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Original Investigation

Spontaneous Primary Intraventricular Hemorrhage in Adults: Clinical Data, Etiology and Outcome

Yetişkinlerde Spontan Primer İntraventriküler Kanama: Klinik Bulgular, Etiyoloji ve Sonuçları

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ABSTRACT

AIM: Primary intraventricular hemorrhage (PIVH), bleeding in the ventricular system without a recognizable parenchymal component, is a rare neurological disorder. The purpose of this study was to identify clinical features, risk factors, etiology and outcome of patients with PIVH.

MATERIAL and METHODS: We retrospectively reviewed the clinical data, complementary examinations, outcome and computed tomography (CT) IVH score of 24 patients in our hospital from 2004 to 2008. We identified 24 patients with the inclusion criteria of non-traumatic PIVH. Their mean age was 60.6±17.4 years (range 38-79). Fourteen patients were male and 10 were female.

RESULTS: The major symptoms included headache (n=24), loss of consciousness (n=6), confusion and disorientation (n=14), nausea/vomiting (n=10). Angiography revealed vascular malformations in five patients (21%). Other possible causative factors were hypertension in 12 patients (50%) and clotting disorder in one. The aetiology remained unknown in six patients. Most PIVH patients had associated hydrocephalus (58%) and 37% of the patients required ventricular drainage. In-hospital mortality was high (41%) and a FOUR score ≤10, GCS ≤8 and early hydrocephalus were independent predictors of mortality.

CONCLUSION: Hypertension is the most common associated risk factor for PIVH followed by vascular malformation. Spontaneous resorption and rebleeding may be seen. The neurological status of the patients and an early developing hydrocephalus are the most important risk factors.

KEYWORDS: Primary intraventricular hemorrhage, Hypertension, Arteriovenous malformation, Hydrocephalus

ÖZ

AMAÇ: Primer intraventriküler kanamalar (PİVK), intraparankimal kanama olmaksızın ventriküler sistem içinde kan olması şeklinde tanımlanmıştır. Spontan ve non-travmatik olarak da adlandırılan PIVK'lar oldukça nadir gözlenir. Özellikle PİVK'ların klinik özellikleri ve prognozlarına ait veri oldukça azdır. Bu çalışmanın amacı, PİVK'lı 24 erişkin hastanın klinik özelliklerini, risk faktörlerini, etiyolojisini ve prognozunu araştırmaktır.

YÖNTEM ve GEREÇ: Hastanemizde 2004-2008 yılları arasında takip edilen 24 PİVK'lı hasta retrospektif olarak klinik bulgular, geniş nörolojik değerlendirme, prognoz ve bilgisayarlı beyin tomografi-intraventriküler kanama skorları açısından değerlendirilmiştir. Travmatik olmayan PİVK'lı hasta sayısı 24 idi. Hastaların yaş ortalaması 60,6±17,4 yıl idi.

BULGULAR: Ana bulgular başağrısı (n=24), bilinç kaybı (n=6), konfüzyon veya dezorientasyon (n=14), bulantı/kusma (n=10) idi. Beş hastada (%21) anjiyografide malformasyon tespit edildi. Diğer muhtemel nedenler; 12 hastada hipertansiyon (%50) ve bir hastada pıhtılaşma bozukluğu saptandı. Altı hastada ise etiyoloji tespit edilemedi. PİVK'lı hastaların %58'sinde hidrosefali gelişti ve %37'sinde eksternal ventriküler drenaj takılmasına ihtiyaç duyuldu. Hastane mortalitesi yüksek bulundu (%41). Full Outline of Unresponsiveness Score ≤10, Glasgow Koma Skorunun ≤8 olması ve erken hidrosefali mortaliteyi etkileyen faktörlerdi.

SONUÇ: Vasküler malformasyonlar hipertansiyondan sonra PİVK'lara en sık eşlik eden risk faktörüdür. Spontan rezorbsiyon ve tekrarlayan kanama görülebilir. Hastaların nörolojik durumları ile gelişebilecek erken hidrosefali prognozu belirleyen en önemli faktörlerdir. PİVK olgularında mortalite oranını düşürecek yeni tedavi stratejilerine ihtiyaç duyulmaktadır.

ANAHTAR SÖZCÜKLER: Primer intraventriküler kanama, Hipertansiyon, Arteriovenöz malformasyon, Hidrosefali

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INTRODUCTION

Primary intraventricular hemorrhage (PIVH), bleeding in the ventricular system without a discernable parenchymal fragment, is a rare neurological disorder (2,6,23,26,27,32). PIVH was defined for the first time by Sanders in 1881 as the flooding of the ventricle by blood without the presence of any rupture or laceration in the ventricular wall (26). The incidence of PIVH among all the patients with intracranial hemorrhage is 3.1% where this rises to 9% among the patients having intraparenchymal hematoma (2,6,23,26). The prognosis of patients with PIVH has been reported to be better than the prognosis of the patients with a diagnosis of secondary intraventricular hemorrhage (23,27).

Intraventricular hemorrhage was considered a fatal disease in the era before modern brain imaging because it could be diagnosed only during postmortem examination. The introduction of brain imaging techniques has enabled diagnosis of PIVH and therefore also in patients with less extensive hemorrhage but relatively little is known about the clinical and imaging features, etiology and prognosis. Most series of patients with intraventricular hemorrhage also include those with secondary or traumatic hemorrhages (5,12,17,20,21) or the few reported series of patients have often been small (6,9,28).

The purpose of this study was to identify clinical features, risk factors, etiology and outcome in a sample of 24 adult patients with primary IVH.

MATERIALS and METHODS

We retrospectively reviewed the medical records of all patients at a tertiary referral hospital from 2004 to 2008 with a diagnosis of PIVH. We defined PIVH as hemorrhage detected by CT in the ventricular system only. We excluded patients with intraparenchymal hemorrhage, even if the hemorrhage was small or very close to the ventricular system, and also patients with intracerebral hemorrhage related to trauma or with subarachnoid hemorrhage.

We recorded the following clinical data (variables): (i) individual characteristics and risk factors – sex, age, history of arterial hypertension (systolic pressure higher than 160 mmHg or diastolic higher than 90 mmHg or the patient was under antihypertensive therapy), diabetes (previous diagnosis of diabetes and/or past or present use of antidiabetic agents or need of antidiabetic treatment on discharge), previous stroke (ischemic or hemorrhagic) and use of antiplatelet or anticoagulant medication; (ii) clinical features at onset; (iii) initial neurological examination and clinical parameters on admission such as blood pressure, Full Outline of Unresponsiveness (FOUR) Score, Glasgow Coma Scale (GCS); (iv) initial features of PIVH (ventricles involved, amount of blood, presence of hydrocephalus) and (v) clinical course and outcome. All living patients were interviewed by telephone to identify neurological sequelae and educational or occupational achievement after the hemorrhage.

The volume of blood was assessed by CT (IVH score) with a semiquantitative scale as described by Graeb et al. (12). Lateral ventricles: 1, less than one quarter of ventricle filled with blood; 2, less than half ventricle filled with blood; 3, less than three quarters of ventricle filled with blood; 4, more than three quarters of ventricle filled with blood. Third and fourth ventricles: 1, blood present, ventricle size normal; 2, ventricle filled with blood and expanded (hence a maximum score of 12 points). Ventricular enlargement was graded with a semiquantitative scale (16) and recorded as present or absent. Adverse in-hospital events such as serious medical complications and neurological deterioration because of CT proven rebleeding and brain ischaemia were recorded.

The Chi square test, Student's t-test, and Spearman rank correlation were used where indicated to compare the outcomes. Predictors of in-hospital mortality were assessed by univariate and multivariate analysis using the Cox proportional hazards model, in which baseline characteristics were treated as fixed covariates and variables that developed in time (adverse in-hospital events) were treated as time dependent covariates. Categorical and continuous variables were converted to binary variables using the following cut point: FOUR score ($\leq$ 10 vs >10), GCS score ($\leq$ 8 vs > 8), mean arterial blood pressure (MABP <120 vs $\geq$ 120 mmHg), CT ventricular blood score (<6 vs $\geq$ 6), age ($\leq$ 65 vs >65). The odds ratio (OR) and 95% confidence interval (95 % CI) are indicated.

RESULTS

We identified 24 patients with the inclusion criteria of non-traumatic PIVH over a 5-year period. Their mean age was 60.6±17.4 years (range 38-79), 17 were older than 50 years (Table I). Fourteen patients were male and 10 were female. The most frequent risk factor was arterial hypertension observed in 12 patients (two untreated and ten on antihypertensive treatment). Four patients were current smokers, two patients had diabetes, four had previous ischemic stroke, two were on antiplatelet treatment and one on anticoagulants.

Onset was sudden in 16 patients and fluctuating or progressive in eight. Loss of consciousness was the first manifestation of primary IVH in four patients and occured after other symptoms had appeared in two. In the other patients, the onset was characterized by confusion and disorientation (n=14), or by headache with or without vomiting (n=6). In general, headache was the most frequent symptom, being reported by all noncomatose patients; other symptoms were confusion and disorientation (n=14), vomiting/nausea (n=10), loss of consciousness (n=6), and vertigo (n=3). Focal symptoms or signs were rarely observed (n=3) as

Table I: Clinical data, etiological diagnosis, and prognosis of 24 patients with primary intraventricular hemorrhage.

Age/sex	Initial symptoms	GCS/FOUR on admission	IVH score	Hydrocephalus	Etiology (or risk factor)	Outcome (cause of death)
49/F	C	3/8	10	Early(+),EVD	AVM	Death(PIVH)
46/M	LOC	5/7	10	Early(+),EVD	AVM	Death(PIVH)
63/M	He,V/N	13/13	5	No	HT	Mild memory problems
38/F	C,PS	14/13	5	No	HT	Mild hemiparesis
60/M	C,He,V/N	6/9	8	Early(±)	AVM	Death (PIVH)
64/M	C,He,V/N	13/13	4	Early(-)	HT	Mild memory problems
47/M	C,He,V/N	14/13	3	No	HT	Asymptomatic
45/F	C,PS	6/8	6	Early(+),EVD	AVM	Death (PIVH)
63/F	LOC,ES,PS	14/13	3	Early(-)	HT	Mild hemiparesis
52/F	C	15/14	3	No	AVM	Asymptomatic
46/M	He,then LOC	14/13	4	No	Unknown	Asymptomatic
66/M	LOC,ES	7/9	6	Early(+),EVD	Unknown	Death (PIVH)
59/M	LOC	14/14	2	Early(-)	Unknown	Mild memory problems
79/F	C	6/8	6	Early(-)	Unknown	Death (PIVH)
68/M	C	4/9	8	Early(-)	HT	Death (PIVH)
53/M	He,then LOC	15/16	2	Early(+),EVD	HT	Asymptomatic
78/F	C,He,V/N	15/16	3	No	Unknown	Asymptomatic
70/M	C,He,V/N	15/16	3	No	HT	Asymptomatic
74/F	C	6/9	4	Early(+)	HT	Death (PIVH)
50/M	He,V/N	15/16	1	No	HT	Asymptomatic
70/M	C,He,V/N	6/8	6	Early(+)	Unknown	Death (rebleeding)
73/M	He,V/N,ES	15/16	1	No	HT	Asymptomatic
72/F	He,V/N	15/16	2	No	HT	Asymptomatic
69/F	C	10/10	4	Early(+)	Anticoagulant	Death (rebleeding)

M, male; F, female; He, headache; V/N, vomiting and nausea; C, confusion; LOC, loss of consciousness; ES, epileptic seizure; PS, pyramidal sign; (+) increased or (-) decreased on repeated CT brain scan; EVD, external ventricular drainage; HT, hypertension; AVM, arteriovenous malformation.

were epileptic seizures. The former included nuchal rigidity (n=3), mild hemiparesis (n=2) and unilateral/bilateral extensor plantar responses (n=3). In addition, neuro-ophthalmological abnormalities were rarely detected (n=2) as were third cranial nerve palsies. On examination, 22 patients had some degree of impairment of consciousness:13 were confused, one stuporous, two lethargic and six comatose.

CT was performed on all patients and initial CT revealed extravasation of blood into all four ventricles in 10 patients (41%), both lateral and third ventricles in four, both lateral ventricles in eight and to the third and fourth ventricles in two. The IVH score varied extensively (range 1-10, median 4). Signs of transtentorial herniation were not observed in any case. Digital subtraction angiography (DSA) was performed in 17 patients (71%), with normal results in twelve and arteriovenous malformation (AVM) detected in five patients (21%). No cause was detected in six patients with four having a normal DSA and two refusing this procedure. Other possible causative factors were hypertension in 12 patients (eight of whom underwent angiography) and clotting disorder in one. DSA was not performed in a total of 7 patients.Hypertension (n=2) and anticoagulation (n=1) were suspected as the causes of bleeding in three patients who died before cerebral angiography. Another two patients refused this procedure and it was not considered in the remaining two patients (due to medical complications).

Most PIVH patients had associated early hydrocephalus (n=14, 58%) and approximately one-third required external ventricular drainage (EVD,n=5). Early hydrocephalus was associated with poor prognosis. EVD was performed in five patients after clinical worsening, and reduced ventricular enlargement, but improvement was only seen in one patient. A second CT brain scan revealed increased ventricular enlargement in eight patients and decreased in six patients. Ten patients (42%) died during the treatment, two soon after admission as a direct consequence of PIVH, six after worsening of clinical status associated with increased hydrocephalus and two of rebleeding after clinical worsening because of hydrocephalus. Control CT brain scan showed spontaneous resorption of the hemorrhage performed by the end of the second week after the event in ten patients (n=10, 42%). Medical complications included gastrointestinal bleeding, pneumonia, supraventricular tachycardia, acute renal insufficiency, hypokalemia, hypernatremia and hyponatremia, each of which was observed only once.

For outcome assessment, the variables that appeared as significant univariate predictors of in-hospital mortality were FOUR score (OR 4.20; 95% CI 1.90-8.46, P=0.0003), GCS (OR 4.25; 95% CI 1.97-9.56, P=0.0005), early hydrocephalus (OR 4.64; 95% CI 1.64-12.79, P=0.05), and IVH score (OR 4.39; 95% CI 1.94-9.86, P=0.0003). Multivariate analysis indicated that the FOUR score, GCS and early hydrocephalus were the only independent predictors of poor outcome (Table II).

After a mean follow-up of 20 months, outcome in the surviving patients was asymptomatic in nine, mild hemiparesis in two, and mild memory problems in three patients. No surviving patient deteriorated to a severely disabled or vegetative state.

DISCUSSION

In our study, we retrospectively reviewed the clinical data and imaging features of 24 patients with a diagnosis of PIVH. We found these cases in a 5-year period at our institution, which cares for almost 200 patients per year with intracerebral hemorrhages, thus demonstrating the rarity of PIVH (2.4%). Sanders have described that the ventricular hemorrhage is distributed over the life span but makes a peak between the ages of 40-60 years (26). In our study, the mean age of the patients was found to be 60.6±17.4 years (range 38-79). There was no difference in terms of age between the males and the females. In addition, the male to female ratio was found to be 1.4:1.

Some series have separated PIVH into two clinical groups related to the type of presentation (9,10,11,26). The first group, so-called classical clinical scenario, was characterized clinically by sudden (acute) onset, with coma or decreased level of consciousness, nausea or vomiting, nuchal rigidity and severe headache, and bilateral extensor plantar responses, followed by fatal brainstem dysfunction (1,4,6,12). The second group was characterized clinically by fluctuating or progressive (subacute) onset (4,18), with gradually increased intracranial pressure with headache, vomiting, confusion,

Table II: Predictors of in-hospital mortality via the Cox proportional hazards model.

Variable	Univariate analysis		Multivariate analysis	
	OR (95% CI)	*p-value*	OR (95% CI)	*p-value*
Age >65 years	1.33 (0.70-2.66)	0.47		
Male sex	0.65 (0.34-1.25)	0.2		
Admission MABP>120 mmHg	0.77 (0.35-1.44)	0.32		
GCS≤8	4.25 (1.97-9.56)	0.0005	4.72 (1.27-18.67)	0.034
FOUR≤10	4.20 (1.90-8.46)	0.0003	4.22 (1.17-21.47)	0.037
Early hydrocephalus	4.64 (1.64-12.79)	0.05	4.99 (1.23-22.63)	0.044
Adverse events	1.65 (0.90-2.99)	0.24		
IVH score≥6	4.39 (1.94-9.86)	0.0003		

MABP, mean arterial blood pressure.

drowsiness and transient loss of consciousness developing over hours or days.

Primary intraventricular haemorrhage has been described with arteriovenous malformations (AVM), aneurysms, arteriovenous fistulae, moyamoya disease, tumors of the choroid plexus, fibromuscular dysplasia, colloidal cysts, rupture of the galen vein, and coagulopathy (1,9,14,20,24,33). The studies of Van den Bergh et al. and Butler at al. have provided much valuable information about the etiology of the PIVH (3,30). It has been reported that developmental anomalies of the vascular system like AVM and aneurysms within this area are more frequently seen due to the presence of dense vascularization hence may be leading to hemorrhages. Moreover, the literature contains information on hemorrhages protruding into the ventricles from periventricular angiomas and saccular aneurysms of the deep vascular structures (19,20,33). Gates et al. have reported the occurrence of PIVH in a patient with moyamoya disease (9). In the present study, in which 29% of patients were under 50 years and 71% underwent cerebral angiography, AVM was found in 21% of patients, the same percentage as reported by other authors (0-31%)(9,15,18,24,28,31). In addition, most of the AVM patients in our study were younger than 50 years as in other studies (6,15,18,24).

Hypertension is also one of the most commonly associated risk factors for primary intraventricular haemorrhage. PIVH was attributed to arterial hypertension in twelve of our patients (50%). In fact, hypertension was the only identified risk factor in these patients. Caplan has declared that the blood that extravasates into the ventricle in PIVH associated with arterial hypertension usually comes from the choroid plexus, caudate and thalamus (4). In a series Little et al. have reported the presence of hypertension in 44% and saccular aneurysms in 26% (17). However the series Pia et al. reported the rate of hypertension, aneurysms and AVMs as 54%, 19% and 27% respectively (25). Hypertension was the only identified risk factor in 50% of patients in the series by Angelopoulos et al (1), in 75% in the series by Tembl et al. (28) and in 80% in the series by Hameed B et al. (13). In our study, hypertension and AVM made up 71% of all the etiological factors.

Various treatment protocols like medical treatment, external ventricular drainage and surgical intervention are applied to the patients with PIVH (14,15,17,24,25). As reported in other studies (1,15,18,28), hydrocephalus was also a frequent complication in our patients and was probably due to obstruction of cerebrospinal fluid circulation or impairment in meningeal absorption. It usually self-corrects spontaneously, although a temporal or definite surgical shunt is sometimes needed (12). In this study we treated 19 patients with medical therapy and applied EVD to 5 (21%) of the 14 (58%) patients in whom hydrocephalus was established. One of the patients showed a regression in ventricular diameters after the application of EVD and hence received a ventriculo-peritoneal shunt. Also, six patients died after worsening of clinical status associated with increased hydrocephalus (four of whom underwent EVD). The number of patients

who underwent EVD in this study was too small to allow conclusions on the effect of ventriculostomy at presentation or after deterioration from obstructive hydrocephalus. Our findings reveal that, the rate of poor outcome for patients with EVD was alarmingly high. Rebleeding was established in two of the deceased patients. In fact, rebleeding occurred after a hypertensive attack in these two patients. Passero et al. have reported the importance of the treatment for hydrocephalus in the PIVH patients and added that rebleeding would make the treatment more complicated (24). Our study also reveals that this complication is associated with a poor outcome. CT scan is the primarily preferred method in the follow up of patients with IVH. Spontaneous resorption of the hemorrhage leaving only minimal ventricular changes on the CT scans performed on the 12th day in patients with a good prognosis has been reported in the series by Little et al. (17). In our study, we found resorption of the hemorrhage to a great extent on the CT scans performed by the end of the second week of the patients with a good prognosis after the event (42%). In recent years, some reports have foreseen better results for the future with the application of urokinase and tissue plasminogen activator in PIVH patients that have ventricles flooded with blood together with low GCS scores (7,8,22,29,35).

The prognosis of PIVH has altered as our diagnostic ability has improved, allowing the definition of more benign courses. Poor prognosis and death are thought to occur inevitably from fatal brainstem dysfunction. In-hospital mortality in our series was 42%; that reported in other series has been 20% (9), 28% (6), 36% (1), 42% (24) and 46% (15). The outcome of patients with PIVH has been reported to be better than the outcome of the patients with secondary intraventricular hemorrhage (6,23,27). Focal motor symptoms or signs were absent or mild in patients with PIVH, and this probably was related to a midline hemorrhage without parenchymatous damage. On the other hand, agitation, confusion, disorientation, complaints of poor concentration and poor memory, and gait ataxia are relatively frequent (1,6,18).

The literature contains a limited number of studies explicating the factors affecting the prognosis of the patients that have PIVH (9,15,24,27,34). Some studies have ascertained factors like hypertension and its degree, neurological status at the time of application, and progression of the symptoms or an accelerated deterioration in the consciousness to be prognostic in these patients (15,17). Univariate analysis has revealed an association between poor outcome and some clinical or imaging features (13,34) while other studies did not indicate any predictors of mortality due to the small number of patients (18,31). In our study, age, sex and hypertension did not prove to be effective on the outcome but GCS and FOUR score and development of early hydrocephalus were estimated to be prognostic risk factors. The total result of IVH scores was a significant predictor of mortality according to univariate analysis, but lost its predictive value when multivariate analysis was performed.

In conclusion, hypertension is the most commonly associated risk factor for primary IVH followed by vascular malformations. Spontaneous resorption and rebleeding may be seen. The neurological status of the patients and an early developing hydrocephalus are the most important risk factors for the determining the prognosis. New treatment strategies are needed to improve the mortality rates in the PIVH patients.

REFERENCES

1. Angelopoulos M, Gupta SR, Azat Kia B: Primary intraventricular hemorrhage in adults:clinical features, risk factors, and outcome. Surg Neurol 44:433-436,1995
2. Brott T, Thalinger K, Hertzberg V: Hypertension as a risk factor for spontaneous intracerebral haemorrhage. Stroke 17:1078-1083,1986
3. Butler AB, Partain RA, Netsky MG: Primary intraventricular hemorrhage. A mild and remediable form. Neurology 22:675-687,1972
4. Caplan LR: Primary intraventricular haemorrhage. In: Kase CS, Caplan LR, ed,. Intracerebral hemorrhage. London: Butterworth-Heinemann, 1994:383-401
5. Cohn DF, Avrahami E: Intraventricular hemorrhage. CT and arteriographic findings in thirty-five patients. J Neurol 230:137-140,1983
6. Darby DG, Donnan GA, Saling MA, Wals KW, Bladin PF: Primary intraventricular hemorrhage: Clinical and neuropsychological findings in a prospective stroke series. Neurology 38:68-75,1988
7. Findlay JM, Weir BK, Stollery DE: Lysis of intraventricular hematoma with tissue plasminogen activator: Case report. J Neurosurg 74:803-807,1991
8. Fountas KN, Kapsalaki EZ, Parish DC, Smith B, Smisson HF, Johnston KW, Robinson JS: Intraventricular administration of tr-PA in patients with intraventricular hemorrhage. South Med J 98:767-773,2005

9. Gates PC, Barnett HJM, Vinters HV, Simonsen RL, Sıu K: Primary intraventricular hemorrhage in adults. Stroke 17:872-877,1986
10. Gordon A: Ventricular hemorrhage: A symptom group. Arch Intern Med 17:343-353,1916
11. Gordon A: Primary ventricular hemorrhage. Further contribution to a characteristic symptom group. Arch Neurol Psychiat 39:1272-1276,1938
12. Graeb DA, Robertson WD, Lapointe JS, Nugent RA, Harrison PB: Computed tomographic diagnosis of intraventricular hemorrhage. Etiology and prognosis. Radiology 143: 91-96,1982
13. Hameed B, Khealani BA, Mozzafar T, Wasay M: Prognostic indicators in patients with primary intraventricular haemorrhage. J Pak Med Assoc 55:315-317, 2005
14. Irie F, Fujimoto S, Uda K, Toyoda K, Hagiwara N, Inoue T, Okada Y: Primary intraventricular hemorrhage from dural arteriovenous fistula. J Neurol Sci 15;(215):115-118, 2003
15. Jayakumar PN, Taly AB, Bhavani UR, Arya BYT, Nagaraja D: Prognosis in solitary intraventricular hemorrhage: Clinical and computed tomographic observations. Acta Neurol Scand 80:1-5,1989
16. Lisk DR, Pasteur W, Rhoades H, Putnam RD, Grotta JC. Early presentation of hemispheric intracerebral hemorrhage: Prediction of outcome and guidelines for treatment allocation. Neurology 44:133-139, 1994
17. Little JR, Blomquist GA Jr, Ethier R: Intraventricular hemorrhage in adults. Surg Neurol 8: 143-149,1977
18. Marti-Fabregas J, Piles S, Guaria E, Mart-Vilalta J-L:Spontaneous primary intraventricular haemorrhage: Clinical data, etiology, and outcome. J Neurol 246:287-291,1999
19. Miyasaka Y, Tanaka R, Tokiwa K, Itikawa H, Suwa T, Takano S, Ohtaka H, Kurata A, Endo M, Saito M. Clinical significance of intracranial hemorrhage caused by cerebral arteriovenous malformations: with special reference to intraventricular hemorrhage. No Shinkei Geka 17:133-138,1989
20. Mohr G, Ferguson G, Khan M, Malloy D, Watts R, Benoit B, Weir B: Intraventricular haemorrhage from ruptured aneurysm. Retrospective analysis of 91 cases. J Neurosurg 58:482-487,1983
21. Müller H, Brock M: Primary intraventricular traumatic hemorrhage. Surg Neurol 27: 398-402,1987
22. Naff NJ, Hanley DF, Keyl PM, Tuhrim S, Kraut M, Bederson J, Bullock R, Mayer SA, Schmutzhard E: Intraventricular thrombolysis speeds blood clot resolution: Results of a pilot, prospective, randomized, double-blind, controlled trial. Neurosurgery 54:577-583,2004
23. Ojeman RG, Heros RC: Spontaneous brain hemorrage. Stroke 14:468-475, 1983
24. Passero S, Ulivelli M, Reale F: Primary intraventricular hemorrhage in adults. Acta Neurol Scand 105:115-119,2002
25. Pia HW: The diagnosis and treatment of intraventricular haemorrhage. In: Lujendijk W,ed. Progress in brain research. Amsterdam: Elsevier Pub. Co. 1968: 463-470
26. Sanders E: A study of primary, immediate, or direct hemorrhage into the ventricles of the brain. Am J Med Sci 82:85-128,1881
27. Steiner I, Gomori JM, Melamed E: The prognostic value of the CT scan in conservatively treated patients with intracerebral hematoma. Stroke 15:279-282,1984
28. Tembl J, Lago A, Baquero M, Blasco R: Hemorragia intraventricular primaria. Analisis de ocho casos. Rev Neurol 25:215-218,1997
29. Todo T, Usui M, Takakura K: Treatment of severe intraventricular hemorrhage by intraventricular infusion of urokinase. J Neurosurg 74:81-86,1991
30. van den Bergh R: The periventricular intracerebral blood supply. In: Meyer Js, Lechner H, Eichborn O, ed. Research on the cerebral circulation. Springfield III: Charles C Thomas 1969: 52-65
31. Verma A, Maheshwari MC, Bhargava S: Spontaneous intraventricular hemorrhage. J Neurol 234:233-236,1987
32. West CG, Forbes WS: Intraventricular blood without parenchymal clot following spontaneous subarachnoid haemorrhage. Neuroradiology 27:254-258, 1985
33. Yamamoto Y, Waga S: Persistent intraventricular hematoma following ruptured aneurysm. Surg Neurol 17:301-303,1982
34. Young WB, Lee KP, Pessin MS, Kwan ES, Rand WM, Caplan LR: Prognostic significance of ventricular blood in supratentorial haemorrhage: A volumetric study. Neurology 40:616-619,1990
35. Ziai WC, Triantaphyllopoulou A, Razumovsky AY, Hanley DF: Treatment of sympathomimetic induced intraventricular haemorrhage with intraventricular urokinase. J Stroke Cerebrovasc Dis 12:276-279,2003