. Central neurocytoma should be considered in the differential diagnosis of intraventricular hemorrhage, especially in adolescents and young adults. It often has a favorable prognosis when completely removed. In some patients the clinical course is less favorable and additional treatment such as radiotherapy, radiosurgery or chemotherapy is needed (Metellus *et al*., 2000 ; Rades *et al*., 2002).

Case report

A 25 year old man was transferred to our clinic, complaining of acute headache and vomiting for three days. Analgesics did not relieve the pain. Clinical examination showed nuchal rigidity, but there were no focal neurological signs. Blood pressure was 150/90 mm Hg. EEG, X-ray of the sinuses and fundoscopy were normal. Blood analysis showed a sedimentation rate of 23 mm/h and a thrombocytopenia of 100000/mm^3, other blood coagulation tests were normal. There was also a chronic hepatitis B infection. CT scan of the brain showed an intraventricular hemorrhage surrounding a small round lesion (Fig. 1). On MRI, performed two days later, there was blood in the lateral ventricles, more prominent on the right side on T1weighted images (T1WI). In the right lateral ventricle a small nodular lesion in relation with the corpus callosum was seen. The mass was isointense to white matter on T1WI and slightly inhomogeneous and hyperintense on T2WI (Fig. 2). The nodule measured 17 mm. There was no enhancement after injection with Gadolinium. No relationship with the circle of Willis and no vascular malformation were seen on MR angiographic sequences. MRI of the spine showed no additional masses on T2WI and T1WI.

The blue-grayish tumor mass was completely removed by neurosurgical transcortical-transventricular approach. On histological analysis the

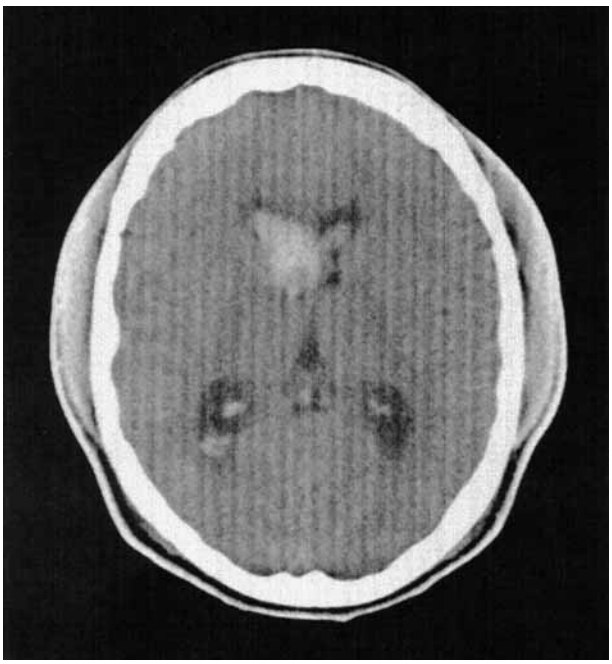

FIG. 1. — (day of admission) Noncontrast axial CT image of the brain revealed a small round lesion within an intraventicular hemorrhage.

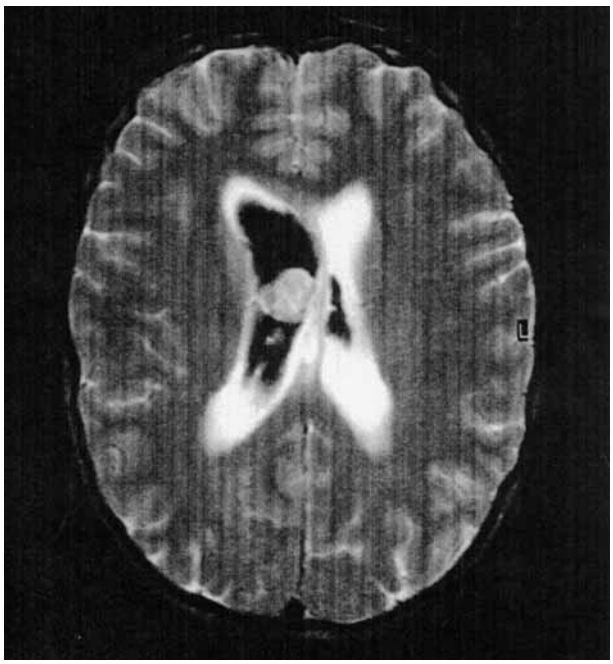

FIG. 2. — (two days after admission) Axial T2-weighted MRI of the brain showed blood in the lateral ventricles, more prominent on the right size and a small nodular lesion in relation with the corpus callosum. The mass was slightly inhomogeneous and hyperintense. There was no enhancement after injection with Gadolinium.

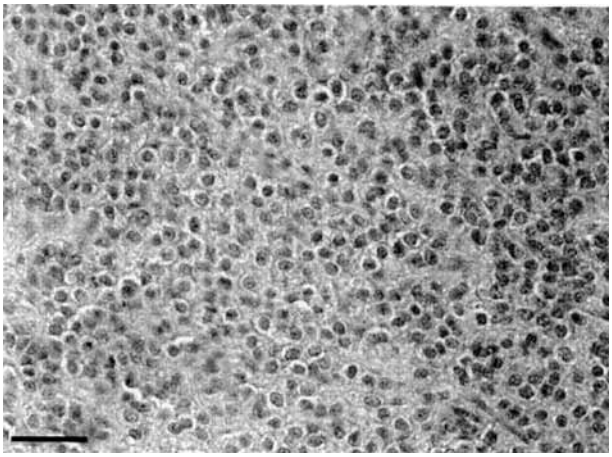

FIG. 3. — Central neurocytoma consisting of small isomorphous tumor cells (hematoxylin eosin stain, magnification : ×200).

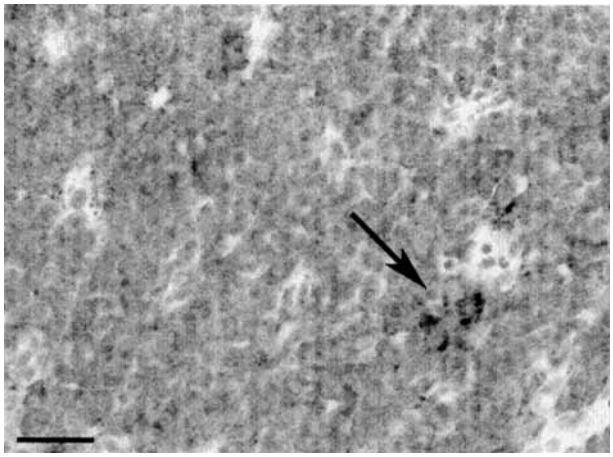

FIG. 4. — Cytoplasmic staining of synaptophysin in tumor cells (magnification : ×200).

lesion had oligodendroglial-like features and was composed of small, round cells exhibiting minimal atypical features (Fig. 3). No areas of necrosis, no vascular proliferation were evident. There were no mitotic figures and labeling with the nuclear marker Ki-67 disclosed few proliferating cells. The growth pattern was rather diffuse, focally exhibiting a nested pattern, surrounded by small capillaries. The cells were isomorphous and had a minimal, mostly clear cytoplasm with a small, round nucleus having speckled chromatin. The tumor cells were immunoreactive for synaptophysin (Fig. 4). There was no labeling for chromogranin. All these features were compatible with the diagnosis of central neurocytoma. After the operation a control CT scan showed a small subdural collection in the right frontal region. The thrombocytopenia remained. Patient was discharged in good health and no additional treatment was needed.

Discussion

CLINICAL PRESENTATION AND MICROSCOPIC FINDINGS

The classical site for a central neurocytoma is within the ventricular system (Hassoun *et al.*,

1982). Extraventricular neurocytomas are definitely rare (Goergen *et al.* 1992). In the reviewed literature of 17 central neurocytomas with hemorrhage, 15 tumors were situated in the ventricles and only 2 were found in the cerebral parenchyma (see table). We describe these 17 tumors, according to their localization and according to their bleeding pattern. The 2 main divisions in this description are : tumors, located in the ventricle and tumors, located in the parenchyma. Those 2 divisions are again subdivided according to the hemorrhage pattern : a purely intratumoral hemorrhage, an intratumoral hemorrhage with breakthrough to the ventricle, an intratumoral hemorrhage with breakthrough to the parenchyma, and both intraparenchymal and intraventricular hemorrhage caused by an aneurysm of a feeding artery of the tumor (see table).

Central neurocytomas are most often found in adolescents and young adults. The reviewed patients have a mean age of presentation of 22 years ; 11 male patients and 4 females patients were found (in 2 cases no information about age and gender was found). In a large series of Hassoun *et al*. (Hassoun *et al.*, 1993) the mean age of presentation was 29 years and the incidence of this tumor was similar in both males and females.

In the literature, most patients with a central neurocytoma, presented with signs of raised intracranial pressure. The 17 presented patients had an acute onset of symptoms, because of the hemorrhage in the tumor (see table, symptoms).

Central neurocytomas comprise 0,25%-0,50% of brain tumors (Schmidt *et al*., 2004). Since Hassoun's original description in 1982, more than 500 cases have been reported (Rades *et al.*, 2002). Their true incidence may be higher, because many central neurocytomas were previously described as oligodendrogliomas (Agranovich *et al*., 1993 ; Yamshidi *et al.*, 2001). The light microscopic appearances of central neurocytomas and oligodendrogliomas are similar, but the tumor cells of central neurocytomas have ultrastructural features of neurons. Also immunohistochemical markers of neuronal differentiation, like synaptophysin or neuron specific enolase are used to differentiate them from oligodendrogliomas (von Deimling *et al*., 1990 ; Barbarossa *et al*., 1990 ; von Deimling *et al*., 1991).

Central neurocytomas are not known as tumors prone to hemorrhage, in contrast with oligodendrogliomas, which have a high tendency to bleed (Russell and Rubinstein, 1977 ; Okamura *et al*., 1995). Thus, it is possible that the incidence of central neurocytomas with hemorrhage has been underestimated because some were previously diagnosed as oligodendrogliomas. Our own case was immediately diagnosed as central neurocytoma. In the reviewed literature, 2 cases were first misdiagnosed as oligodendroglioma (see table, case 1, case14). Case 16 showed features of a central neurocytoma combined with neoplastic ganglion and glial cells and was called 'gangliogliocytoma', because of the presence of these ganglion and glial cells (Taylor *et al*., 1998, Tortori-Donati *et al*., 1999). One could ask if this tumor is not merely another type and that we should separate this tumor from the other 16 cases we have found.

Central neurocytomas usually exhibit rare or no mitotic activity, no apoptosis, no necrosis, nor vascular endothelial proliferation. Atypical forms have been reported (Mc Cutchen *et al*., 1999 ; Söylemezoglu *et al*., 1997 ; Elek *et al*., 1999). Although anaplasia has been demonstrated in central neurocytoma, the influence of this feature on prognosis remains uncertain. Elevated MIB-1 monoclonal antibody index might be more useful in predicting relapse (von Deimling *et al*., 1990 ; von Deimling *et al*. 1991). The presented case report showed no anaplasia and no elevated proliferative potential was seen. In the reviewed cases, only case 17 showed anaplastic features. The tumor had numerous mitoses and frequent apoptosis, giving it a more aggressive appearance than the classical central neurocytoma (Mc Cutchen *et al*., 1999).

Medical imaging

Central neurocytomas have heterogeneous appearances on CT or MRI (Chang *et al*., 1993) because of the variable components, like cysts, calcifications and vascular structures and also the radiological findings of hemorrhages may change in time. So, it is not always easy to differentiate a tumor hemorrhage from other etiologies. Careful attention to small lesions in the hemorrhage zone is needed. If a CT scan on admission only reveals a large amount of blood in the ventricles, further investigations, like MRI imaging, MR angiography and angiography are certainly needed and imaging should be repeated in order to detect the cause of the hemorrhage. In our own case the diagnosis of a tumor as cause of the hemorrhage was quite easily made, because a small round lesion was noticed on the CT scan on admission. In case 12 and 15 the etiology of the hemorrhage was not immediately found, but detailed review of the previously performed CT scans on admission also showed tumor masses within the hemorrhages.

Angiographic findings in central neurocytoma have been reported and lesions may be vascular or avascular (Yasargil *et al*., 1992 ; Smoker *et al*., 1991 ; Namiki *et al*., 1998 ; Peak *et al*., 2003). Angiographic findings can be very useful in excluding aneurysms or vascular malformations as the cause of the hemorrhages or they can help locating a tumor. In our case no aneurysm nor vascular malformation was seen. In case 10 and

case 13 angiographic findings showed an avascular mass effect, bowing vessels laterally and this suggested the presence of a tumor. In case 15, a cerebral angiogram revealed a small fusiform aneurysm on a lenticulostriate artery, which was the cause of the hemorrhage.

Hemorrhages truly originating within the tumors

Intraventricular hemorrhages can be caused by small parenchymatous hypertensive bleeds originating in the tissues close to the ventricular system. Some of these hemorrhages go undetected by CT. Some very small hemorrhages arise in the plexus choroideus (Graeb *et al*., 1982 ; Caplan *et al*., 1994 ; Marti-Fabregas *et al*., 1999). Intraventricular hemorrhages can also be caused by vascular malformations adjacent to the ependymal lining or by very small malformations that self-destruct as a result of the hemorrhage or which do not opacify in the angiography. Those malformations are often found at autopsy (Graeb *et al*., 1982 ; Caplan *et al*., 1994). In our case and almost all the other presented cases, anatomopathological evidence for tumor hemorrhage was found. Microscopically, hemosiderin deposits have been found in the tumors and macroscopically, hemorrhagic zones were also found in some tumor biopsies (see table : anatomopathological evidence). Radiological findings also suggested that the hemorrhages arose in the tumors, because large hemorrhagic zones or hemosiderin deposits were seen in the tumors on MRI or CT images. Thus, we believe that the bleedings arose within the tumors. Case 15 is the only exception, this case shows an aneurysm on a feeding artery of the tumor.

Causative factors for tumor hemorrhage

Peak *et al*. (Peak *et al*., 2003) found that the degree of tumor staining on angiography seemed to be related to the size of the tumor. One could conclude that larger central neurocytomas are more prone to cause hemorrhage. In these vascularized tumors, abnormally developed and friable blood vessels can cause the hemorrhage. In the reviewed literature, 4 tumors (cases 1, 3, 6 and 9) were very large and they also had a marked vascularity on anatomopathological examination. However, not all large central neurocytomas are well vascularized and even avascular large tumor masses have been described (Bolen *et al*., 1989). In our own case, the tumor was small, but the patient showed a thrombocytopenia that could have contributed to the bleeding.

Rupture of arteries, invaded by neoplasms, has also been reported as cause of tumor hemorrhage (Cowen *et al*., 1970 ; Hart *et al*., 1974). This mechanism of hemorrhage, was not found in the 12 described patients, where angiography was performed. Also rapid tumor growth is often a cause of spontaneous bleeding in malignant tumors, but central neurocytomas are considered benign. All the reviewed cases, with exception of case 17, did not show malignant features, so this could not have caused the hemorrhages. In Case 17, tumor growth could have caused the vessels to rupture, although no sequential CT images, that would have confirmed rapid tumor growth and no MIB-proliferation index were performed. In case 3, the authors (Balko *et al*., 1999) suggested that the remarkable cardiomegaly, attributed to hypertensive heart disease, predisposed the tumor to the possibility of acute hemorrhage and in case 14, the authors (Namiki *et al*., 1998) suggested that abnormal tissue vascularisation after radiotherapy could have caused the bleeding.

Conclusion

When a young patient presents with an intraventricular bleeding, repeated imaging and MR imaging is of great importance to elucidate the underlying cause, such as a tumor. The importance of accurate diagnosis of a central neurocytoma lies in the relatively good prognosis of this tumor after maximal surgical removal.

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Table

Characteristics in 17 patients with hemorrhagic central neurocytomas (AP anatomopathological, AVM arteriovenous malformation, CE contrast enhancement, CN central neurocytoma, CT computer tomography, d days/y years/m months, GTR gross total resection, Ma macroscopical findings, Mi microscopical findings MRI magnetic resonance imaging, OG oligodendroglioma, RT radiotherapy, STR subtotal resection, T1WI T1 weighted images, T2WI T2 weighted images, ? no clear information in article)

Localization/ hemorrhage pattern	N°	Author	Age/ sex	First clinical presentation	CT	MRI	Angiographic findings vascularisation	AP evidence of hemorrhage/	First AP diagnosis	Treatment
Tumor located in the ventricle										
Purely intratumoral hemorrhage (6)	1	Kim, 1992	25y/M	Signs of raised intracranial pressure hemorrhage, CE	Isodense mass+intratumoral	Not performed	Vascular staining Mi: high vascularity	Ma: hemorrhage in tumor	1. OG 2. CN	STR/RT
	2	Metellus, 2001	38y/M	Sudden headache, vomiting, left hemiparesis	Intraventricular hemorrhage, no blood at bottom of trigonum	T1W1: isointense mass CE T2W2: hyperintense mass	No AVM, no aneurysms	/	CN	GTR
	3	Balko, 1999	51y/M	Found dead at home	/	/	/ Ma:mass, friable, areas of hemorrhage, hemosiderin deposits Mi:acute hemorrhage, numerous capillaries	Autopsy:	CN	no
	4	Chang, 1993	?	?	?	High intensity areas on all pulse sequences = intratumoral hemorrhage	?	Not performed	CN	/
	5	Chang, 1993	?	?	?	High intensity areas on all pulse sequences = i ntratumoral hemorrhage	?	Not performed	CN	/
	6	Kubota, 1991	25y/M	/	/	/	/	Autopsy: Ma: giant hemorrhagic mass Mi: multiple blood vessels	CN	Dead after STR
Intratumoral hemorrhage with breakthrough to the ventricles (7)	7	Smets, 2005	25y/M	Sudden headache, nuchal rigidity, vomiting	Intraventricular hemorrhage, round lesion	T1W1: isointense mass T2W2: heterogeneous mass No CE	No AVM, no aneurysms	Small capillaries	CN	GTR

Localization/ hemorrhage pattern	N°	Author	Age/ sex	First clinical presentation	CT	MRI	Angiographic findings vascularisation	AP evidence of hemorrhage/	First AP diagnosis	Treatment
	8	Hanel, 2001	35y/F	Sudden headache, nuchal rigidity, vomiting	Lesion with intraventricular hemorrhage	T1W1:heterogeneous lesion+hematoma at bottom of trigonum T2W2: hypointense regions = hemorrhage, CE	No AVM	No information on hemosiderin or vascularisation	CN	Ventricu-lar drain, GTR, drainage abscess and antibiotics
	9	Yamshidi, 1998	22y/F	Acute headache, vomiting	Huge mass with heterogeneous density, small hematoma at bottom of left trigone	T1W1: isointense mass T2W2: hyperintense mass with low intensity area = hemorrhage, no CE	Slight blush in posterior choroidal artery	Some blood clots within the tumor	CN	Two surgical stages: GTR
	10	Smoker, 1991	26y/M	Tonic clonic seizure	Intraventricular and subarachnoidal hemorrhage 3m later: mass	5d later: intraventricular hemorrhage 5m later: T1W1: hyperintense, heterogeneous mass T2W2: hyperintense mass with hypointense areas = hemorrhage No CE	Avascular mass effect displacing other vessels laterally	Mi: hemosiderin deposits in tumor biopsy	1) trom-bosed AV malfor-mation 2) CN	Ventriculo-stomy, GTR
	11	Goergen, 1992	18y/F	Sudden headache	Mass, hemorrhage within the tumor, hematoma at the bottom of the left trigone, no CE	Not performed on hemosiderin or vascularisation	Not performed	No information	CN ventri-cular	GTR, shunt
	12	Okamura 1995	23y/M	1) unconsciousness 2) 2y later: sudden headache	1) intraventricular hemorrhage ➛review: mass lesion 2) non calcified mass	2) T1WI: isointense mass T2W2: heterogeneous mass, increased signal intensity, CE	No AVM, no aneurysms	Ma:soft hemorrhagic tumor Mi: hemosiderin deposits, numerous capillaries	1) intra-ventricu-lar hematoma 2) CN	1) Ventricular drainage, 2) GTR in two surgical stages
	13	Agranovich, 1993	28y/M	1) Sudden headache, vomiting 2) 2m later: headache 3) severe headache	1) intraventricular hemorrhage 2) mass, CE	3) multilobulated mass, rapid T2 relaxation = hemosiderin within the mass	Displacement of peri-calossal arteries, no AVM, no aneurysms	No information on hemosiderin or vascularisation	1) intra-ventricu-lar hema-toma 3) CN	1) refused operation 3) RT
Intratumoral hemorrhage with breakthrough to the parenchyma (1)	14	Namiki, 1998	50y/M	1) headache, gait disturbances 2) 15y later: right hemiparesis, speech disturbances	1) isodense mass, CE 2) intratumoral and intracerebral hemorrhage	2) T1W1: isointense mass T2W2: heterogeneous mass, CE, high intensity areas on T1 an T2 = hemorrhage	1) Mild staining 2) faint vascular staining	Ma: intracere-bral hematoma, dark red tumor, highly vascular Mi: prominent small vessels, hemosiderin	1) OG 2) CN	1) STR + RT + Ventriculo-peritoneal shunt 2) hematoma removal, tumor biopsy

Localization/ hemorrhage pattern	N°	Author	Age/ sex	First clinical presentation	CT	MRI	Angiographic findings vascularisation	AP evidence of hemorrhage/	First AP diagnosis	Treatment
Intraparen-chymal and intraventricular hemorrhage caused by an aneurysm of a feeding artery (1)	15	Vates, 2001	35y/M	1) left hemiparesis after sexual intercourse 2) left sided weakness	1) intraventricular hemorrhage with casting of the right ventricle (review➛mass lesion)	2) T1W1: heterogeneous mass, CE and large intraparenchymal clot in putamen, extending into ventricle	Fusiform aneurysm on lenticulo-striate artery, abnormal tumor blush	Ma: not particularly vascular tumor	CN	1) ventricular drainage 2) GTR + hematoma removal+ dissection of aneurysm
Tumor located in the parenchyma										
Purely intratumoral hemorrhage (1)	16	Taylor, 1998	17y/M	Acute headache, vomiting, motor disturbances in right hand	Heterogeneous left parietal mass + central hematoma	Left hemispheric mass CE superior and anterior to the hematoma	No tumor blush, no AVM, no aneurysms	Ma: hypervascular tumor Mi: highly vascular stroma	Ganglio-glio-Neuro-cytoma	Left parietal craniotomy + GTR
Intratumoral hemorrhage with breakthrough to the parenchyma (1)	17	Mc Cutchen, 1999	48y/F	1) past medical history: right hemiparisis 2) right hemiplegia, dysarthria, acute headache	2) hematoma, filled with fluid levels left frontoparietal, mass, CE	1) T1WI: heterogeneous mass with high signal intensity = methemo-globin T2WI: mixed signal in mass, CE in superficial non hemorrhage portion of mass	Not performed	Ma: very hemorrhagic tumor, Mi: liquefied clot, many mitoses and apoptosis	CN	1) Anti-epileptic 2) STR, clot resection, RT