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Impact of surgical evacuation on chronic Subdural Hematoma patients' cognitive functions outcome

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ABSTRACT

Background: Chronic subdural haematoma (CSDH) patients represent one of common disease in neurosurgical practice. Those patients may suffer from various disabilities including intellectual function decline with or without motor deficit. *Aim of the study*: to address the reversible cognitive function improvement after burr hole evacuation of CSDH. *Patients and Material:* This prospective study of observation 55 CSDH patients who admitted to neurosurgery department in Mansoura Emergency Hospital in the period between January 2017 till January 2019. It includes 42 male, 13 female; patient's age was ranging from 46-72 (mean age 67.1±4.6) years. Each patient cognitive performance was evaluated pre, postoperatively and after 8 weeks in Out Patient Clinic (OPC) by using MMSE. All cases had also pre and post-operative CT Head to document presence of CSDH. The surgical procedure for all cases was two burr holes evacuation. The data of clinical features, intellectual functions and images had been collected and analysed. *Results:*

The presenting manifestation was impairment in intellectual functions in 34 cases (62%). The sever persistent headache was presenting symptom in 21 patients (38.1%), hemi or mono paresis as presenting symptom was in 19 cases (34.5%), the dysphasia was present in 7 cases (12.7%) and seizures in 5 cases (9.1%).there is statistically significant in CSDH patient's cognitive performance improvement (p=0.00), headache (p=0.00), motor deficit (p=0.02) and speech dysphasia (p=0.38). *Conclusion:* The decline of intellectual functions represents the most common manifestation of CSDH patients in geriatric population which can be reversible after the simple surgical procedure. CSDH must be in deferential diagnosis of dementia in old age patients beside neurodegenerative disease. The neuro imaging is mandatory in such cases to eliminate presence of CSDH.

Keywords: Chronic subdural hematoma, cognitive impairment, burr hole, dementia, CSDH

1. INTRODUCTION

In recent years we can find increasing number of aging people across the general population. Their problems including dementia became important issue to the society for early diagnosis and better management. The definition of dementia in most literatures is a disease associated with acquired progressive loss of cognitive in addition to emotional capabilities that prevent the normal person from doing his daily functions & reducing the quality of his life. The prevalence of dementia among 60 years old is 1% and become 2% in over 65 years' old people (Clarfield 1988, Evans, 1989).

The aging people may present with decline in intellectual functions including progressive amnesia. These patients had a dementia. If these patients had also tremor or and motor deficit may be the diagnosis became Parkinson disease and Alzheimer disease with other manifestation of neurodegenerative diseases (Kostanian, 2000). In neuropsychiatric point of view these patients treated medically even in some cases without CT scan or MRI Brain imaging, but in neurosurgery point of view these cases may had: anterior frontobasal meningioma, normal pressure hydrocephalous and CSDH. These cases diagnosed clinically besides imaging studies which is the corner stone element in diagnosis. CSDH patients represent one of common disease in neurosurgical practice that had good prognosis if the treatment was adequate in timing and the procedures. CSDH incidence was about 0.001-0.002% annually. Nobuhiko 1990 suggested that in developing countries may reach 0.0074% annually in over 70 year's age group in general population as a result of increased life expectancy. CSDH may caused by minor trauma in brain atrophy patient that in most cases un recognised especially it happened several weeks to months prior to be manifested clinically (Oyama et al., 1998; Machulda & Haut, 2000).

Aim of our prospective study is to address the reversible cognitive function improvement after burr hole evacuation of CSDH. We will analyse also the predictors that affect the prognosis.

2. PATIENTS AND METHODS

This prospective study was observed in 55 CSDH patients who admitted to neurosurgery department in Mansoura University Emergency Hospital during the periods January 2017 to January 2019. Each patient cognitive performance was evaluated pre, postoperatively and after 8 weeks in Out Patient Clinic (OPC) by using Mini Mental state Examination (MMSE) which is fast and reliable as bedside examination.

MMSE test consist of five items: (a) Orientation with ten points, (b) Registration that had three points, (c) Calculation with five points in the score, (d) Recall with three points and (e) language that include naming, repetitioning addition to three stages commands. Those elements make the maximum score of MMSE of 30. The specificity & sensitivity of that test was 0.97 and 0.76 respectively. We adopt 23 in score or below to discriminate patients with dementia from healthy intellectual functions persons of besides recording other manifestations: severe headache, seizures, dysphasia, abnormal behaviour and weakness. All cases had also pre and post-operative CT Head to document presence of CSDH (Clarfield, 1988).

It appeared crescent-shaped iso or hypo dense in comparison to the brain parenchyma fluid collection. The surgical procedure for all cases was two burr holes evacuation. It represents the most accepted procedure for management of CSDH all over the world with low morbidity and mortality rate in such fragile population who had other comorbidity (Richter et al., 1984, Markwalder, 1998, Krupp & Jans, 1995). The frontal burr hole was done behind hair line in most thickened part of CSDH with curved skin incision while the posterior burr hole was applied in the parietal area in the most thickened part of the hematoma. We opened the dura gradually to avoid sudden reduce the intracranial pressure with subsequent hyper perfusion and developing intraparenchymal hematoma, we opened the dura in cruciate manner and coagulate it to prevent its closure then we did frequent wash with worm saline gently till become clear fluid. At the end we put a small catheter in the cavity of CSDH then anatomical closure in water sealed manner to avoid Pneomocephalous. The drain removal was after 3 to 5 days postoperatively. The data of clinical features, intellectual functions

(that had been assessed by MMSE exam) and images had been collected and analysed statistically (Foelholm & Waltimo, 1975; Cameron, 1984; Clarfield, 1988).

The study protocol was approved by The Local Ethical Committee. Patients signed written fully informed consent for study participation and undergoing the assigned investigations. Authors have declared that no competing interests existing and project has been approved from ethical committee of Mansoura Emergency Hospital at March 15, 2017 (Project ID No. M/37241/17). As per international standard or university standard, written consent has been collected from parent and preserved by the authors. None of the funding sources played any role in study design; in the collection, analysis, and interpretation of data; in the writing of the manuscript; or in the decision to submit the manuscript for publication.

3. RESULTS

It included 42 males (76.4%), 13 females (23.6%), patients age was ranging from 46-72(mean age 67.1±4.6) years. The presenting manifestation was impairment in intellectual functions in 34 cases (62%). The sever persistent headache was presenting symptom in 21 patients (38.1%), hemi or mono paresis as presenting symptom was in 19 cases (34.5%), the dysphasia was present in 7 cases (12.7%) and seizures in 5 cases (9.1%) (Table 1 & figure 1).

Table 1 Pre and post-operative manifestation of 55 cases with CSDH

Manifestation	Preoperative complaint	Postoperative improvement	Postoperative complaint
intellectual functions deficit	34 cases (62%).	23 patients (41.8%)	11 patients (20%)
Persistent headache	21 patients (38.1%)	14 cases (25.4%)	7 cases (12.7%)
Hemi or mono paresis	19 cases (34.5%)	15 patients (27.3%)	4 patients (7.3%)
Dysphasia	7 cases (12.7%)	3 cases (5.4%).	4 cases (7.3%)
seizures	5 cases (9.1%)	5 cases (9.1%)	0

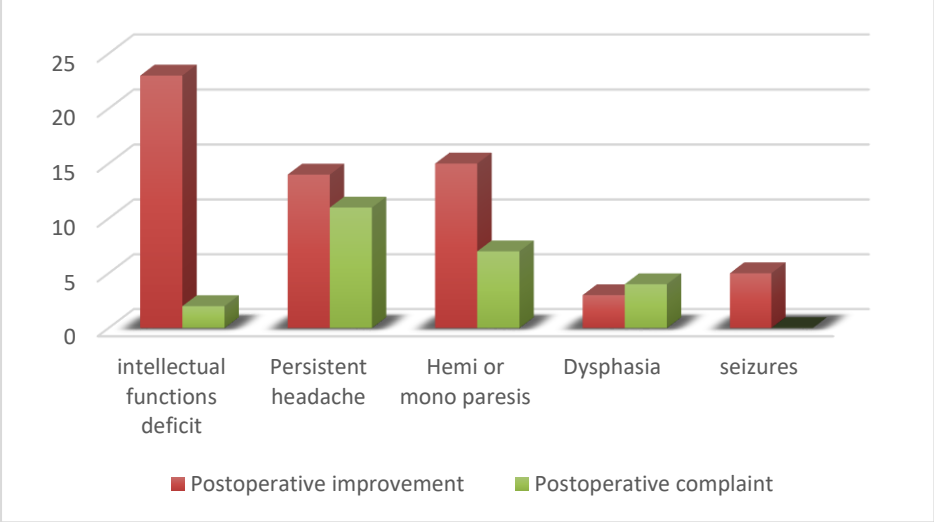

Figure 1 Pre and post-operative manifestation of 55 cases with CSDH

In our study we found the higher incidence of intellectual function declined in patient who was older than 65 years age in comparison to the patient who was 60 years or younger (Table 2 and fig. 2).

The thickness of CSDH"measured in CT Brain scan" did not significantly correlated with the degree of intellectual function deficit. Post operatively, the cognitive functions improved in 23 patients (67.6%), the Headache subsided in 14 cases (25.4%), hemiparesis improved in 15 patients (27.3%) while the dysphasia improved in only 3 cases (5.4%). The age was the corner stone as a predictor for post-operative reversibility of intellectual function. the group of patients who their age was younger than 60 years (62.7±5.3years) had better recovery of the dementia than the group who had older age (68.3±3.7years) ($p<0.05$) (Table 3).

Table 2 Preoperative manifestation in relation to the age

Manifestation	<60 years (20 cases)	>60 years (35 cases)	P - value
intellectual functions deficit	9 cases (45%).	25 patients (71.4%)	0.001
Persistent headache	16 patients (80%)	5 cases (14.3%)	0.001
Hemi or mono paresis	7 cases (35%)	12 patients (34.3%)	0.684
Dysphasia	3 cases (15%)	4 cases (11.4%)	0.594
seizures	2 cases (10%)	3 cases (8.6%)	0.835

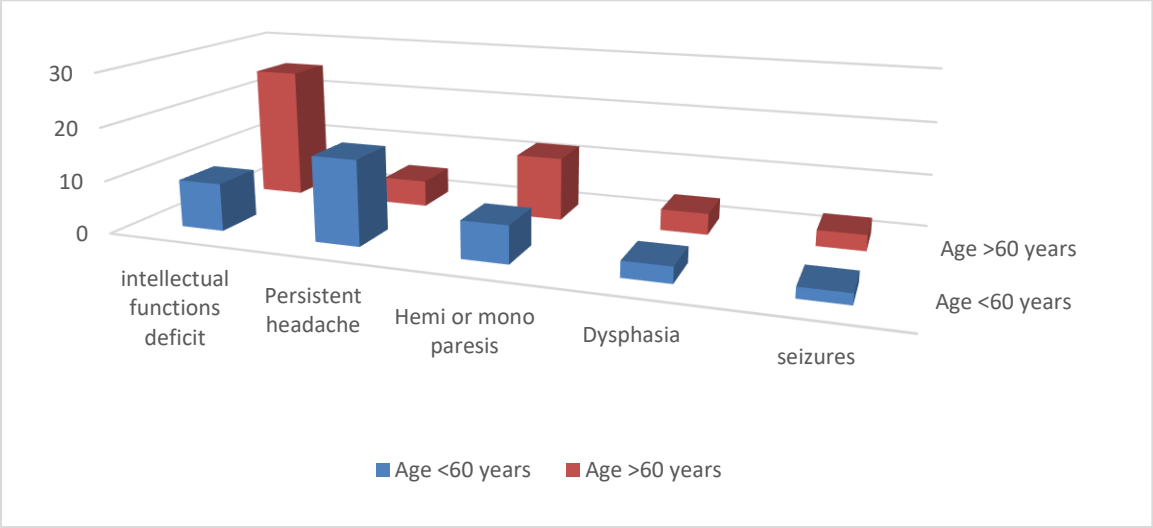

Figure 2 Preoperative manifestation in relation to the age

Table 3 The outcome of preoperative intellectual function deficit in relation to multiple factors

Factor	Improved group (23 cases)	Not improved (11cases)	P - value
Age	62.7±5.3	68.3±3.7	0.003
Side of hematoma			0.027
Left	11	5	
Right	7	4	
Both	5	2	
(CSDH) Thickness (CM)	2.7	2.9	0.41
MMSE	3 cases (15%)	4 cases (11.4%)	0.594

The preoperative MMSE was 15.4 in improved group while was 6.7 in the unchanged group. In recurrent cases there was no predictors of any kind to define the potentially those (Table 4). There is statistically significant in CSDH patient's cognitive performance improvement (p=0.00), headache (p=0.00), motor deficit (p=0.02) and speech dysphasia (p=0.38).

Table 4 Recurrent and non-recurrent (CSDH) groups in relation to age and preoperative CT Imaging

Factor	Recurrent group (23 cases)	Not recurrent (11cases)	P - value
Age	65.1±7.2	64.7±5.6	0.85
Side of hematoma			0.74
Left	3	22	
Right	2	19	
Both	1	8	
(CSDH) Thickness (CM)	2.3	2.1	0.54

4. DISCUSSION

As a result of more availability of medical tools, wider medical health coverage beside the improvement of living condition, the geriatric population and life expectancy increasing all over the globe. Thus, put larger group at risk of traumatic injuries. The older age group traumatic patients had worse prognosis regarding the mortality and morbidity than the younger one (Finelli et al., 1992; Osler et al., 1998). CSDH defined as blood collection in the space between the brain and the dura. It represents one of the diseases of geriatric population with average age 63 years old, its incidence triple fold in elderly (Levy et al., 1993). In other studies, the annual incidence in USA was 1.72 cases/100,000 population/year and in aged group (from 60 to 69) years, the incidence elevated to 8.4 cases/100,000 population/year (Kudo et al., 1992). It doesn't not have specific manifestations but include large varieties of symptoms and signs. These manifestations may be: altered consciousness, behaviour abnormalities, persistent severe headache & repeated vomiting as sequelae of raise intra cranial pressure (ICP), motor weakness in addition to dysphasia (Fogelholm et al., 1975). The most likely explanation consists of reduction in blood flow in certain brain region as a sequelae of intermittent vascular compression and shift because of brain swelling in addition to the effect of electrophysiological irregularities i.e. seizures & depression of normal cortical excitability (Moster et al., 1983; Black, 1985; Ikeda et al., 1990; Traynelis, 1991; Mishriki, 1999). CSDH can present with epilepsy with incidence about 4-6% that reduced after the surgical procedure to 2-3% (Luxon & Harrison, 1979; Cameron, 1984; Rubin & Rappaport, 1993). CSDH associated with high usage of health care giver (Ernst & Hay, 1994). Every age group showed more specific manifestations (younger patient showed symptoms & signs of (ICP) while the older patients showed motor deficit and intellectual functions deterioration). Our study revealed the exact concept (headache as the main presenting symptom in ≤60 years old patients whereas dementia and motor deficit in >60 years) (Fogelholm et al., 1975; Ishikawa et al., 2002). The explanation of reduced headache as presenting symptom in older age group with CSDH may be due to presence of brain atrophy that can give chance to accumulation of large mass before beginning of (ICP) elevation. The papill edema may absent also partially to the previous explanation in addition presence of optic atrophy in elderly people (Fogelholm et al., 1975; Cameron, 1984; Richter et al., 1984; Okada et al., 2002).

About 40% of CSDH patients had misdiagnosis due similarity of symptoms and signs related to other degenerative diseases of CNS, dementia, stroke or tumours and the overlooked history of trivial trauma (Kostanian et al., 2000). Beside the common cognitive capabilities among elderly population with limited literatures conclude the link between CSDH & intellectual functions deterioration. Dementia can be defined as deterioration of cognitive functions related to many nonspecific causes. More than 50 causes can lead to dementia (Clarfield, 1988; Clarfield, 1997). It may be permanent progressive and in some times may become reversible in correctable causes. Because of that identification of these reversible aetiologies is mandatory to get benefit from the previous concept. These reversible causes related to metabolic a neurological surgery correctable condition (Machulda & Haut, 2000), those disorders account about 1% of geriatric population (Yamamoto et al; 2003). CSDH patients represent one of these correctable conditions by simple surgical procedure that lead to reversibility of accompanied patients' dementia. In our series we conduct that over 62% of CSDH cases presented with cognitive function deterioration as the presenting manifestation that improved more than 70% in >60 years old group and this fact focus a light on CSDH as reversable cause of retaining intellectual function in many patients with reversable dementia .in many literatures we can find specific factors can determine the post-operative clinical conditions after the surgical procedure in CSDH patients with preoperative declined intellectual functions especially patients age, preoperative clinical status including the Activity of Daily Living (ADL) score like Ishikawa et al who mentioned in his series the younger than 74 years old, the ADL less than 5 and Mini-Mental Status Examination (MMSE) greater than 10 represented a good prognostic factors for recovery of the preoperative dementia (Ishikawa et al., 2002). Some scientific paper conducted the poor preoperative neurological condition, the long-time gap between precipitating head trauma, previous multiple brain infarct and the time of surgery as predictor for bad postoperative outcome while other studies deny this fact (Yamamoto et al., 2003; Gelabert-Gonzalez et al., 2005). In our series we verified that the link between reversibility of preoperative dementia and age beside MMSE status.

The recurrence of CSDH after surgery varies in many studies between 9.2% to 26.5% (Asano et al., 1992; Nakaguhi et al., 2001). In our study the recurrent rate was 10.9%. Many studies gave classification of features that associated with CSDH recurrence. The first class was related to the patient specific feature including old age, alcoholic administration and both related to brain atrophy (El-Kadi et al., 2000; Oishi et al., 2001; Yamamoto et al., 2003). The second group of factors related to imaging features: large CSDH mass, bilaterally presence, several thick membranes separate the hematoma in many compartments and various haemorrhage insult that lead to mixed density (Kostanian et al., 2000, Frati et al., 2004; Kim et al., 2005). The third group of risk factor related to the surgical technique like inadequate drainage of CSDH, rapid evacuation with massive brain re-expansion with hyper perfusion state, pneumocephalus (Okada et al., 2002). We failed in our study to reveal any risk factor for CSDH recurrence.

5. CONCLUSION

The decline of intellectual functions represents the most common manifestation of CSDH patients in geriatric population which can be reversable after the simple surgical procedure. CSDH must be in deferential diagnosis of dementia in old age patients beside neurodegenerative disease. The neuro imaging is mandatory in such cases to eliminate presence of CSDH.

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Conflicts of Interest: The authors declare no conflict of interest.

REFERENCE

1. Asano Y, Hasuo M, Takahashi I, Shimosawa S. Recurrent cases of chronic subdural hematoma-its clinical review and serial CT findings. No ToShinkei 1992; 44:827-831.
2. Black DW. Subdural hematoma: A retrospective study of the great neurologic imitator. Postgrad Med. 1985; 78:107-115.
3. Cameron MM. Chronic subdural hematoma: A review of 114 cases. J Neurol Neurosurg Psychiatry. 1984; 1:834-839.
4. Clarfield AM. The reversible dementias: do they reverse? Ann. Intern Med 1988; 109:476-486.
5. El-Kadi H, Miele VJ, Kaufman HH. Prognosis of chronic subdural hematomas. Neurosurg Clin N Am 2000; 11:553-567.
6. Ernst RL, Hay JW. The US economic and social costs of Alzheimer's disease revisited. Am J Public Health 1994; 84:1261-1264.
7. Evans DA, Funkenstein HH, Albert MS, Scherr PA, Cook NR, Chown MJ, et al. Prevalence of Alzheimer's disease in a community population of older persons. Higher than previously reported. JAMA, 1989; 262:2551-2556.
8. Finelli FC, Jonsson J, Champion HR, et al. A case control study for major trauma in geriatric patients. J Trauma. 1992; 9:541-548.
9. Foelholm R, Waltimo O. Epidemiology of chronic subdural haematoma. Acta Neurochir. 1975; 32:247-250.
10. Fogelholm R, Heiskanen O, Waltimo O. Chronic subdural hematoma in adults. Influence of patient's age on symptoms, signs, and thickness of hematoma. J Neurosurg 1975; 42:43-46.
11. Frati A, Salvati M, Mainiero F, Ippoliti F, Rocchi G, Raco A, et al. Inflammation markers and risk factors for recurrence in 35 patients with a posttraumatic chronic subdural hematoma: a prospective study. J Neurosurg 2004; 10:24-32.
12. Gelabert-Gonzalez M, Iglesias-Pais M, Garcia-Allut A, Martinez- Rumbo R. Chronic subdural haematoma: surgical treatment and out- come in 1000 cases. Clin Neurol Neurosurg 2005; 107:223-229.
13. Hiromichi Y, Yutaka H, Hideo H, Nakamasa H, Hideki O, Shunro E. Independent predictors of recurrence of chronic subdural hema- toma: results of multivariate analysis performed using a logistic regression model. J Neurosurg 2003; 98:1217-1221.
14. Ikeda K, Ito H, Yamashita J. Relation of regional cerebral blood flow to hemiparesis in chronic subdural hematoma. Surg Neurol. 1990; 33:87-95.
15. Ishikawa E, Yanaka K, Sugimoto K, Ayuzawa S, Nose T. Reversible dementia in patients with chronic subdural hematomas. J Neuro- surg 2002; 96:680-683.
16. Jeong CA, Kim TW, Park KH, Chi MP, Kim JO, Kim JC. Retrospective analysis of reoperated patients after chronic subdural hematoma surgery. J Korean Neurosurg Soc 2005; 38:116-120.
17. Kentoro M, Minoru M. Surgical treatment of chronic subdural hematoma in 500 consecutive cases: clinical characteristics, surgical outcome, complications, and recurrence rate. Neurol Med Chir (Tokyo) 2001; 41:371-381.
18. Kim HY, Kwon SC, Kim TH, Shin HS, Hwang YS, Park SK. Analysis of management according to CT findings in chronic subdural hematoma. J Korean Neurosurg Soc 2005; 37:96-100.
19. Kostanian V, Choi JC, Liker MA, Go JL, Zee CS. Computed tomographic characteristics of chronic subdural hematomas. Neu- rosurg Clin N Am 2000; 11:479-489.
20. Krupp WF, Jans PJ. Treatment of chronic subdural hematoma with Burr-hole craniostomy and closed drainage. Br J Neurosurg. 1995; 9:619-627.
21. Kudo H, Kuwamura K, Izawa I, et al. Chronic subdural hematoma in elderly people: Present status on Awaji Island and epidemiological prospect. Neurol Med Chir. (Tokyo) 1992; 32:207-209.
22. Levy DB, Hanlon DP, Townsend RN. Geriatric trauma. Clin Geriatr Med. 1993; 9:601-620.
23. Luxon LM, Harrison MJ. Chronic subdural hematoma. Q J Med. 1979; 48:43-53.
24. Machulda MM, Haut MW. Clinical features of chronic subdural hematoma: neuropsychiatric and neuropsychologic changes in pa- tients with chronic subdural hematoma. Neurosurg Clin N Am 2000; 11: 473-477.
25. Mark RI, Richard HS. Chronic subdural hematoma in adult and elderly patients. Neurosurg Clin N Am 2000; 11:447-454.
26. Markwalder T-M. Chronic subdural hematomas: A review. J Neurosurg. 1998; 154:637-645.

27. Mayeux R, Foster NL, Rossor M, Whitehouse PJ. The clinical eval- uation of patients with dementia, in Whitehouse PJ(ed): Dementia. Philadelphia: FA Davis, 1993; pp92-129.
28. MF, Folstein SE, McHugh PR. "Mini-mental state". A practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res 1975; 12:189-198.
29. Mishriki YY. Subdural hematoma mimicking a transient ischemic attack due to antihypertensive medication. South Med J.1999; 92:905-906.
30. Moster ML, Johnston DE, Retnmuth OM. Chronic subdural hematoma with transient neurological deficits: A review of 15 cases. Ann Neurol. 1983 Nov; 14(5):539-42.
31. Murakami H, Hirose Y, Sagoh M, Shimizu K, Kojima M, Gotoh K, et al. Why do chronic subdural hematomas continue to grow slowly and not coagulate? Role of thrombomodulin in the mechanism. J Neurosurg 2002; 96:877-884.
32. Nakaguhi H, Tanishima T, Yoshimasu N. Factors in the natural his- tory of chronic subdural hematomas that influence their postoperative recurrence. J Neurosurg 2001; 95:256-262.
33. Nobuhiko A. Chronic subdural haematoma in infancy. Clinical analysis of 30 cases in CT-era. J Neurosurg 1990; 73:201-205.
34. Oishi M, Toyama M, Tamatani S, Kitazawa T, Saito M. Clinical fac- tors of recurrent chronic subdural hematoma. Neurol Med Chir (Tokyo) 2001; 41:382-386.
35. Okada Y, Akai T, Okamoto K, Iida T, Takata H, Iizuka H. A comparative study of the treatment of chronic subdural hematoma burr hole drainage versus burr hole irrigation. Surg Neurol 2002; 57:405-409.
36. Osler T, Hales K, Baack B, et al. Trauma in the elderly. Am J Surg. 1988; 156:537-543.
37. Oyama H, Ueda M, Inoue S, Ikeda A, Shibuya M, Katsumata T, et al. [Improvement of cognition after trepanation for the chronic subdural hematoma]. No ToShinkei 1998; 50:249-252.
38. Richter HP, Klein HJ, Schafer M. Chronic subdural hematomas treated by enlarged Burr hole craniotomy and closed system drainage retrospective study of 120 patients. Acta Neurochir (Wien). 1984; 71:179-188.
39. Rubin G, Rappaport ZH. Epilepsy in chronic subdural hematoma. Acta Neurochir (Wien). 1993; 123:39-42.
40. Russell NA, Goumnerova L, Atack EA, et al. Chronic subdural hematoma mimicking transient ischemic attack. J Trauma. 1985; 25:1113-1114.
41. Traynelis VC. Chronic subdural hematoma in the elderly. Clin Geriatr Med 1991; 7:583-598.
42. Tsutsumi K, Maeda K, Iijima A, Usui M, Okada Y, Kirino T. The relationship of preoperative magnetic resonance imaging findings and closed system drainage in the recurrence of chronic subdural hematoma. J Neurosurg 1997; 87:870-875.
43. Weigel R, Schmiedek P, Krauss JK. Outcome of contemporary surgery for chronic subdural haematoma: evidence based review. J Neurol Neurosurg Psychiatry 2003; 74:937-943.
44. Yamamoto H, Hirashima Y, Hamada H, Hayashi N, Origasa H, Endo S. Independent predictors of recurrence of chronic subdural haematoma: results of multivariate analysis performed using a logistic regression model. J Neurosurg 2003; 98:1217-1221.