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# Papilledema in chronic subdural hematoma: how often it is seen and what has changed?

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***Background:*** *A chronic subdural hematoma (CSDH) is defined as an encapsulated accumulation of a clot of blood and degraded blood products on the surface of the brain between the dural and arachnoid membranes. Papilledema had for a long time been referred to as a characteristic objective symptom of CSDH. The incidence of papilledema is however, variable and depends on a variety of factors.*
***Purpose:*** *To analyze the data on the diagnosis of papilledema in patients with CSDH and to review the place of papilledema among the clinical findings in CSDH.*
*Material and Methods: We retrospectively reviewed the medical records of 164 patients with CSDH who underwent treatment at the Romodanov Neurosurgery Institute and Zaporizhzhia City Clinical Emergency Care Hospital from 2011 through 2021. Patients underwent clinical-and-neurological, ophthalmological, and neuroimaging examinations.*
***Results:*** *Papilledema was found in 4 (2.4%) of the patients. All these four patients had moderate papilledema which regressed after surgery. Details of these cases were reviewed.*
***Conclusion:*** *A shift in the peak incidence of CSDH towards an older age group and an increasing use of neuroimaging modalities exert an impact on the incidence of papilledema in patients with CSDH. Papilledema was found in 2.4% of the patients of the study.*

## Introduction

A chronic subdural hematoma (CSDH) is defined as an encapsulated accumulation of a clot of blood and degraded blood products on the surface of the brain between the dural and arachnoid membranes. The prevalence in a population has been reported to depend on the susceptibility, and CSDH is more commonly encountered in the elderly population [1]. Bartek and colleagues [2] have reported the crude incidence rates of CSDH for patients of 18-49 years, 50-66 years and 80-89 years to be 0.6, 7.1, and 59.5 CSDHs per 100,000 populations, respectively. With an increase in the population age and the number of elderly patients, CSDH has become a common condition in neurosurgical practice [3], and, according to a study by US researchers [4], CSDH is projected to become the most common cranial neurosurgical condition among adults by the year 2030.

Papilledema had for a long time been referred to as a characteristic objective symptom of CSDH. Neurosurgeons believed the presence of papilledema to be a criterion of subdural hematoma severity and an indicator of requirement for surgery. It has been reported, however, that the incidence of papilledema is variable and depends on a variety of factors. We suppose that today, given the active use of neuroimaging in current neurosurgical and neurological practice, more patients receive early treatment before papilledema occurs, and the incidence of papilledema is significantly lower than it was in recent decades. In addition, the incidence of papilledema varies among age groups. Until now, this question has been given little attention from the point of view of ophthalmological assessment.

**The purpose** of this study was to analyze the data on the diagnosis of papilledema in patients with CSDH and to review the place of papilledema among the clinical findings in CSDH.

## Material and Methods

We retrospectively reviewed the medical records of 164 patients with CSDH who underwent treatment at the Romodanov Neurosurgery Institute from 2011 through 2021. Sixty nine percent were men and 31% were women. Patient age ranged from 38 to 80 years, with a mean ± standard deviation of 55.5 ± 2.07 years.

Patients underwent clinical-and-neurological, ophthalmological, and neuroimaging examinations. Neuroimaging studies included magnetic resonance imaging (MRI) and computed tomography (CT). The severity of the headache was assessed on a ten-point scale (1-3, mild pain; 4-6, moderate pain; 7-8, severe pain; and 9-10, very severe pain). The neuro-ophthalmological examinationincluded included visual acuity, biomicroscopy, perimetry, and direct and indirect ophthalmoscopy. Patients with satisfactory general health status underwent automated Humphrey perimetry, whereas bed patients received Donders' test. The stage of papilledema was assessed as per the Tron's classification (as modified by Eliseeva) and papilledema was graded using the Frisén Scale [5-7]. Papilledema was classified as early, moderate, marked, regressive stage or secondary atrophy, which corresponded to Frisén Scale stages I to V. Early papilledema is characterized by swollen and blurred optic disc margin and hyperemic retinal veins. Moderate papilledema is characterized by swollen and hyperemic optic disc, vessel engorgement, and punctate hemorrhages in the peripapillary area. Marked papilledema is characterized by increased disc swelling, and significant hemorrhages in the peripapillary area, along the vessels and in the paramacularly area. Regressive papilledema is characterized by reduced hyperemia, a grayish optic disc and blurred disc margin. Secondary atrophy is characterized by optic disc discoloration, clear disc margin, and absorbed hemorrhage (if present).

This study followed the ethical standards stated in the Declaration of Helsinki and was approved by the Local Ethics Committee of the Romodanov Institute. Written informed consent was obtained from all individuals enrolled in the study or their guardians.

## Results

Papilledema was found in 4 (2.4%) of the 164 patients with CSDH who underwent treatment at our institution from 2011 through 2021. All these four patients had moderate papilledema, but not marked or atrophic papilledema. This indicates that CSDH was early diagnosed, and patients were early referred for neuroimaging procedures before papilledema could progress to more severe stages and before clinical decompensation could increase. Regression of manifestations of papilledema in the fundus was seen in the four patients in the postoperative period. Patients with papilledema complained of headache of various severities. Headache pain was usually unilateral, located in the frontal temporal area, and not accompanied by apparent nausea or vomiting.

### Case 1

A 56-year-old male patient presented with complaints of severe headache (indicated by a score of 8 on a visual analogue scale of 10), altered speech, and dizziness. He reported that the onset was acute, with an increase in blood pressure to 200/100 mmHg, but he was not on regular hypotensive treatment, and the details were unavailable. The patient was treated at a neurology department over three weeks before he was transferred to the Romodanov Neurosurgery Institute due to negative changes in his health status. Neurological examination findings included obtundation, a positive palmomentum reflex, right hemiparesis, hemihypesthesia, a positive Brudzinski's sign and Kernig's sign, and nuchal rigidity. A brain MRI showed a heterogenous isohyperintense CSDH 35 mm thick or less, overlying the left hemisphere in the fronto-parieto-temporo-occipital region. There was a midline shift to the right of up to 17.9 mm along with compression of the left lateral and third ventricles, and hyrdocephalic dilation of the right lateral ventricle. Periventricular edema and signs of occlusive hydrocephalus of the ventricles were seen. Neuro-ophthalmological examination did not include visual acuity assessment due to the poor general health status of the patient. Direct and consensual pupillary responses were normal. On fundus examination, the patient's optic discs were hyperemic, the edges were blurred, the veins were dilated, and there were streak-like hemorrhages around the optic discs (more intense at the right). The macular area was normal. The patient was diagnosed with bilateral moderate papilledema. He underwent surgery for removal of CSDH. A 150-ml hematoma extruded under pressure. A postoperative brain CT showed as much as 10-mm remnants of the hematoma and a midline shift to the right of up to 5 mm. Regression of manifestations of papilledema in the fundus was seen. The patient was discharged in satisfactory condition.

### Case 2

A 64-year-old male patient presented with complaints of moderate headache (indicated by a score of 6 on a visual analogue scale of 10) and weakness. He reported that he had injured his head (without loss of consciousness) a month before. He was treated for "chronic brain circulation disorder" at a neurological inpatient department, and, after having a brain CT scan was diagnosed with CSDH and transferred to the neurosurgical inpatient department of the Romodanov institute. The patient history was significant for mild head traumas, arterial hypertension and type 2 diabetes with moderate glycemic control. Neurological examination findings included positive palmomentum reflex bilaterally, negative Brudzinski's sign and Kernig's sign bilaterally and left hemiparesis. A brain CT showed signs of a 20-mm CSDH having a density of 36-37 Hounsfield units in the right fronto-parietal region. There was a midline shift to the right of up to 11 mm. The left lateral ventricle appeared compressed. On neuro-ophthalmological examination, visual acuity OU was 1.0, and no change in visual fields was observed. Direct and consensual pupillary responses were normal. There was no loss of ocular motility. The ocular media were transparent. On fundus examination, the patient's optic discs were hyperemic, the edges were blurred, the retinal veins were dilated, and the retinal arteries appeared narrowed. The macular area was normal. The patient was diagnosed with bilateral moderate papilledema. He underwent surgery

for removal of CSDH, and a 120-ml hematoma extruded under pressure. A postoperative brain CT scan showed accumulation of the cerebrospinal fluid, hemorrhage and air above the right hemisphere. There was a hematoma with a thickness of 11 mm and a midline shift to the left of up to 3 mm. Regression of neurological symptoms as well as papilledema regression was observed in the postoperative period.

**Case 3**

At presentation, a 64-year-old male patient did not present any complaints because of a severe health condition and motor aphasia. His wife reported that he was found lying on the floor after falling from his height. Thereafter he was taken to a neurological inpatient department where he received conservative treatment. After having a brain CT scan, the patient was diagnosed with "occipital bone fracture, contusio cerebri, and left epidural subdural hematoma" and transferred to the Romodanov Neurosurgery Institute. The patient's medical history was also significant for essential hypertension. Neurological examination findings included obtundation, motor aphasia, right facial nerve paresis, severe right hemiparesis, positive bilateral Brudzinski's and Kernig's signs bilaterally, and nuchal rigidity. A brain MRI showed a 24-mm irregular focal mass having a density of 39-41 Hounsfield units, with distinct margins, and a midline shift to the right of up to 1.5 mm. Neuro-ophthalmological examination did not include visual acuity assessment due to the poor general health status of the patient. Direct and consensual pupillary responses were normal. The ocular media were transparent. On fundus examination, the patient's optic discs were hyperemic, the edges were blurred, the retinal veins were dilated and tortuous, and the macular area was normal. The patient was diagnosed with bilateral moderate papilledema. He underwent surgery for removal of CSDH. The patient was discharged with an improvement in neurological status and without pupilledema.

**Case 4**

A 54-year-old male patient presented with complaints of severe headache (indicated by a score of 9 on a visual analogue scale of 10), altered speech, and limb weakness. He reported that he had suffered a traumatic head injury in a traffic accident 2 months before. His brain CT was unremarkable. A month after the traumatic event, he began suffering from headache, which increased in severity over a week before presentation. A brain MRI showed a CSDH with a thickness of 27 mm and a midline shift to the right of up to 15 mm, and a perifocal edema of 5 mm. There were signs of subfalcine herniation on MRI. Neurological examination findings included somnolence, positive palmomentum reflex, a negative Kernig's sign and left hemiparesis. On neuro-ophthalmological examination, uncorrected visual acuity in both eyes was 0.7, and corrected visual acuity was 1.0 with a spherical correction of +0.75D in both eyes. No change in visual fields was observed. There was no loss of ocular motility. Direct and consensual pupillary responses were normal. On fundus examination, the patient's optic discs were hyperemic, their edges were somewhat blurred, retinal veins were dilated and somewhat swollen, retinal arteries appeared narrowed, and the macular area was normal. The patient was diagnosed with bilateral moderate papilledema and bilateral mild hyper-opia. He underwent surgery for removal of CSDH. A 200-ml CSDH extruded under pressure. Regression of neurological symptoms as well as papilledema regression was observed in the postoperative period, and the patient was discharged in satisfactory condition.

**Discussion**

There have been contradictory reports on the development of pappiledema in patients with CSDH. Until now, most textbooks on ophthalmology, neurology and neurosurgery consider papilledema to be a common finding in patients with CSDH [8], with an incidence ranging from 30% to 50% [9]. The incidence of papilledema in patients with CSDH was higher than in patients with acute intracranial hematoma (50% vs 3.5% [9] and 12% vs 4% [10]). Studies vary in the reported incidence of papilledema in patients with CSDH. It has been reported that the incidence of papilledema in patients with CSDH in the studies conducted before the nineteen nineties ranged from 20% to 75% [11–16], and in more recent studies was as low as 0.4%-2% [17, 18]. Over the two recent decades, there have been anecdotal reports on bilateral papilledema in patients with CSDH [19, 20, 21]. Bilateral papilledema in patients with CSDH may be caused by an increased gradient of pressure between the subarachnoid space around the bilateral optic nerves and the intracranial subarachnoid space [15]. The difference in age structure of the study sample and early diagnosis of CSDH are likely to explain the difference in the reported incidence of papilledema in patients with CSDH between studies. It has been reported that papilledema is seen more often in children and young adults [14, 22]. Consequently, it should be also taken into account that, with aging of the general population, there will be a shift in the peak incidence of CSDH from the younger age group to the older age group. For example, there was a shift in the peak incidence of CSDH from the 50-59 year-old group in 1972 to the 80-89 year-old group in 2010 to 2013 in Japan [1]. In late nineteen seventies, Kaste and colleagues [23] found papilledema in 41% of patients surgically treated for chronic bilateral subdural hematomas, with the mean interval from trauma to operation as large as eleven weeks, and the mean age of the patients of 60 years, which may indicate a possible association between the duration of the diagnostic assessment of CSDH and the presence of papilledema in patients with CSDH. In addition, in that study [23], 83% of patients had a history of head injury, which is less than the percentage of CSDH patients with a history of head trauma (30-75%) reported in more recent studies [24], in the presence of an increasing use

of antiplatelet/anticoagulant agents in the elderly patients in recent decades. Moreover, papilledema was found in patients with CSDH substantially more frequently in the studies from pre-CT era (22-34%) than in those from pre-CT era (4%) [25].

In the current study, papilledema was found in 4 (2.4%) of the 164 patients with CSDH. Of these four patients, two were younger than 60 years (3.1% of the study pa-tients younger than 60 years), and another two were 60 years old or older (1.9% of the study patients of 60 years or older), the difference being not statistically significant. The study sample should be thrice of present size to make the difference significant. It is noteworthy that, in the current study, papilledema was not found in patients aged 75 years or above. In addition, by the end of in-patient treatment, there was a tendency to papilledema regression in all the four patients.

Therefore, numerous studies have demonstrated that the change in age structure of the study sample significantly affected the assessment of the incidence of papilledema in patients with CSDH. The efficacy of the diagnostic process at presentation of a patient suspected of CSDH may be compromised if being based on the statement that papilledema is a common clinical feature of CSDH. We hope that an acknowledgement of the fact that papilledema is rarely seen in CSDH may change the direction of clinical thinking and improve the efficacy and objectivity of the diagnostic process. It should be, however, noted that, today, neuroimaging diagnostic techniques are used much more commonly than in recent decades, and the state of the fundus is not a major criterion for the diagnosis.

The current retrospective study is a single-center study, and a multicenter study should be conducted to obtain the statistical significance and verify the fact that papilledema is rarely seen in CSDH.

### Conclusion

Therefore, papilledema is rarely seen in CSDH, and fundus examination results are not a major criterion for the diagnosis of CSDH. It has been reported that populations around the world are rapidly aging, and, with aging of the general population, there will be a shift in the peak incidence of CSDH from the younger age group to the older age group. In addition, neuroimaging techniques facilitate early detection of CSDH, and are being increasingly used in the diagnosis, leading to a decreased incidence of papilledema in patients with CSDH. Papilledema was seen only in 2.4% of patients with CSDH in the current study. Neuroimaging techniques (CT and MRI) should be used as major diagnostic methods, and fundus examination, as an addition diagnostic method, in a patient suspected of CSDH.

### References


1. Feghali J, Yang W, Huang J. Updates in Chronic Subdural Hematoma: Epidemiology, Etiology, Pathogenesis, Treatment, and Outcome. *World Neurosurg.* 2020 Sep;141:339-345. doi: 10.1016/j.wneu.2020.06.140.
2. Bartek J Jr, Sjavik K, Stahl F, Kristiansson H, Solheim O, Gulati S, et al. Surgery for chronic subdural hematoma in nonagenarians: a Scandinavian population-based multicenter study. *Acta Neurol Scand.* 2017 Nov;136(5):516-20. doi: 10.1111/ane.12764.
3. Ou Y, Dong J, Wu L, Xu L, Wang L, Liu B, et al. A comparative study of chronic subdural hematoma in three age ranges: Below 40 years, 41-79 years, and 80 years and older. *Clin Neurol Neurosurg.* 2019 Mar;178:63-69. doi: 10.1016/j.clineuro.2019.01.018.
4. Balser D, Farooq S, Mehmood T, Reyes M, Samadani U. Actual and projected incidence rates for chronic subdural hematomas in United States Veterans Administration and civilian populations. *J Neurosurg.* 2015 Nov; 123(4):1209–15. doi: 10.3171/2014.9.JNS141550
5. Eliseeva NM, Serova NK. [Papilledema and its differential diagnosis: A guide for physicians]. Moscow: Triada; 2018. Russian.
6. Scott CJ, Kardon RH, Lee AG, Frissen L. Diagnosis and grading of papilledema in patient with raised intracranial pressure using optical coherence tomography vs clinical experts assessment using a clinical staging scale. *Arch Ophthalmol.* 2010;128(6):705-11. doi:10.1001/archophthalmol.2010.94
7. Mohamed MT, Aldghamy AH, Elsoghair OA. Diagnosis and grading of papilledema using optical coherence tomography compared to clinical staging by Frise'n Scale. SVU-IJMS. 3(2):14-19
8. Greenberg D, Aminoff M, Simon R, eds. Lange Clinical Neurology. 11th edition. New York, NY: McGraw-Hill Education; 2020.
9. Liu GT, Volpe NJ, Galetta SL, eds. Neuro-ophthalmology: Diagnosis and Management. 3rd edition. London: Elsevier Saunders; 2018.
10. Advances in Neurotraumatology. Vigouroux RP, Editor-in-Chief. Volume 1. Extracerebral Collections. McLaurin RL, Managing Editor. Wien: Springer–Verlag; 1986.
11. Handbook of ophthalmologic emergencies: a guide for emergencies in ophthalmology. Gombos GM, edr. Flushing, NY: Medical Examination Pub.; 1973.
12. Zasler ND, Katz DI, Zafonte RD, eds. Brain injury medicine: principles and practice. 2nd ed. New York, NY: Demos Medical Publishing; 2012.
13. Mahapatra AK, Kamal R, Kumar R, eds. Textbook of Traumatic Brain Injury. New Delhi, India: Jaypee Brothers Medical Publishers; 2012.
14. Kuddus R, Hossain MA, Hossain SKS, Hossain ATMM, Debnath H, Rahman A, et al. Study on clinical features and factors associated with thickness of chronic subdural hematoma in adult. Bangabandhu Sheikh Mujib Med Univ J. 2018; 11: 209-12. DOI: 10.3329/bsmmuj.v11i3.37701.
15. Yamada SM, Teramoto A, Yamada S, Hoshiai YC. Severe papilledema identified 3 weeks after head injury. *Neurol Med Chir (Tokyo).* 2002 Jul;42(7):293-6. doi: 10.2176/nmc.42.293.
16. Park SH, Kim YS, Chung SS, Lee KC. Clinical Analysis of Treatment of Chronic Subdural Hematoma. *J Korean Neurosurg Soc.* 1984 Dec; 13(4): 669-79.
17. Yamada SM, Tomita Y, Murakami H, Nakane M, Yamada S, Murakami M, et al. Headache in patients with chronic subdural hematoma: analysis in 1080 patients. *Neurosurg Rev.* 2018 Apr;41(2):549-56. doi: 10.1007/s10143-017-0889-x.

18. Bartek J, Sjåvik K, Dhawan S, Sagberg LM, Kristiansson H, Ståhl F, et al. Clinical Course in Chronic Subdural Hematoma Patients Aged 18-49 Compared to Patients 50 Years and Above: A Multicenter Study and Meta-Analysis. Front Neurol. 2019 Apr 5;10:311. doi: 10.3389/fneur.2019.00311.
19. Wang HS, Kim SW, Kim SH. Spontaneous chronic subdural hematoma in an adolescent girl. *J Korean Neurosurg Soc.* 2013 Mar; 53(3):201–3. doi:10.3340/jkns.2013.53.3.201.
20. Emamhadi M, Chabok SY, Alijani B, Behzadnia H, Rasoulian J, Andalib S. Spontaneous Chronic Subdural Hematoma: *A Case Report and Literature Review. Trauma Mon.* 2017 November; 22(6):e38529. doi:10.5812/traumamon/38529.
21. Dulani S, Dulani R, Lele S, Diagavane S, Anjankar S, Jaiswal N, et al. Chronic Subdural Haematoma in a Case of Hyperthyroidism Presenting with Papilledema. *Chinese Medicine.* 2012; 3 (4):179-81. DOI:10.4236/cm.2012.34027.
22. Won YD, Yi HJ, Lee YJ, Chun HJ, Cho H, Bak KH. Chronic Subdural Hematoma in Young Adult: An Age Comparison Study. *Korean J Neurotrauma.* 2013;9:6-11.
23. Kaste M, Waltimo O, Heiskanen O. Chronic bilateral subdural haematoma in adults. Acta Neurochir (Wien). 1979;48(3-4):231-6. doi: 10.1007/BF02056971.
24. Ou Y, Yu X, Liu X, Jing Q, Liu B, Liu W. A Comparative Study of Chronic Subdural Hematoma in Patients With and Without Head Trauma: *A Retrospective Cross Sectional Study. Front Neurol.* 2020 Nov 27;11:588242. doi: 10.3389/fneur.2020.588242.
25. Iantosca MR, Simon RH. Chronic subdural hematoma in adult and elderly patients. *Neurosurg Clin N Am.* 2000 Jul;11(3):447-54. doi:10.1016/S1042-3680(18)30107-4.

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***Abbreviations:*** *CSDH, chronic subdural hematoma; MRI, magnetic resonance imaging; CT, computed tomography.*