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Intraventricular Cerebrovascular Pathologies of Hydrocephalus and Managements

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1. Introduction

Hydrocephalus, "water in the brain", had been amazed and challenged clinicians throughout the history of medicine till Galen. Vascular causes of hydrocephalus had been a mystery until the discovery of modern neuroradiological techniques such as digital subtraction angiography and magnetic resonance imaging.

Vascular lesions of the ventricular system are rare. Despite the rarity, they may cause symptomatic hydrocephalus. These kinds of lesions cause hydrocephalus in the CSF pathways by either obstruction or hemorrhage. Because of the rarity of this entity, there is no conciliation for the treatment. Four types of intraventricular vascular lesions causing hydrocephalus were categorized as follows:

1. Cavernomas
2. Aneurysms
3. Arteriovenous malformations
4. Venous malformations

2. Intraventricular Cavernomas (IVCs)

Cavernous malformations (CM), also called cavernous angiomas and cavernomas, are low-flow vascular malformations that constitute 5-13% of all central nervous system vascular malformations (Moriarity et al., 1999; Raychaudhuri et al., 2005). On the other hand, IVC are rare pathological entities, constituting 2,5-10.8% of cerebral CM (Kivelev et al., 2010). The first report of an IVC was published in 1905 by Finkelnburg. As to our knowledge, so far 102 IVCs have been summarized published cases in the literature (Table-1).

2.1 Embryogenesis

The exact pathogenesis of the CM is still unknown; but they are thought to arise from early stages of embryogenesis and may be due to aberrant vasculogenesis (Sure et al., 2005). The CMs are known to exhibit an unpredictable dynamic behavior and may increase in size

(Moriarity et al., 1999). The growth most likely occurs by a process of cavern proliferation in the setting of repetitive hemorrhages (Shenkar et al., 2007).

2.2 Pathology

CMs are histologically benign, hamartomatous vascular malformations, consisting of lobulated sinusoidal vascular channels which are lined with thin endotelia. CMs are classified along with capillary telangiectasia, venous angiomas and arteriovenous malformations (AVM) as vascular malformations (Raychaudhuri et al., 2005). CMs are typically lacking interventing neural parenchyma, large feeding arteries or large draining veins; but may have surrounding gliosis. Hemorrhages at all stages of evolution are present within the lesion and cause occlusion and thrombosis of the vascular channels. Organization of the hematoma results in hyaline-degenerative changes, chronic granulation and scar formation; and includes pseudotumorous evolution of the mass (Voigt & Yaşargil, 1976). Further bleeding may occur in the immediate vicinity of CM leading to hemosiderin deposits and gliosis (Chen et al., 2006).

2.3 Location and symptoms of the intraventricular Cavernomas (IVCs)

The most frequent symptoms for all intracranial CM are seizures (60%), progressive neurological deficits (50%) and hemorrhages (20%) (Coin et al, 1977). IVCs are more likely to present with increased intracranial pressure.

The most common location of the IVC is lateral ventricles followed by the third and the fourth ventricles. CM can be asymptomatic; when symptoms are present they depend on the size and the location of the lesion.

Third ventricular CMs have different symptomatology due to its location. The most common presentation of third ventricle CM is hydrocephalus followed by hemorrhage. Some patients presented with memory loss, diabetes insipidus, seizures, visual field deficits and intermittent postural headaches (Fagundes-Pereyra et al., 2000; Katayama et al., 1994; Mizutani et al., 1981; Milenkovic, 2005; Reyns et al., 1999).

On the other hand, symptoms of IVCs are most likely to present late, since the ventricular cavity allows for tumor growth to large sizes (Kumar et al., 2006). This could be explained by the fact that the surrounding cerebrospinal fluid (CSF) allows the increase in size of the lesion without restrictions from parenchyma. Surprisingly, in spite of being intraventricular lesions, hydrocephalus is seldom reported unlike choroids plexus papillomas in the same location (Nieto et al., 2003). For CM of the third and the fourth ventricles, the presence of acute obstructive hydrocephalus is anticipated and can easily explain the patients' symptoms of intracranial hypertension. For CM of the lateral ventricles intracranial hypertension is not so readily explained. Although CSF outflow obstruction from hemorrhagic elements present within the ventricle or on the arachnoid villi from previous microhemorrhages cannot be excluded as the reason for the raised intracranial pressure. A focal non-communicating hydrocephalus due to entrapment of a horn (most commonly temporal horn) may be the cause (Stavrinou et al., 2009). The temporal horn contains the choroids plexus where CSF is produced continuously, so focal obstructive hydrocephalus will result from the CSF production-absorption imbalance. Moreover temporal horn dilatation and the subsequent stretching of the ventricular wall vessels results in disturbance

of the venous blood flow and contributes significantly to the development of periventricular edema and intracranial hypertension (Tsugane et al., 1992).

Authors & Year	Age (y),Sex	Presentation	Hydrocephalus	Location	Treatment	Outcome
Finkelburg, 1905	2, M	mass effect	NR	4V	PR	died
Dandy, 1928	31, M	mass effect	yes	4V	TR	improved
Meritt, 1940	16, F	mass effect	none	LV	TR	comatose
Arnstein et al., 1951	2 days, M	mass effect	none	LV	no op	died
Latterman, 1952	68, F	mass effect	NR	3V	no op	died
McGuire et al., 1954	3 mos, M	mass effect	yes	LV	NR	NR
Schneider & Liss, 1958	33, F	mass effect	NR	LV	TR	HH
Jain, 1966	15, M	mass effect	yes	LV	TR	improved
McConnel & Leonard, 1967	31, F	IVH	none	LV	no op	death
Coin et al., 1977	36, F	seizure	none	LV	TR	hemianopia
Numaguchi et al., 1977	43, M	mass effect	none	LV	TR	hemiplegia & hemianopia
Giombini & Morello, 1978	27, M	mass effect	yes	4V	PR	died
Terao et al., 1979	29, F	IVH	yes	4V	TR	improved
Pau & Orunesu, 1979	56, NA	IVH	NR	LV	no op	died
Namba et al., 1979	45, F	IVH	NR	LV	PR	improved
Vaquero et al., 1980	18, F	mass effect	none	3V	TR	improved
Britt et al., 1980	11, F	mass effect	none	LV	TR	improved
Pozzati et al., 1981	31, F	mass effect	yes	3V	TR	improved
Iwasa et al., 1983	8 days, F	mass effect	yes	LV	TR	improved
Kendall et al., 1983	60, F	mass effect	yes	4V	PR	symptom recurrence
Lavyne & Patterson, 1983	48,F	mass effect	yes	3V	PR	hydrocephalus, bleeding
Amagasa et al., 1984	40, M	mass effect	none	3V	TR	improved
Harbaugh et al., 1984	44, F	IVH	yes	3V	TR	improved
Chadduck et al. 1985	21, F	seizure	none	LV	TR	hemianopia
	29, F	mass effect	none	LV	TR	improved
	4 mos, F	seizure	none	LV	TR	improved
Simard et al., 1986	22, M	mass effect	NR	LV	NR	NR
	13, F	mass effect	NR	LV	NR	NR
Yamasaki et al., 1986	73, M	mass effect	NR	LV	TR	improved
	9, M	mass effect	NR	3V	PR	improved
	36, M	mass effect	yes	3V	TR	improved
	47, M	mass effect	NR	4V	TR	improved
	15, F	mass effect	yes	3V	NR	NR

Authors & Year	Age (y),Sex	Presentation	Hydrocephalus	Location	Treatment	Outcome
Suzuki, 1988	40, M	mass effect	none	LV	TR	improved
Sabatier et al., 1989	9 mos, M	IVH	NR	LV	no op	cerebellar dysfunction
Voci et al., 1989	19, F	IVH	NR	3V	TR	improved
Ogawa et al., 1990	16, M	mass effect	yes	3V	TR, shunt	improved
	40, M	mass effect	none	3V	TR	transient DI, HH
Andoh et al., 1990	62, F	mass effect	none	LV	TR	HH
Tatagiba et al., 1991	33, M	IVH	none	LV	TR	improved
	35, M	seizure	none	LV	TR	died
	24, F	mass effect	yes	LV	TR	improved
Itoh & Usui, 1991	44, F	IVH	yes	4V	TR	improved
Miyagi et al., 1993	3, F	IVH	none	LV	TR	mild hemiparesis
Lynch et al., 1994	39, F	seizure	none	LV	TR	improved
	5, M	seizure	none	LV	TR	improved
	10, F	mass effect	none	LV	TR	improved
Katayama et al., 1994	9, F	seizure	yes	3V	PR, shunt	died
	50, F	mass effect	yes	3V	NR	NR
	45, F	IVH	NR	3V	NR	NR
	49, M	mass effect	NR	3V	NR	NR
	47, F	mass effect	NR	3V	TR	transient DI
Sinson et al., 1995	43, F	mass effect	yes	3V	TR	died
	36, F	mass effect	yes	3V	TR	hemiparesis, hydrocephalus
	52, F	mass effect	yes	3V	TR	improved
	32, F	mass effect	yes	3V	TR, shunt	improved
Hashimoto et al., 1997	2 days, M	mass effect	yes	LV	TR, shunt	mild MR
Kaim et al., 1997	64, M	mass effect	yes	3V	TR	NR
Gaab & Shroeder, 1999	44, F	mass effect	yes	LV	TR	permanent memory loss
Reyns et al., 1999	16, F	mass effect	none	LV	TR	improved
	36, M	seizure	none	LV	TR	hemihypertonia
	42, M	asymptomatic	none	3V	PR	improved
Fagundes-Pereyra et al., 2000	15, F	mass effect	none	LV	TR	improved
Attar et al., 2001	30, M	mass effect	none	LV	NR	improved
	30, M	mass effect	none	LV	NR	improved
	18, M	mass effect	none	LV	NR	improved
	30, M	mass effect	none	LV	NR	improved
Suess et al., 2002	36, F	mass effect	yes	3V	TR	improved
Crivelli et al., 2002	38, M	mass effect	yes	3V	TR	improved

Authors & Year	Age (y),Sex	Presentation	Hydrocephalus	Location	Treatment	Outcome
Nieto et al., 2003	11, F	seizure	none	LV	TR	HH
Tatsui et al., 2003	17, F	seizure	none	LV	TR	improved
	52, M	IVH	none	LV	TR	improved
Wang et al., 2003	62, F	mass effect	yes	3V	TR	improved
Anderson et al., 2003	45, F	mass effect	none	LV	TR	improved
Michaelson et al., 2004	22, F	mass effect	none	LV	TR	improved
Darwish et al., 2005	47, F	asymptomatic	none	3V	TR, shunt	improved
Milenkovic et al., 2005	56, M	mass effect	yes	3V	TR	improved
Chen et al., 2006	51, F	mass effect	yes	3V	TR	improved
Kumar et al., 2006	8, M	mass effect	none	LV	TR	improved
	19, F	seizure	none	LV	TR	seizure remained
	20, M	mass effect	none	LV	TR	improved
Longatti et al., 2006	35, M	mass effect	yes	3V	TR	improved
Zakaria et al., 2006	8, M	mass effect	yes	3V	TR	improved
Sato et al., 2006	47, F	mass effect	yes	3V	TR	improved
Gonzalez-Darder et al., 2007	25, M	mass effect	none	LV	TR	improved
Prat & Galeano, 2008	56, NA	mass effect	yes	3V	TR	improved
Stravrinou et al., 2009	52, F	mass effect	yes	LV	TR	improved
Carrasco et al., 2009	60, F	mass effect	none	LV	TR	hemiparesis
	70, M	mass effect	yes	LV	PR, shunt	improved
	66, M	seizure	none	LV	TR	seizure remained
Kivelev et al., 2010	66, M	mass effect	yes	LV	shunt only	improved
	43, F	mass effect	none	4V	TR	improved
	65, M	IVH	yes	LV	TR	improved
	58, F	IVH	none	4V	TR	mild deficit
	20, M	IVH	none	LV	PR	improved
	15, M	IVH	none	4V	TR	mild deficit
	52, M	mass effect	none	3V	TR	improved
	49, F	mass effect	none	4V	TR	mild deficit
	35, M	IVH	none	LV	no op	improved
	49, F	IVH	yes	4V	TR	improved
	65, M	IVH	yes	LV	no op	improved
	53, M	IVH	none	LV	TR	improved

IVH=intraventicular hemorrhage, LV=lateral ventricle, 3V=third ventricle, 4V=fourth ventricle, PR=partial resection, TR=total resection, NR=not registered, HH=homonymous hemianopia, MR=mental retardation, DI=diabetes insipidus

Table 1. Presented cases of IVC in the literature.

2.4 Natural History of the IVCs

According to the review of the literature, the natural history of IVCs may be different from intraparenchymatous lesions. In IVCs, most common symptoms occurred due to mass effect (65%) followed by hemorrhage (20%) and seizure (15%). In children, clinical presentation does not differ significantly from adults. The annual risk of hemorrhage from supratentorial CM is about 0.25-0.7% (Moriarity et al., 1999; Raychaudhuri et al., 2005). The natural history of the IVC cannot be determined due to small number of cases.

2.5 Diagnosis

2.5.1 CT scan

Typical computed tomography (CT) findings associated with CM consist of a well circumscribed high density nodular lesion with minimal or no mass effect, absence of perifocal edema and mild or no contrast enhancement (Chen et al., 2006; Iwasa et al., 1983; Stavrinou et al., 2009). Sometimes calcification of the lesion and intraventricular bleeding may be demonstrated (Tatagiba et al., 1991). Calcifications may appear on conventional x-rays. Several authors have described atypical images, such as hypodense areas within the lesion caused by cystic components (Khosla et al., 1984; Ogawa et al., 1990; Ramina et al., 1980). Nonetheless, these CT findings can be mimicked by AVM, venous angioma, low grade glioma, craniopharyngioma, meningioma, teratoma, neurocytoma, ischemia enhancing infarct and inflammatory lesions (Chadduck et al., 1985).

2.5.2 MRI

The major diagnostic tool of choice in the detection of the IVC is magnetic resonance imaging (MRI). MRI is both highly sensitive and specific. The introduction of MRI has led to diagnosis of an increasing number of CM that had been clinically silent, angiographically occult and undetected by CT. The common MRI features include a heterogeneous core with multiple foci of high signal on short and long TR/TE images, which correspond to hemorrhages of different ages. Interspersed fibrosis shows low signal intensity. The lesions are well delineated by a pseudocapsulate and typically show a low signal hemosiderin rim on T2-weighed images (T2WI). Edema surrounding the CM is unusual and, if present, always mild. Contrast enhancement ranges from strong to moderate or none (Gomori et al., 1986; Kaim et al., 1997; Lemme-Plaghos et al., 1986; Sigal et al., 1990). The radiological appearance of IVC differs from the parenchymatous CM. The typical hypointese perilesional rim on T2WI is absent, probably because no gliotic reaction towards the hemosiderin is established. Another differential aspect is the intense gadolinium enhancement similar to that of neoplastic lesions (Nieto et al., 2003).

2.5.3 DSA

Since CMs are lack of well-formed vessels supplying or draining them, they are often angiographically occult (Simard et al., 1986). Despite CMs have been classically considered as angiographically occult or cryptic vascular malformations, a tumoral blush (supplied by an enlarged choroidal artery) or a feeding artery can be identified on cerebral angiography (Chadduck et al., 1985). Numaguchi et al., have described the presence of tiny strands of the

contrast medium in the avascular mass in the capillary and venous phase, without large draining veins or early venous filling being observed. But digital subtraction angiography (DSA) is also indicated to exclude AVM.

2.5.4 Differential diagnosis

The differential diagnosis on MRI includes primary and secondary hemorrhagic neoplasms that can be seen at the ventricle. Anaplastic astrocytomas, glioblastomas and oligodendrogliomas are usually heterogeneous because of intratumoral necrosis, hemorrhage or calcification and may mimic CM. However anaplastik tumor tissue showing nonhemorrhagic, abnormal signal intensity with contrast enhancement and surrounded by prominent high signal edema on long TR/TE images should permit distinction from CM (Sze et al., 1987). In young adults rare but important differential diagnosis include central neurocytoma and subependymal giant cell astrocytoma (Kaim et al., 1997). One should also consider cystic and hemorrhagic metastases which may occur together with metastatic melanoma, adenocarcinoma or bronchogenic carcinoma (Atlas et al., 1987). Most of these malignancies, however have multiple lesions and present with known systemic metastases. Furthermore, ventricles are very uncommon site for solitary metastasis. Colloid cysts and germinomas may occur at foramen of Monro, but can be excluded by their different appearance on CT and MRI (Kaim et al., 1997). IVCs are frequently misdiagnosed as tumors and this can lead the use of invasive diagnostic procedures such as steriotactic biopsy which can cause iatrogenic bleeding (Carrasco et al., 2009). On the other hand, several authors have reported that steriotactic biopsies have been performed safely in patients with CM despite the apparent danger of hemorrhage (Sedan et al., 1989).

2.6 Managements

Still, little is known about natural history of the IVC. The lack of through prospective series and long-term follow-up make decision making the treatment of the IVC difficult. Surgery is advocated when rebleedings are frequent and the mass effect causes hydrocephalus and progressive neurological deficits.

2.6.1 Conservative treatment

A conservative approach to an asymptomatic supratentorial CM is appropriate. However, the tendency for rapid growth and extralesional hemorrhage of IVC may suggest the need to treat these lesions more aggressively (Katayama et al., 1994; Reyns et al., 1999; Sinson et al., 1995). In addition, the radiological diagnosis of IVC may be difficult as these lesions may mimic neoplasms. Incorrect preoperative diagnosis has sometimes resulted in inappropriate treatment, such as radiotherapy (Reyns et al., 1999).

2.6.2 Surgical treatment

The management of hydrocephalus associated with IVC has not been well established. An early resection of the mass might solve the CSF obstruction. In fact, the presence of ventricular dilatation may help during surgery. However insertion of a ventriculoperitoneal shunt or external ventricular drainage before removing the lesion represents a safe choice, because it allows an early relief of the symptoms of high intracranial pressure while

studying the mass. The avascular nature of the CM minimizes the risk of shunt device obstruction caused by intraoperative bleeding during lesion removal (Carrasco et al., 2009). On the other hand, shunting CSF may contribute to lesion's rapid growth by altering the hydrodynamic equilibrium between malformation and the ventricular system (Sinson et al., 1995).

The preferable routes for the resection of CM located within the frontal horn are either the transcortical, transventricular or the interhemispheric transcallosal approaches. Transtemporal and superior parietal approaches have been used for the excision of trigonal and temporal horn lesions. The transsylvian transventricular approach is a good alternative for the resection of trigonal lesions with the benefit of a minimal disruption of the visual pathways (Carrasco et al., 2009).

Surgical approaches used to reach foramen of Monro and the third ventricle are transcallosal, transfrontal transventricular and translamina terminalis approaches.

Surgery for an IVC in the lateral or third ventricles is safer than in the fourth ventricle. Patients with CM close to the brainstem frequently present preoperatively with cranial nerve deficits as a sign of brainstem damage. Thus surgery in this already affected region can worsen neurological status and cause new deficits; even after minimal manipulation (Kivelev et al., 2010).

Endoscopic ventriculoscopy may be very useful in establishing the diagnosis or narrowing the differential diagnosis. Despite the increasing role of neuroendoscopy in the treatment of intraventricular lesions, in the cause of IVC the use of endoscopy has been used to confirm the diagnosis under direct vision of the lesion (Sato et al., 2006). Complete endoscopic resection of an IVC has been reported to be performed successfully only in two cases (Gaab & Schroeder, 1999; Prat & Galeano, 2008).

3. Intraventricular Aneurysms (IVAs)

Intraventricular localization of an aneurysm is a very rare entity. To our knowledge, 59 cases were presented in the literature (Table-2).These aneurysms are either true intraventricular or its dome extending into the ventricle cavity(Sanli et al.,2011)For the former aneurysms, most common location is lateral ventricle followed by the third ventricle. Only few cases were located in the fourth ventricle. Most aneurysms in the lateral ventricle are originated from anterior choroidal artery. More specifically, aneurysms in the third ventricle arise from a major branch of the circle of Willis and aneurysms in the fourth ventricle arise from a distal branch of posteroinferior cerebellar artery. Most IVAs are idiopathic, but the most common association is with Moyamoya disease. IVAs can also be found in association with AVM, atherosclerosis and trauma (Lévêque et al., 2011).

Main clinical presentation of IVA is hydrocephalus with either mass effect or hemorrhage.

3.1 Diagnosis

3.1.1 CT scan

CT shows pure intraventricular hematoma (IVH) in most ruptured IVA and IVH with slight subarachnoid hemorrhage (SAH) in some. In some cases, the site of the IVA can be

Author, year	Age, sex	Presentation	Location	Origin	Treatment	Outcome	Associated disease
Lemmen et al., 1953	8mo, ND	HCP	3V	PostChoA	Conservative	dead	none
Strully, 1955	27, F	HCP	LV	AntChoA	Trapping	poor	none
Cressman & Hayes, 1966	34, M	SAH	LV	AntChoA	Conservative	died	trauma
Schürmann et al., 1968	23, F	IVH	LV	MCA	resection	good	none
Butler et al., 1972	15, F	SAH	LV	AntChoA	Trapping	fair	AVM
Papo et al., 1973	57, F	SAH + HCP	LV	AntChoA	Trapping	poor	Atherosclerosis
Kodoma & Suzuki, 1978	16, F	SAH	LV	PostChoA	Conservative	good	Moyamoya
	39, M	SAH	LV	PostChoA	Conservative	good	Moyamoya
	48, F	SAH	LV	PostChoA	Conservative	good	Moyamoya
Babu & Eisen, 1979*	52, M	HCP	3V	AComA	VPS + Ligation	died	none
Tanaka et al., 1980	57, F	IVH	LV	AntChoA	Conservative	died	Moyamoya
Tyson et al., 1980*	75, F	IVH	3V	Basilar tip	External ventriclostomy	died	none
Pasqualin et al., 1981*	32, M	SAH	4V	PICA	clipping	good	none
Bose et al., 1983*	55, F	HCP	3V	Basilar tip	VPS + Clipping	died	none
Koga et al., 1983*	65, F	HCP	3V	Basilar tip	VPS	good	none
Piek et al., 1983*	60, F	HCP	3V	Basilar tip	V-A shunt	good	not registered
Kasamo et al., 1984	55, F	SAH + HCP	LV	AntChoA	clipping	died	Moyamoya
Nehls et al., 1985	18, F	ICH	LV	PostChoA	resection	good	AVM
Konishi et al., 1985	13, M	IVH	LV	AntChoA	Conservative	fair	Moyamoya
	57, F	IVH	LV	AntChoA	Conservative	died	Moyamoya
	34, F	IVH	LV	AntChoA	Conservative	died	Moyamoya
Borrie et al., 1985*	72, F	HCP	3V	Basilar tip	VPS	good	none
	70, M	HCP	LV	MCA	V-A shunt	good	none
Ungersböck & Perneczky, 1986	18, F	NR	LV	PostChoA	clipping	good	none
Knuckey et al., 1988	46, F	SAH	LV	AntChoA	Ligation	good	Atherosclerosis
Morota et al., 1988*	69, F	HCP	3V	ICA bif	VPS	good	none
Inagawa et al., 1990	75, F	SAH + HCP	LV	AntChoA	Conservative	died	none
Nishihara et al., 1993	34, F	SAH + IVH	LV	AntChoA	Excision	good	none
Hamada et al.,	48, F	IVH	LV	AntChoA	Trapping	good	Moyamoya

Author, year	Age, sex	Presentation	Location	Origin	Treatment	Outcome	Associated disease
1994							
Smith et al., 1994*	60, F	HCP	3V	PComA	clipping	good	none
Uranishi et al., 1994	65, F	IVH	4V	PICA	resection	moderate	none
Bergsneider et al., 1994	65, M	IVH	3V	PComA	resection	moderate	none
Urbach et al., 1995*	55, M	IVH	4V	PICA	clipping	good	not registered
Morgenstren et al., 1996	33, M	Ischemia	LV	AntChoA	Conservative	good	none
Koyama et al., 1996*	52, F	HCP	3V	Basilar tip	VPS	died	none
Kawai et al., 1997	19, M	IVH	LV	AntChoA	Conservative	fair	Moyamoya
Watanabe et al., 1999*	60. M	HCP	3V	Basilar tip	Coil embolization	good	none
Yanaka et al., 2000	8, F	IVH	LV	AntChoA	Trapping	good	AVM
Miyake et al., 2000	30, F	IVH + HCP	4V	SCA	resection	good	Moyamoya
	47, M	IVH	LV	PostChoA	resection	good	Moyamoya
	11, F	IVH	LV	PostChoA	Conservative	good	Moyamoya
Lee et al., 2001	48, M	ICH + IVH	LV	AntChoA	Trapping	good	Moyamoya
Hongo et al., 2001*	70, F	HCP	3V	Basilar tip	Endovascular occlusion	died	none
Gelal et al., 2002*	58, M	HCP	3V	Basilar tip	VPS	good	none
Kwok-chu Wong, 2003	62, F	ICH + IVH	LV	AntChoA	clipping	good	Moyamoya
Horie et al., 2003	77, F	IVH + HCP	4V	PICA	resection	died	none
Ali et al., 2004	26, M	IVH	LV	PostChoA	trapping + resection	good	Moyamoya
Liu et al., 2005*	55, M	HCP	3V	Basilar tip	ETV	good	none
Inci et al., 2007	19, F	ICH + IVH	LV	AntChoA	Trapping	good	none
	37, F	ICH + SAH	LV	AntChoA	Trapping	died	none
Koç & Ceylan, 2008*	58, F	SAH	3V	AComA	clipping	good	AVM
Tsutsumi et al., 2008*	58, M	HCP	3V	Basilar tip	VPS + Embolization	good	none
Oertel et al., 2009*	80, M	HCP	3V	Basilar trunk	ETV	good	none
	55, M	HCP	3V	Basilar tip	ETV + Coil embolization	died	none
	32, F	HCP	3V	Basilar trunk	ETV + Endovascular occlusion	died	none
Yurt et al., 2009	70, M	IVH	LV	AntChoA	clipping	good	none

Author, year	Age, sex	Presentation	Location	Origin	Treatment	Outcome	Associated disease
Leveque et al., 2011	50, F	IVH	LV	AntChoA	Endoscopic resection	good	Moyamoya
Sanli et al., 2011*	41, M	Mass effect	LV	Distal ACA	Coil embolization	good	none

ND= not determined, HCP= hydrocephalus, NR=not registered, LV=lateral ventricle, 3V=third ventricle, 4V=fourth ventricle, IVH=intraventricular hematoma, SAH=subarachnoid hemorrhage, ETV=endoscopic third ventriculostomy, VPS=ventriculoperitoneal shunt, PostChoA=posterior choroidal artery, AntChoA=anterior choroidal artery, MCA=middle cerebral artery, PComA=posterior communicating artery, ACA=anterior cerebral artery, PICA=posteroinferior cerebellar artery, SCA=superior cerebellar artery, asterics(*)=aneurysm's dome extending into the ventricle cavity.

Table 2. Presented cases of IVA in the literature.

estimated on the basis either as the IVH defect or as the prolonged presence of a hyperdense area on the ventricle wall (Hamada et al., 1994). In the course of a giant aneurysm, the appearance on CT is characteristic, consisting of a well-circumscribed round an oval mass (Artmann et al., 1984). The presence of thrombus determines the central attenuation characteristics, which may range from iso- to hyperdense. A peripheral zone of increased attenuation is frequently seen due to mural thrombus (Schubiger et al., 1987). After administration of the contrast media, CT scans show intense enhancement of the residual aneurysm lumen, and this rapidly declines after cessation of contrast media injection (Artmann et al., 1984).

3.1.2 MRI

MRI taken after resolution of IVH usually shows the site of the aneurysm clearly, precise location within the ventricle and the relation of the lesion to the parent vessel (Smith et al., 1994). The aneurysm usually appeared as flow void signs with marked gadolinium enhancement (Fig.1). Old blood clots often surround the aneurysm, with signal intensity depending on clot age.

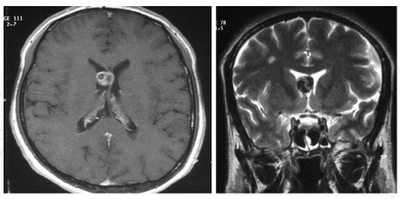

Fig. 1. MRI view of intraventricular aneurysm. Left, T1W image with gadolinium shows a nodular lesion in the lateral ventricle; right, T2W image shows a heterogenous lesion hypointense to brain tissue.

3.1.3 DSA

DSA is the method of choice in diagnosing aneurysms and provides reliable delineation of the aneurysmal lumen and its relationship to the parent vessel and adjacent arteries (Fig.2). But it has limitations; first it is invasive. Especially in the posterior fossa, where invasive surgical treatment is less often considered, less invasive diagnostic methods may be

preferred. Second, in DSA only the patent lumen is visualized, thus the thrombosed lumen and the aneurysm may be missed. However MRI-angiography and/or CT-angiography may provide complementary information in cases of thrombosed aneurysm, where the mass of the aneurysm is much larger than what is seen on angiographic filling. Although, IVA often cannot be found during the acute stage after hemorrhage, since the aneurysm or its parent artery is easily compressed by a packed IVH (Miyake et al., 2000). Therefore neuroradiological examination shall be repeated to confirm the diagnosis.

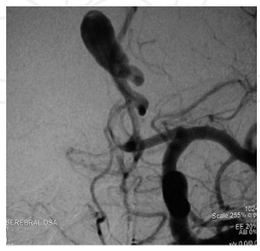

Fig. 2. Left carotid angiogram shows a saccular aneurysm of the distal anterior cerebral artery in the right lateral ventricle.

3.1.4 Differential diagnosis

Sometimes aneurysms in a ventricle cavity can cause difficulties in the differential diagnosis with mass lesions including AVM, CM, and tumors (colloid cysts and non-enhancing meningiomas) (Inci et al., 2007). Cross-sectional imaging modalities can usually be used to distinguish aneurysms from other mass lesions (Smith et al., 1994). Initially, a correct preoperative diagnosis is vital, and the wrong diagnosis of an aneurysm in an unusual location may encourage surgical removal of these lesions (Bose et al., 1983; Liu et al., 2005).

3.2 Managements

3.2.1 Conservative treatment

Conservative treatment can be effective for IVA, which may disappear spontaneously (Kodama & Suzuki, 1978; Konishi et al., 1985; Miyake et al., 2000). Rebleeding during observation may also occur (Hamada et al., 1984; Konishi et al., 1985). So, neuroradiogical examinations should be repeated; DSA is essential for this purpose.

3.2.2 Surgical treatment

Direct surgery should be seriously considered for most of the IVAs, especially for persistent IVAs, enlarged or reruptured aneurysms. Cerebral revascularization should be performed when the parent artery needs an important collateral route or in the case of misery cerebral perfusion.

Aneurysmal clipping and resection are the treatments of choice for IVAs depending on the size of the parent artery. For very small arteries, aneurysmal resection rather than clipping is preferred to prevent postoperative distortion and tears of the parent artery.Neuronavigation is helpful to minimize surgical damage when treating small and deeply situated lesions.

Direct aneurysmal coil embolization is difficult because the lesion is usually situated on a deep small branch and the aneurysm wall is fragile (Watanabe et al.,1999; Sanli et al., 2011). Endovascular occlusion of the parent artery is a choice when the parent artery can be sacrifided.

After aneurysmal SAH, ventriculoperitoneal shunt dependence due to hydrocephalus is a frequent observation with an incidence up to almost 20% (de Oliveira et al., 2007). Most patients require shunt treatment for malabsorbtive hydrocephalus, although some patients with aneurysms suffer from hydrocephalus due to CSF pathway obstruction rather than malresorption. CSF shunting is the most frequently performed procedure for treating obstructive hydrocephalus induced by a non-ruptured IVA (Hongo et al., 2001). For a ruptured aneurysm, experimental and clinical evidence suggests an increased risk of rebleeding when the pressure gradient across the aneurysm wall is increased (Nornes, 1973; Rosenørn et al., 1983). Several reports explain that ventricular drainage is not conclusively associated with an increased incidence of repeated aneurysmal rerupture (Hasan et al., 1989; Voldby & Enevoldsen, 1982). On the other hand, Pare et al., demonstrated an increased risk of aneurismal rebleeding in patients undergoing ventricular drainage, particularly in the presence of hydrocephalus. Obstructive hydrocephalus due to IVA was also treated with microsurgical ventriculostomy via transcallosal approach (Liu et al., 2005). Also, Oertel et al., reported 3 cases with obstructive hydrocephalus due to basilar artery aneurysm that were treated by endoscopic third ventriculostomy.

4. Intraventricular Arteriovenous Malformations (AVMs)

AVM located entirely in the ventricular system are uncommon and account for 4% of AVM in child (Humphreys, 1986) and 1.3% of AVM in adults (Jomin et al., 1985). Less than 40 intraventricular AVM have been reported. The most common locations are lateral ventricle and foramen of Monro. Only two well documented cases have been reported in the third ventricle in the literature (Heafner et al.,1985; Sanli et al., 2007).

Most intraventricular AVM have been manifested with spontaneous hemorrhage into the ventricle, and this causes posthemorrhagic hydrocephalus via blockage CSF absorption or mechanical blockage of CSF pathways. There were only two cases with an aqueductal lesion causing hydrocephalus have been reported (Song et al., 2008).

4.1 Diagnosis

4.1.1 CT scan

CT may be better than angiography in detecting angiographically occult intraventricular AVM (Song et al., 2008). AVM characteristically have high signal attenuation on CT scans and can be enhanced intensely (Fig.3). Miyasaka et al., considered that CT is more useful for;

- precise determination of the anatomical location of the lesion
- detection of angiographycally occult vascular malformations including CM or venous agiomas.
- recognition of the extent of the hemorrhage or the degree of hydrocephalus accompanying the small vascular malformation

Therefore, when angiography fails to reveal the responsible lesion, CT is very helpful for detection of the intraventricular vascular lesion. But it may give very little information about the type of the vascular lesion.

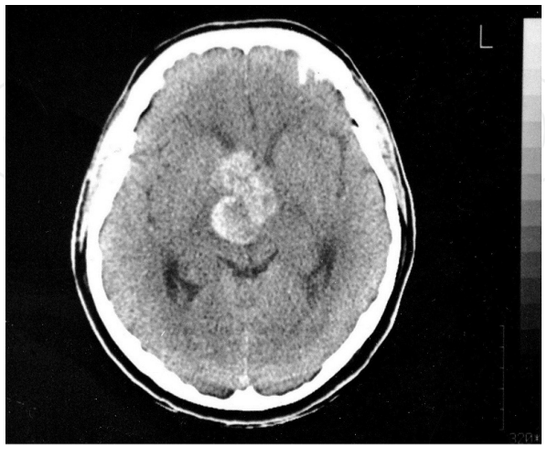

Fig. 3. CT scan shows an irregular hyperdense lesion in the third ventricle and mild hydrocephalus due to mass effect.

4.1.2 MRI

At this point MRI offers more detailed information about the primary lesion and its differential diagnosis. In MRI the nidus is hypointense on T1 weighed images (T1WI) and hyperintense on T2WI. This high signal intensity on T2WI is due to vascular stasis. The signal void linear straits belonging to drainage vessels seen on angiography give a clue to AVM (Fig.4). Therefore, MRI can be helpful in differential diagnosis of granulomatous or highly vascular neoplastic lesions such as choroids plexus papilloma and carcinoma (Gürcan et al., 1998).

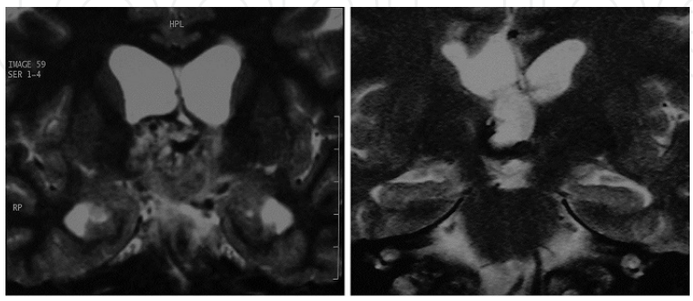

Fig. 4. left, coronal T2W image shows a mass with heterogeneous signal intensity in the third ventricle and dilated temporal horns of the lateral ventricles. The lesion includes tubular signal void areas. Right, early postoperative MRI after right transcallosal approach.

4.1.3 DSA

Cerebral DSA is the most reliable radiological examination for the diagnosis of AVM (Fig.5). Intraventricular AVMs are commonly angiographycally occult, because these lesions are often too small to be detected by angiography or because hemorrhage or thrombosis of the involved vessels may destroy them (Roda et al., 1981; Sanli et al., 2007). Angiography showed no evidence of AVM in about 20% of intraventricular AVM (Tamaki et al., 1994).

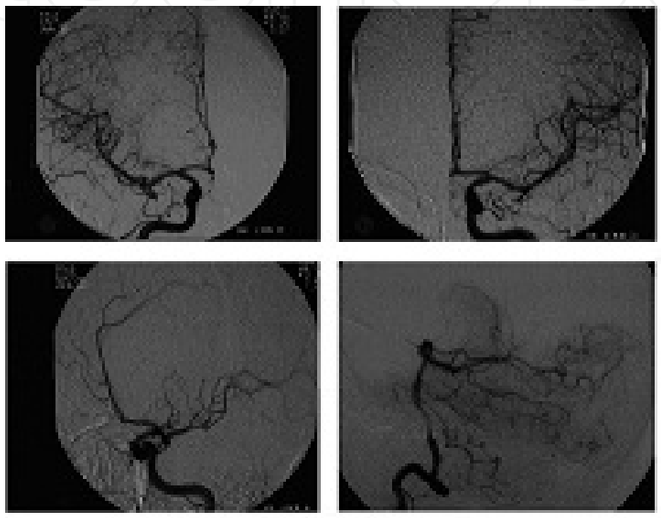

Fig. 5. Both carotid arteries and left vertebral artery angiographies show no any vascular pathologies.

4.1.4 Differential diagnosis

Intraventricular AVM with a typical images are not rare. Misdiagnosis is common because the characteristic signal void may be absent or angiographic results may be negative. Moreover, some intraventricular AVM may radiologically mimic tumors (Britt et al., 1980).

Transfontanel ultrasonography is helpful for finding hydrocephalus, IVH and intracerebral hemorrhage secondary to AVM. Transfontanel ultrasonography is safetly used for diagnosing intraventricular AVM in neonates (Ceylan et al., 1993; Heck et al., 2002). Correct preoperative diagnosis of intraventicular AVM is important for appropriate treatment planning.

4.2 Managements

Overall, 2-4% of brain AVM bleed, and the hemorrhage rates are higher in children than adults (Song et al., 2008). Bleeding rates of the intraventricular AVM are 81% in children and 95%in adults, and incidences of rebleeding and resultant are also high (Tamaki et al., 1994).

4.2.1 Conservative treatment

Despite the fact that intraventicular AVMs are often seemed inoperable because of their deep location and sometimes intimidating vascular patterns, there is evidence to suggest that their natural history is unfavorable with a high incidence of hemorrhagic complications, so that intraventricular AVMs must be treated (Batjer & Samson, 1987). But the treatment of these lesions remains a challenging matter. Surgical excision, endovascular embolization, radiosurgery or a multimodality approach have been used to treat this condition, however studies are not conclusive yet (Ogilvy et al., 2001).

4.2.2 Surgical treatment

The ideal treatment for a cerebral AVM is total surgical resection. AVM of the ventricles are generally small enough to be removed safely by microsurgical techniques (Tamaki et al., 1994). Preoperative embolization may be helpful, although embolization is rarely curative.

Fahim et al., reported that the transtubular microendoscopic approach may be advantageous for resecting intraventricular lesions by avoiding unnecessary retraction; therefore, it may reduce the risk of injury to the surrounding brain tissue.

Yamada et al., reported the endoscopic resection of the intraventricular AVM.

Total surgical excision is very important for intraventricular AVM because any mass left after surgery, radiosurgery or embolization can result in hemorrhage or hydrocephalus.

Regarding the craniotomy microsurgery and neuroendoscopy; former is safer for managing bleedings during surgery (Moftakhar et al., 2006). Surgery for pediatric cases may be delayed because procedures become less difficult as child grows (Heck et al., 2002).

5. Intraventricular venous malformations

Developmental venous anomalies (DVA) and venous loops or varices may cause hydrocephalus by blocking CSF pathways at the Sylvian aqueduct. Also they may cause unilateral dilatation of a ventricle due to blockage at the foramen of Monro. DVA, also known as venous agiomas, represent nonpathological variations in venous drainage. DVA represent the most common intracranial vascular malformations, composing of 63% of such lesions in autopsy series (Garner et al., 1991). To our knowledge, there were only 12 reported cases of intraventricular venous anomalies causing hydrocephalus (Table-3).

DVA and venous varices have similar wall characteristics; where the walls of both are composed of intima and adventitia without presence of media (Kelly et al., 1995). DVA consist of multiple dilated anomalous veins with interposed neural tissue and ≥1 dilated draining vein, typically presenting pathologically as a conical or wedge-shaped lesion with its base at the meninges and its apex toward the ventricle (Fierstien et al., 1979). The intervening parenchyma is normal. Venous varices, on the other hand, are characterized as a focal dilatation of a single vein without neural tissue (Kelly et al., 1995). Although intraventricular DVA may not have the characteristic interposition of neural tissue, they are characterized by dilatation of multiple anomalous veins (Leonardo & Grand, 2009).

The most popular theories for the etiology of these benign lesions include primary dysplasia of capillaries and small transcerebral veins (Ostertun & Solymosi, 1993) or a compensatory mechanism when normal venous pathways are by-passed due to accidental thrombosis in the intrauterine period (Saito & Kobayashi, 1981).

Most common location of the CSF pathway obstruction causing hydrocephalus is Sylvian aqueduct. There are only two reports of unilateral hydrocephalus caused by venous malformation due to obstruction of the foramen of Monro (Leonardo & Grand, 2009; Tien et al., 1990).

Author, Year	Age, Sex	Symptom	Imaging	Treatment	Outcome
Rosenheck, 1937	58, F	Mental deterioration	Postmortem	none	dead
Avman & Dinçer, 1980	35, F	Headache	Ventriculography + CT + angiography	ventriculostomy	good
Tien et al., 1990	37, F	Headache, gait instability	MRI + CT	septum pellucidum fenestration	good
Watanabe at al., 1991	39, M	Headache	CT + MRI + angiography	shunt	good
Oka et al., 1993	43, F	Seizure	CT + MRI (cine) + angiography	ETV	good
Blackmore & Mamourian, 1996	16, F	Headache, behavior abnormalities	MRI (cine)	none	good
Bannur et al., 2002	11, M	Headache	CT + MRI (cine)	shunt	good
Sato et al., 2004	28, F	Headache	CT + MRI + angiography	ETV	good
Yagmurlu et al., 2005	7, F	Headache	MRI	none	good
Giannetti et al., 2008	42, M	Headache, behavior abnormalities	MRI + CT	ETV	good
	18, M	Headache	MRI + CT	ETV	good
Leonarda & Grand, 2009	28, M	Headache	MRI + CT + MR angiography	Endoscopic septum pellucidum fenestration	good

CT=computed tomography, MRI= magnetic resonance imaging, ETV=endoscopic third ventriculostomy

Table 3. Presented cases of intraventricular venous lesions in the literature.

5.1 Diagnosis

CT may not be helpful in the diagnosis, and MRI has proven to be the most sensitive diagnostic tool (Oka et al., 1993). In general, the lesions are hypointense in T1WI and enhance intensely after the contrast media administration. On T2WI they have high or low signal intensity depending on the flow velocity, the orientation and the pulse sequence. Cine

MRI has also used to confirm the location of hydrocephalus in some cases (Blackmore & Mamourian, 1996). The role of DSA is limited and can, at least, document the anomaly of the associated anomalous venous drainage (Bannur et al., 2002).

5.2 Managements

DVA and venous varices are benign lesions that do not need any treatment because there is no risk of rupture or bleeding. Also, they present normal blood outflow pathways, and any attempt in removing them surgically, can cause venous infarction or edema (Bannur et al., 2002; Blackmore & Mamourian, 1996; Yagmurlu et al., 2005).

Hydrocephalus caused by venous anomalies can easily be treated by shunts or endoscopic third ventriculostomy. As listed in the table 3, the outcome is almost always good.

6. Conclusion

The cerebrovascular pathologies cause hydrocephalus by obstruction of CSF pathways due to the slow growing of the vascular lesion or hemorrhage. Since the cerebrovascular lesions are complex pathologies, there is not an agreement among neurosurgeons on treatment. The lack of more clinical and surgical experiences makes a decision with difficulty in the treatment of the intraventricular cerebrovascular lesions.

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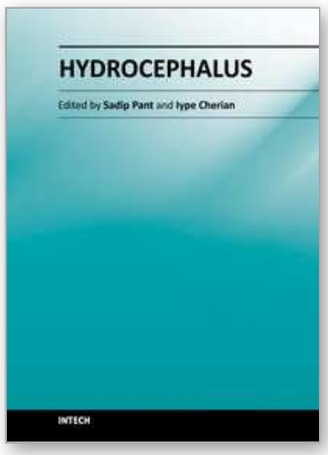

Hydrocephalus

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Description of hydrocephalus can be found in ancient medical literature from Egypt as old as 500 AD. Hydrocephalus is characterized by abnormal accumulation of cerebrospinal fluid (CSF) in the ventricles of the brain. This results in the rise of intracranial pressure inside the skull causing progressive increase in the size of the head, seizure, tunneling of vision, and mental disability. The clinical presentation of hydrocephalus varies with age of onset and chronicity of the underlying disease process. Acute dilatation of the ventricular system manifests with features of raised intracranial pressure while chronic dilatation has a more insidious onset presenting as Adams triad. Treatment is generally surgical by creating various types of cerebral shunts. Role of endoscopic has emerged lately in the management of hydrocephalus.

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