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Original Article

EPIDEMIOLOGY AND PROGNOSTIC FACTORS IN PATIENTS WITH SUBDURAL HEMATOMA

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Abstract. *SDH was first described in 1658 and in 1914 trauma was recognized as one of the causes. Acute SDH is more common in younger population while chronic is common in the elderly with the peak of incidence of 7.35/100000 per year in the age group 70-79 years. Trauma is one of the main causes of SDH although in 30-50% of patients direct trauma to the head can be omitted. Other predisposing factors include: anticoagulant therapy, epilepsy, and hypertension. The aim of the study was to determine risk factors and prognostic factors for the occurrence of SDH, also to show what age group is most at risk for developing chronic and acute SDH. Identify the diagnostic steps in proving SDH and the best method of treatment. The study includes 267 patients treated in the period from 1.1.2019 to 31.12.2019 at the Clinic for Neurosurgery CCS. SDH was diagnosed by neurological examination and brain CT in all patients and all were treated conservatively or surgically. The analytical statistics were used parametric and non-parametric tests of difference. The study included 185 men and 82 women of middle age 68 ± 17.19 years. Most patients were between 6-8 decades. Multiple changes in CT were observed in 63.3% of patients. Chronic SDH had 50.5% of patients and 45.6% had acute SDH. The most common symptoms were headache, psycho-organic syndrome, and hemiparesis. Patients with a GCS score of more than 8 had a better prognosis and outcome. Surgical treatment was the main course of treatment in our study. CT with / without contrast is the gold standard in detecting SDH. Men are at higher risk for the occurrence of SDH. Symptoms can occur later in the clinical presentation so we need to take caution when performing neurological examination. Factors that can lead us to suspect possible SDH are: age, gender, type of injury, clinical presentation, and time of occurrence.*

Key words: *subdural hematoma, trauma, headache, CT, anticoagulation, hemiparesis.*

Introduction

Chronic subdural hematoma (cSDH) is an encapsulated collection of old blood, mainly or totally liquefied and localized between the dura mater and the arachnoid. It was first described by J.J. Wepfer in 1658 and then found himself in the popular novel Pierette written by Honore de Balzac in 1840. Balzac described the traumatic origin and surgical treatment of these hematomas even though they had not yet been recognized as a separate clinical entity by physicians. This view was challenged by Virchow in 1857. giving his term "pachymeningitis hemorrhagica intern" to describe this disease, considering it to be of inflammatory origin. It wasn't until 1914. traumatic origin of subdural hematomas firmly established by Trotter, who gave them the name "subdural hemorrhagic cysts". Hulke described the first successful operation of a chronic subdural hematoma in 1883. In the era before computed tomography (CT) diagnosis was made by angiography or diagnostic trepanation. The introduction of CT diagnostics has set the gold standard in detecting SDH. SDH is a dynamic lesion and its presentation depends on age, shortly after bleeding (acute phase) it is hyperdense due to the presence of fresh blood, after a few weeks (subacute phase) isodensate due to fibrinolysis and after 4 weeks (chronic phase) hypodense due to fluid resorption. Chronic and acute SDH have different presentations on brain CT scan (Figure 1).

Acute SDH generally occurs in the younger population, after severe trauma, followed by structural brain damage, and is presented within 72 hours. In contrast, chronic SDHs often occur in the elderly population after a trivial injury, without any brain injury, and usually take weeks or months before becoming clinically evident. The incidence peak of cSDH is in the sixth and seventh decades of life. The incidence is estimated to be 1.72 / 100,000 per year, rising rapidly with aging to 7.35 / 100,000 per year in the 70-79 age group [1-22]. Other studies have shown there is a rising incidence of chronic subdural hematomas because there are more people in older populations. It has long been known that subdural hematomas are more likely to develop in the elderly even after minor traumas. Generalized cerebral atrophy and increased vein fragility associated with aging are major risk factors. With aging, there is a decrease in brain mass and an increase in space between the brain and skull bones from 6% to 11% of total intracranial space, which leads to vein stretching and increased brain movement within the cranium [1-11, 15-29].

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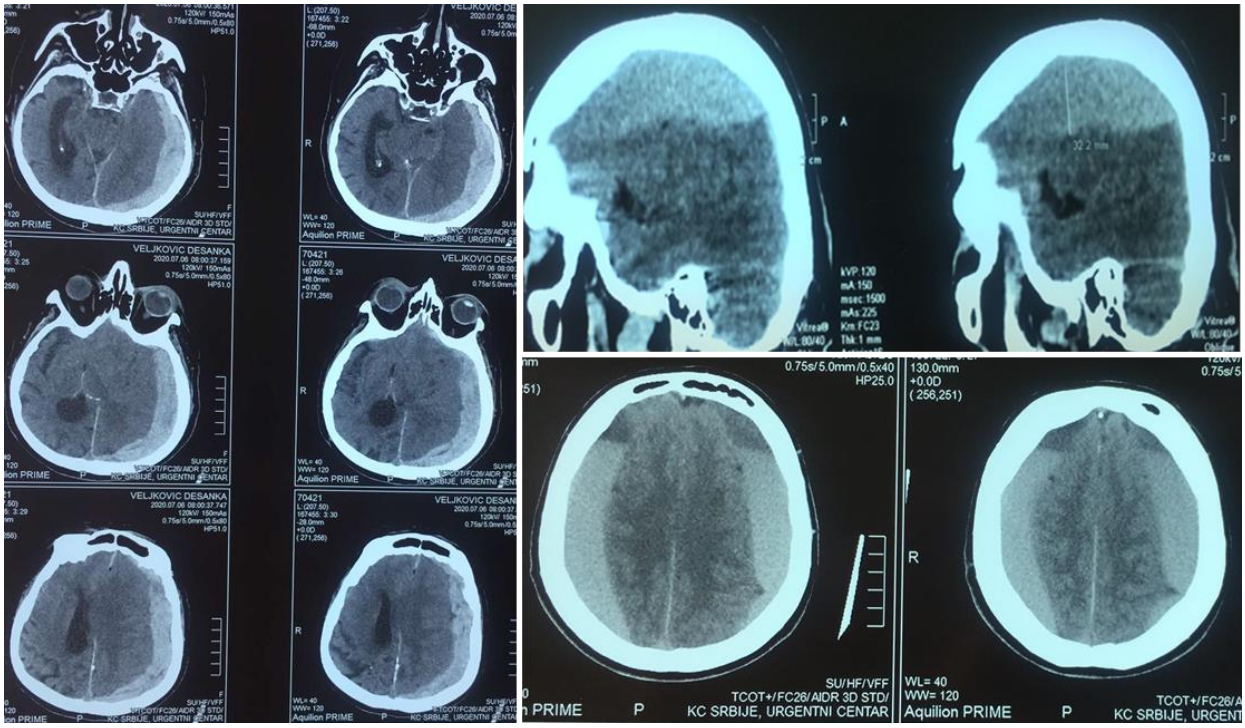


Fig. 1 Acute and chronic subdural hematoma

Trauma is one of the most important risk factors. However, direct head trauma may be absent in 30-50% of cSDH cases. Attention should be paid to indirect head injuries as well as trivial injuries that patients often forget to mention. Other predisposing factors are anticoagulant therapy, epilepsy, alcoholism, hypertension, etc. In the elderly, the most common clinical presentation is in the form of mental disorders (50-70%). It can manifest as varying degrees of state of consciousness. The diagnosis can be overlooked in psychiatric or neurological patients in whom any change in behavior or functional status is usually attributed to their pre-existing illness [1-5, 9-17, 23-33]. Hemiparesis can be found in about 58% of cases in some studies [1-7, 17-26]. Limb weakness is usually mild and contralateral, although cases of ipsilateral symptoms have been seen. The incidence of headache varies from 14% to 80% in different studies [1-3, 7-19, 29-35]. The first reason why headache is less common in the elderly compared to younger patients is partly due to increased intracranial space for hematoma accommodation [1-14].

The second reason is that confusion first occurs in the elderly so they report to the doctor before a headache occurs. Another common symptom is syncope, which occurs in about 74% of cases. Epilepsy has traditionally been considered a rare presentation, although it was detected in as many as 6% of cases as an initial symptom. We often do not suspect the diagnosis of SDH at the time of the initial presentation in most cases. Other possible diagnoses at the time of presentation are tumor, subarachnoid hemorrhage, and stroke. The most important step in the diagnosis is SDH is a high index of suspicion. It should be considered in a patient with/without a history of head injury presenting:

- Changes in mental status or worsening of a pre-existing neurological or psychiatric deficit,
- Focal neurological deficit,
- Headache with or without focal neurological deficit.

SDH treatment is surgical evacuation, although small hematomas can be withdrawn spontaneously or with conservative therapy, but patients should be closely monitored. The most common surgical complications are symptomatic recurrences of hematoma (8-37%) and epi-seizures (about 11%), while mortality and morbidity (15.6%) vary depending on the literature [1-9, 13-19, 24-26, 36-38].

Materials and Methods

This study includes a retrospective review of a database of patients (267 patients) with verified acute and chronic subdural hematoma received in the period 1/1/2019 to 31/12/2019 at the Emergency Center in Belgrade and treated at the neurosurgical department. The analysis included patients who met the following conditions:

1. Clinically, by examination and CT scan verified the presence of SDH,
2. Treated conservatively or surgically

The analysis used:

1. Complete medical histories,
2. Findings obtained by computed tomography (CT),
3. Glasgow coma and outcome scales (GCS, GOS)

Parametric (t-test) and non-parametric difference tests (χ^2 and Median test) were used in analytical statistics. Parametric tests were applied to examine the differences and interactions of observation features in which the condition for performing a parametric test was met. Non-parametric tests were applied to observation fea-

tures in which the conditions for performing analog parametric tests were not met, as well as to observation features that are naturally analyzed by these tests.

Results

Our database contained 267 patients of both gender, 185 males (69.3%) and 82 females (30.7%). A total is 267 patients, with a mean age of 68.35 ± 17.19 years. The youngest patient was 6 months old and the oldest 97 years old. The median age in the observed group was 73. There was a statistically significant difference in favor of the male sex (p <0.001). Most patients were between 60 and 80 years of age, with an incidence peak between 7 and 8 decades. A statistically significant difference in age concerning sex can be observed (p<0.001). The distribution by sex and age is shown in Figures 2 and 3.

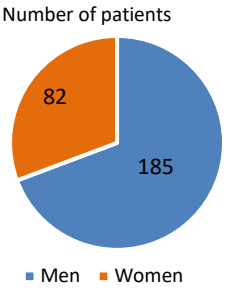


Fig. 2 Patient distribution by gender

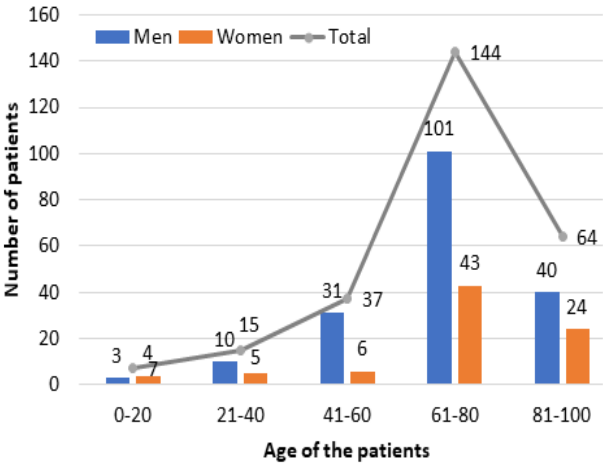


Fig. 3 Patients age distribution

With regard to the manner in which SDH occurred, patients were divided into three groups:

1) SDH resulting from a head impact by blunt force trauma, fall, or other direct head impact,
2) SDH resulting from direct head impact in a traffic accident, and
3) spontaneous SDH.

The distribution of patients concerning the onset of SDH can be seen in Figure4. A statistically significant difference in the benefit of patients in the first group was observed (p <0.001). At admission, there were 7 patients (2.7%) with polytrauma while the rest were only with/without head injury. Multiple changes in CT were observed in 169 patients (63.3%). These changes were in the form of head bone fractures (20.2%), brain contusion (25.84%), epidural hematoma (2.6%), subarachnoid hemorrhage (13.1%), and intracerebral hemorrhage (1.4%) (Figure 5). A highly statistically significant difference in the occurrence of contusions together with SDH during head injury was observed in this group of patients. Based on the findings on CT, we divided in terms of localization, time of onset of SDH, and displacement of central structures, which can be seen in Figures 6 and 7. Chronic SDH had 50.5% of our patients, while acute had 45.6% of patients. Above the left hemisphere, SDH had 45%, over the right hemisphere 39% and over both hemispheres had 16% of patients. In our study, we observed more patients with left-sided hematoma occurrence without compressive effect and displacement of central structures, as well as more patients with chronic SDH at admission (p <0.001).

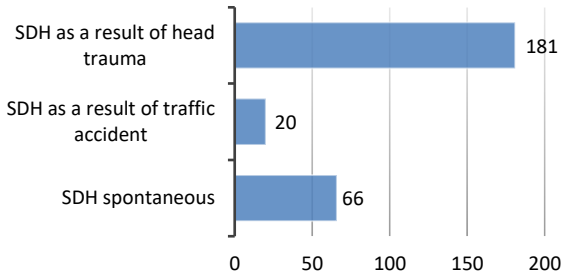


Fig. 4 Patients distribution regarding the mechanism of SDH formation

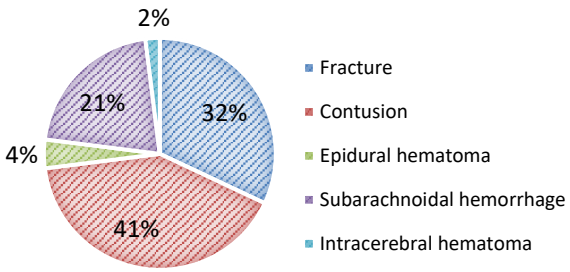


Fig. 5 Most common brain injuries associated with SDH

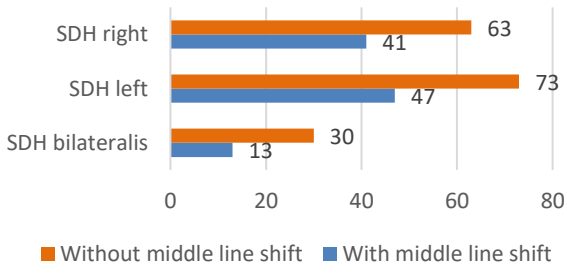


Fig. 6 SDH localization with/without compression and midline displacement

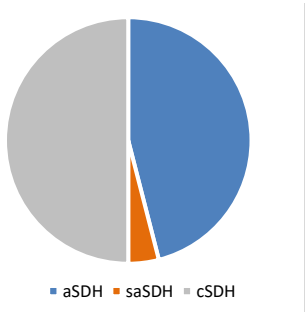


Fig. 7 Type of SDH versus time of onset in our study

Table 1 Symptom presentation of admitted patients in our study

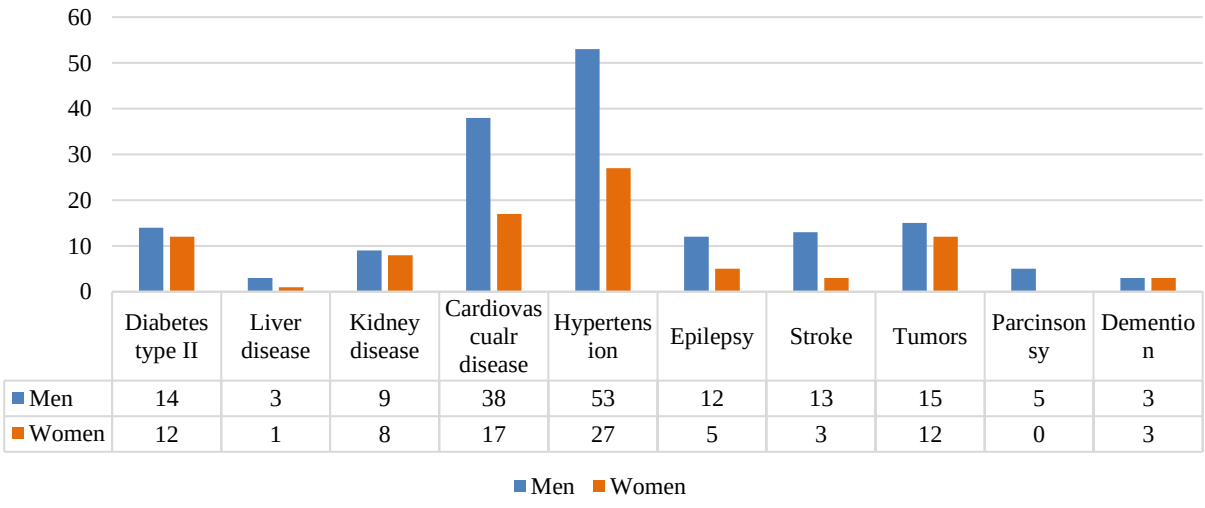
Distribution of symptoms admitted in patients in our study	
Cranial nerves	
Normal	166
Deficit	58
Palsy of n. VII on the right side	15
Palsy of the n. VII on the left side	10
Palsy of the n. III	1
Extremities	
No deficit	124
Decerebration	41
Patient with right sided hemiparesis	56
Patient with left sided hemiparesis	44
Bihemiparesis	2
Different types of hemiparesis at admission:	
the left-sided weakness of mild grade	33
the left-sided weakness of moderate severity	4
the left-sided weakness of severe grade	7
the right-sided weakness of mild grade	38
the right-sided weakness of moderate severity	11
the right-sided weakness of severe grade	7
Headache	86
Nausea	61
Vomiting	24
Psychoorganic syndrome	66
Syncope	35

The clinical presentation is manifested with limb weakness, psychoorganic syndrome, cranial nerve disorder, or syncope. The most common symptoms were headache, psychoorganic syndrome, and hemiparesis. Most patients on admission showed no signs of failure on cranial nerves or limbs ($p<0.001$). Table 1 shows the most common symptoms in our study.

The predisposing factors mentioned in the introduction may play an important role in developing SDH after trauma. As more than 50% of patients over 50 were expected to have associated comorbidities: diabetes, hypertension, epilepsy, dementia, stroke, etc. We observed that about 30% of patients had different types of cardiovascular disease and hypertension. Figure 8 shows the distribution of patients with their comorbidities by gender.

According to the protocol defined for these patient conditions, the Glasgow Coma Score was determined at the time of admission, and the conditions during discharge were determined by the Glasgow Outcome Scale. Based on the number of GCS points on admission, patients were divided into two groups: those with less and those with a score greater than 8, and in relation to the outcome on discharge in five groups. As expected, patients with a GCS score greater than 8 had a better prognosis and the best possible outcome ($p <0.001$). High statistical significance was shown in relation to the outcome in favor of men ($p <0.001$). Tables 2 and 3 show the ratio of GCS and GOS score in relation to gender and age group. Besides the GCS scores on admission, a previous use of anticoagulant therapy also influenced the outcome of the patients. Among our patients, 62 had received anticoagulant therapy prior to admission, and the study did not show statistical significance for the benefit of patients without anticoagulant therapy in terms of outcome (Figure 9).

After assessing the patient's condition, 173 patients (65%) underwent surgical treatment while 94 (35%) patients were treated with conservative therapy (Figure10). Twice as many more men had a heamatoma for surgery than females. We decided on surgical treatment in most of our patients ($p <0.001$). In our study operated patients



	Diabetes type II	Liver disease	Kidney disease	Cardiovascular disease	Hypertension	Epilepsy	Stroke	Tumors	Parcinsonsy	Dementio n
Men	14	3	9	38	53	12	13	15	5	3
Women	12	1	8	17	27	5	3	12	0	3

Fig. 8 Distribution of comorbidities according to gender

were shown to have a better treatment outcome than patients treated conservatively (p <0.001) (Figure 11).

Table 2 GCS and GOS scores by age group in our study

Distribution of the GCS and GOS score considering the age						
	GCS<9	GCS>9				
0-20	0	7				
21-40	3	12				
41-60	13	24				
61-80	30	114				
81-100	16	48				
	GOS 1	GOS 2	GOS 3	GOS 4	GOS 5	
0-20	0	1	0	0	6	
21-40	3	0	1	0	11	
41-60	10	2	2	1	22	
61-80	34	7	16	6	81	
81-100	18	9	7	3	27	

Table 3 GCS and GOS scores relative to gender in our study

GCS and GOS score considering gender					
	GCS<9	GCS >8			
M	36	149			
W	26	56			
	GOS 1	GOS 2	GOS 3	GOS 4	GOS 5
M	38	9	19	8	111
W	27	10	7	2	36

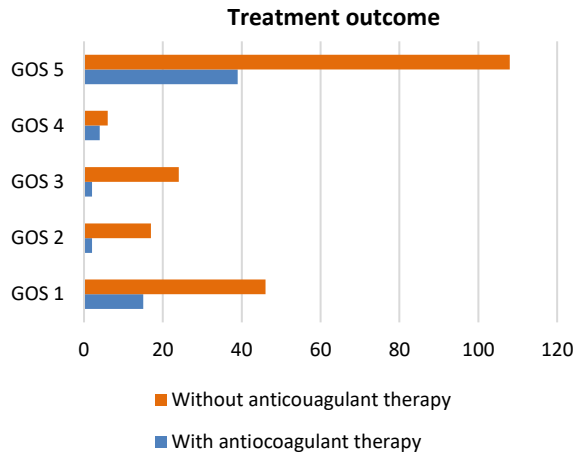


Fig. 9 Treatment outcomes in patients with and without anticoagulant therapy

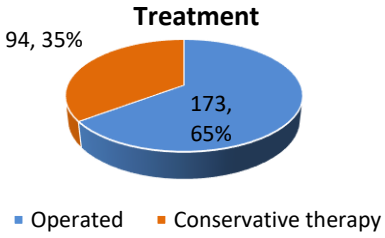


Fig. 10 Distribution of operated patients and patients treated with conservative therapy

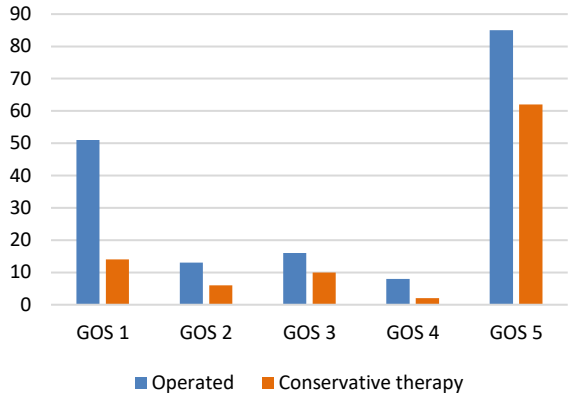


Fig. 11 Treatment outcome of patients treated with surgical and conservative therapy

Discussion

Our group consisted of 185 (69%) men and 82 (30.7%) women, which tells us that SDH occurs 2.25 times more frequently in men. Similar studies conducted recently show that the occurrence of SDH is 3 times more common in men. In the study it was shown that in 56% of cases patients were from age groups from 5 to 6 decades; another study states that more than 50% of cases were from a group of patients over 60 years of age. Our study showed that the majority of patients are consisted of people between the ages of 6 and 8 decades, which are consistet with different studies. Acute SDH has been shown to occur in 5-25% after severe head trauma depending on the study. Men were also shown to have a higher incidence of chronic SDH. In our study, it was shown that 1.44 times more acute SDH was frequent in men than women, and 4 times more chronic SDH was frequent in men than women. When we talk about literature, it was discrabed 1.3 to 2 for acute SDH in men, and 3 to 5 times more in men for chronic SDH.

Mortality is less than 20% for those under 40, about 65% for the 40-80 age group, and patients over 80 have the highest mortality [1, 4, 7, 9, 15, 21, 26, 29, 33, 36, 38]. Our study found mortality of less than 30% both of acute and chronic SDH. Causes of SDH are head trauma, coagulopathy or anticoagulant therapy, spontaneous, etc [1-5, 7-13, 16-28]. Trauma plays a major role in the onset of acute SDH. They are most injured in crashes, violence, or motor accidents [1-9, 23-33]. When it comes to chronic SDH, about 50% of patients do not have a head injury, and if they do, it is usually a minor injury. In 25% of patients who have had a head injury, symptoms of chronic SDH occur within 1-4 weeks, in the other 25% between 5 weeks and 3 months and only 1/3 of patients have no asymptomatic period [1-14].

Proven risk factors for developing SDH are: alcoholism, epilepsy, anticoagulant therapy, cardiovascular disease, diabetes mellitus, etc [1-7, 9-14].The literature and different studies found that 10-25% of patients are on Aspirin therapy, while in our study, Aspirin and/or anticoagulant therapy was used by 23% of patients [3-5, 11-16,

22-27]. In our study, the most important factors for cardiovascular problems and hypertension were singled out, which can be explained by the fact that the majority of patients were elderly. It is a well-known fact that clinical presentation is dominated by headache, confusion, hemiparesis, disorders of consciousness, while to a lesser extent weakness, seizures and incontinence occur.

Headache occur in 90% and confusion in 56% of cases, while in 75% of cases, headache is accompanied by nausea and vomiting [1, 4, 6, 9, 12, 16, 19, 20, 22, 24, 28, 30]. Hemiparesis and syncope occur in approximately 58% and 40% of cases, respectively. Hemiparesis is ipsilateral in 40% of cases [10-18, 22, 25, 31, 32]. In our study, the dominant clinical signs of SDH occurred to a lesser extent, which may be explained by the rapid arrival of patients after trauma before the symptoms could be developed. On admission, 205 patients (76.7%) had a GCS score greater than 8, while 62 (23.2%) patients had a score of less than 9. It has been shown in various studies that one of the most important prognostic factors for treatment outcome is certainly a GCS score [1-9, 25-33]. This is supported by the fact that as many as 82% of patients with a score less than 8 from our study died, while in the group of patients with a score greater than 8 that percentage was less than 1. Seventy one percent of our patients had complete recovery (GOS 5). The better treatment outcome in our study was experienced by men, which can be explained by their majority share in our study. Surgical treatment is taken as the main choice of treatment (65% patient in our study), and 35% of patients was treated with conservative therapy in our study but other studies have shown that around 23% of patients did not undergo surgery because the size of the hematoma was small [2-7, 15-23, 34-38]. In almost all studies, the operative approach is considered to be the main form of therapy for patients with SDH associated with midline shift, while conservative therapy is reserved for patients who are asymptomatic and with a small hematoma on brain CT [9-12, 24, 27, 33, 37].

Conclusion

Since the introduction of CT diagnostics, detection of both acute and chronic SDHs has progressed significantly and is now considered the gold standard in the detection of SDH. Men are at higher risk for developing SDH due to both lifestyle and associated comorbidities. Trauma has been shown to be one of the most common causes of SDH especially acute in the younger population, so care should be taken when screening as symptoms can often occur later. Efforts should be made to educate medical professionals because the diagnosis of SDH is first made with suspicion and only then with diagnostic procedures. Significant factors for suspecting a possible diagnosis of SDH are age, gender, type of injury, clinical presentation, and time of developing symptoms. In the population group between 6 and 8 decades, the most common is chronic, while in the younger age group 40, acute SDH is more common. The most common symptoms are headache, confusion, hemiparesis, state of consciousness. Surgical treatment is certainly an option of choice except in patients with very small hematoma and asymptomatic clinical imaging.

References

1. Adhiyaman V, Asghar M, Ganeshram KN, Bhowmick BK. Chronic subdural hematoma in the elderly. Postgrad Med J 2002; 78:71–75
2. Fogelholm R, Waltimo O. Epidemiology of chronic subdural haematoma. Acta Neurochir 1975; 32:247–250.
3. Ellis GL. Subdural haematoma in the elderly. Emerg Med Clin North Am 1990; 8:281–294.
4. Jones S, Kafetz K. A prospective study of chronic subdural haematomas in elderly patients. Age Ageing 1999; 28:519–521
5. Rozzelle CJ, Wofford JL, Branch CL. Predictors of hospital mortality in older patients with subdural haematoma. J Am Geriatr Soc 1995; 43:240–244.
6. Traynelis VC. Chronic subdural haematoma in the elderly. Clin Geriatr Med 1991; 7:583-598.
7. Luxon LM, Harrison MJG. Chronic subdural haematoma. Q J Med 1979; 189:43–53.
8. Cameron MM. Chronic subdural haematoma: a review of 114 cases. J Neurol Neurosurg Psychiatry 1978; 41:834–839.
9. Mulligan P, Raore B, Liu S, Olson JJ. Neurological and functional outcomes of subdural hematoma evacuation in patients over 70 years of age. J Neurosci Rural Pract 2013; 4:250–256.
10. O'Phelan K. Traumatic Brain Injury, Definitions and Nomenclature. In: Zollman FS, ed. Manual of Traumatic Brain Injury Management. Chicago, Illinois: Demos Medical Publishing; 2011. p. 3–9.
11. Lombard LA. Characterization of Traumatic Brain Injury Severity. In: Zollman FS, ed. Manual of Traumatic Brain Injury Management. Chicago, Illinois: Demos Medical Publishing; 2011. p. 16–22.
12. Alexander DN. Imaging in Moderate to Severe Traumatic Brain In: Zollman FS, ed. Manual of Traumatic Brain Injury Management. Chicago, Illinois: Demos Medical Publishing; 2011. p. 157–165.
13. Rosenow JM. Neurosurgical Management of Skull Fractures and Intracranial Hemorrhage In: Zollman FS, ed. Manual of Traumatic Brain Injury Management. Chicago, Illinois: Demos Medical Publishing; 2011. p. 166–174.
14. Narayan RK. Head Injury In: Grossman RG, ed. Principles of Neurosurgery. New York: Raven Press, Ltd; 1991. p. 235–286.
15. van Havenbergh T, van Calenbergh F, Goffin J, Plets C. Outcome of chronic subdural haematoma: analysis of prognostic factors. Br J Neurosurg 1996; 10:35–39.
16. Kotwica Z, Brzezinski J. Acute subdural haematoma in adults: an analysis of outcome in comatose patients. Acta Neurochir (Wien) 1993; 121:95–99.
17. Morinaga K, Matsumoto Y, Hayashi S, et al. [Subacute subdural hematoma: findings in CT, MRI and operations and review of onset mechanism]. No Shinkei Gek 1995; 23:213–216. Japanese.
18. Matsuyama T, Shimomura T, Okumura Y, Sakaki T. Rapid resolution of symptomatic acute subdural hematoma: a case report. Surg Neurol 1997; 48:193–196.
19. Gennarelli TA, Thibault LE. Biomechanics of acute subdural hematoma. J Trauma 1982; 22:680–686.
20. Foelholm R, Waltimo O. Epidemiology of chronic subdural haematoma. Acta Neurochir (Wien) 1975; 32:247–250.
21. Servadei F. Prognostic factors in severely head-injured adult patients with acute subdural haematoma's. Acta Neurochir (Wien). 1997; 139:279–285.

22. Massaro F, Lanotte M, Faccani G, Triolo C. One hundred and twenty-seven cases of acute subdural haematoma operated on. Correlation between CT scan findings and outcome. Acta Neurochir (Wien) 1996; 138:185–191.
23. Phuenpathom N, Choomuang M, Ratanalert S. Outcome and outcome prediction in acute subdural hematoma. Surg Neurol 1993; 40:22–25.
24. Kapsalaki EZ, Machinis TG, Robinson JS 3rd, Newman B, Grigorian AA, Fountas KN. Spontaneous resolution of acute cranial subdural hematomas. Clin Neurol Neurosurg 2007; 109: 287–291.
25. Mori K, Maeda 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.
26. Stanisic M, Lund-Johansen M, Mahesparan R. Treatment of chronic subdural hematoma by burr-hole craniotomy in adults: influenza of some factors on postoperative recurrence. Acta Neurochir (Wien) 2005; 147:1249–1256; discussion 1256–1257.
27. Suman S, Meenakshisundaram S, Woodhouse P. Bilateral chronic subdural haematoma: a reversible cause of parkinsonism. J R Soc Med 2006; 99: 91–92.
28. Giray S, Sarica FB, Sen O, Kizilkilic O. Parkinsonian syndrome associated with subacute subdural haematoma and its effective surgical treatment: a case report. Neurol Neurochir Pol 2009; 43: 289–292.
29. Luxon LM, Harrison MJ. Chronic subdural haematoma. Q J Med 1979; 48:43–53.
30. Stein SC, Young GS, Talucci RC, et al. Delayed brain injury after head trauma: significance of coagulopathy. Neurosurgery 1992; 30:160–165.
31. Wilms G, Marchal G, Geusens E, et al. Isodense subdural hematomas on CT: MRI findings. Neuroradiology 1992; 34:497–499.
32. Brain Trauma Foundation, AANS, Joint Section of Neurotrauma and Critical Care. Guidelines for the management of severe head injury. J Neurotrauma 1996; 13:641–734.
33. Wong CW. Criteria for conservative treatment of supratentorial acute subdural hematomas. Acta Neurochir (Wien) 1995; 135: 38–43.
34. Ducruet AF, Grobelny BT, Zacharia BE, et al. The surgical management of chronic subdural hematoma. Neurosurg Rev 2012; 35: 155–169; discussion 169.
35. Lega BC, Danish SF, Malhotra NR, Sonnad SS, Stein SC. Choosing the best surgery for chronic subdural hematoma: a decision analysis. J Neurosurg 2010; 113:615–621.
36. Chesnut RM, Marshall LF, Klauber MR, et al. The role of secondary brain injury in determining the outcome of severe head injury. J Trauma 1993; 34:216–222.
37. Itshayek E, Rosenthal G, Fraifeld S, Perez-Sanchez X, Cohen JE, Spector S. Delayed posttraumatic acute subdural hematoma in elderly patients on anticoagulation. Neurosurgery 2006; 58: E851–856.
38. Stroobandt G, Fransen P, Thauvoy C, Menard E. Pathogenetic factors in chronic subdural hematoma and causes of recurrence after drainage. Acta Neurochir (Wien) 1995; 137:6.