Idiopathic Intracranial Hypertension: Epidemiological, Clinical, and Therapeutic Characteristics in a Moroccan Tertiary Neurology Center – A Retrospective Study of 105 Patients.

Abstract

Background:

Idiopathic intracranial hypertension (IIH) is a neurological disorder characterized by increased intracranial pressure without an identifiable intracranial cause. The condition predominantly affects women of reproductive age and may lead to significant visual impairment if diagnosis and treatment are delayed. Data on IIH from North African populations remain limited.

Objective:

This study aimed to describe the epidemiological, clinical, radiological, and therapeutic characteristics of patients diagnosed with idiopathic intracranial hypertension in a tertiary neurology center in Morocco and to evaluate their clinical outcomes.

Methods:

We conducted a retrospective descriptive study over an 11-year period (January 2010 to April 2021) at the Neurology Department of Mohammed VI University Hospital in Marrakech. Patients diagnosed with IIH according to the revised modified Dandy criteria were included. Demographic data, clinical presentation, ophthalmological findings, neuroimaging results, cerebrospinal fluid characteristics, treatment modalities, and outcomes were analyzed.

Results:

A total of 105 patients were included, representing approximately 2% of neurological hospitalizations during the study period. The mean age was 29.7 ± 8.5 years, with a marked female predominance (female-to-male ratio: 10.6:1). Headache was present in all patients, followed by diplopia (50%), pulsatile tinnitus (48%), and visual disturbances (48%). Papilledema was observed in 96% of cases. Elevated cerebrospinal fluid opening pressure was documented in all patients with normal CSF composition. Brain magnetic resonance imaging revealed indirect signs of intracranial hypertension in the majority of patients, including empty sella, optic nerve sheath distension, and transverse sinus stenosis. Medical management, mainly acetazolamide therapy combined with therapeutic lumbar puncture, was effective in most cases. Surgical treatment was required in 21% of patients, primarily in fulminant cases with rapid visual deterioration. Visual improvement was observed in the majority of treated patients, although permanent blindness occurred in 4.7% of cases.

Conclusion:

Idiopathic intracranial hypertension predominantly affects young women and may lead to severe visual impairment if not diagnosed and treated promptly. Early recognition, appropriate neuroimaging, and

timely therapeutic intervention are essential to improve visual outcomes and reduce the risk of permanent vision loss.

Keywords: idiopathic intracranial hypertension; papilledema; lumbar puncture; magnetic resonance imaging; acetazolamide; visual loss; Morocco.

Idiopathic Intracranial Hypertension: Epidemiological, Clinical, and Therapeutic Characteristics in a Moroccan Tertiary Neurology Center – A Retrospective Study of 105 Patients

Introduction

Idiopathic intracranial hypertension (IIH) is considered an uncommon neurological disorder, with an estimated annual incidence ranging from 0.5 to 2 cases per 100,000 individuals in the general population. However, the incidence increases markedly among women of reproductive age with obesity, reaching up to 15–20 cases per 100,000 in some populations. Over the past decades, the incidence of IIH has risen in parallel with the global increase in obesity, suggesting a strong association between metabolic factors and disease development. These epidemiological trends highlight the growing clinical relevance of IIH as a potential cause of chronic headache and visual impairment worldwide [1–4].

IIH predominantly affects women of childbearing age, particularly those with overweight or obesity, and represents a potentially vision-threatening condition. Although once referred to as "benign intracranial hypertension," this terminology has been abandoned due to the significant risk of irreversible visual loss and the substantial impact on quality of life. Visual impairment, chronic headaches, and cranial nerve palsies are common manifestations, underscoring the importance of early diagnosis and appropriate treatment.

The diagnosis of IIH is based on the revised modified Dandy criteria, which include papilledema, normal neurological examination except for cranial nerve abnormalities, normal cerebrospinal fluid (CSF) composition, absence of structural abnormalities on neuroimaging, and elevated CSF opening pressure. Advances in magnetic resonance imaging (MRI) and venography have improved diagnostic accuracy by identifying indirect radiological signs of raised intracranial pressure, such as empty sella, optic nerve sheath distension, posterior globe flattening, and transverse sinus stenosis.

Despite growing recognition of IIH worldwide, data from North African populations remain limited. Regional studies are essential to better characterize epidemiological patterns, clinical presentations, and treatment outcomes, particularly in healthcare settings with varying diagnostic and therapeutic resources.

The aim of this study was to describe the epidemiological, clinical, radiological, and therapeutic features of idiopathic intracranial hypertension in patients hospitalized at a tertiary neurology center in Morocco and to compare our findings with previously published data.

Materials and Methods

Study Design and Setting

We conducted a retrospective descriptive study over an 11-year period, from January 1, 2010, to April 1, 2021, at the Neurology Department of Mohammed VI University Hospital, Marrakech, a tertiary referral center serving southern Morocco.

Study Population

All patients hospitalized during the study period with a diagnosis of idiopathic intracranial hypertension were eligible for inclusion. The diagnosis was established according to the revised modified Dandy criteria. Patients with incomplete medical records or evidence of secondary causes of intracranial hypertension were excluded.

Data Collection

The following variables were analyzed: demographic characteristics (age and sex), medical history and risk factors, clinical presentation, ophthalmological findings, neuroimaging findings, cerebrospinal fluid opening pressure and composition, therapeutic management (medical treatment, lumbar puncture, and surgical intervention), and short- and long-term outcomes.

All patients underwent ophthalmological evaluation within the first 24 hours of admission, including best-corrected visual acuity assessment and fundus examination. Visual field testing was performed when feasible.

Neuroimaging: Brain computed tomography (CT) was used as a first-line imaging modality in selected cases. Brain MRI was performed in all patients to exclude secondary causes and to identify indirect signs of intracranial hypertension. MR venography was used to assess venous sinus abnormalities.

Lumbar puncture was performed in the lateral decubitus position with measurement of CSF opening pressure using a standardized technique. CSF samples were systematically analyzed for cytological and biochemical abnormalities.

Statistical Analysis

Descriptive statistical analysis was performed using Microsoft Excel 2021. Categorical variables were expressed as frequencies and percentages, while continuous variables were presented as mean ± standard deviation.

Ethical Considerations

This study was conducted in accordance with the ethical standards of the institutional research committee and the principles of the Declaration of Helsinki. The study protocol was approved by the ethics committee of Mohammed VI University Hospital, Marrakech. Due to the retrospective nature of the study, the requirement for informed consent was waived. Patient confidentiality and anonymity were strictly maintained. The authors declare no conflict of interest.

Results

Epidemiological Characteristics

A total of 105 patients were included (Table 1), accounting for approximately 2% of neurological hospitalizations during the study period. The mean age was 29.7 ± 8.5 years. There was a marked female predominance, corresponding to a female-to-male ratio of 10.6:1. The most affected age group was between 18 and 30 years, representing 57% of cases.

Variable	Value
Number of patients	105
Mean age (years ± SD)	29.7 ± 8.5
Age range (years)	12–73
Female sex	96 (91.4%)
Male sex	9 (8.6%)
Female-to-male ratio	10.6:1

Table 1. Demographic Characteristics of the Study Population
Legend: SD: standard deviation.

Risk Factors and Medical History

Obesity or overweight was documented in 33% of patients for whom body mass index was available. Among these, 45% had morbid obesity. Oral contraceptive use was reported in 48% of female patients. Endocrine disorders were present in 18% of cases, including diabetes mellitus, thyroid dysfunction, and Cushing's disease. Anemia was identified in eight female patients. Five patients were pregnant at the time of diagnosis (Table 2).

Risk factor / condition	n (%)
Overweight or obesity	35 (33%)
Morbid obesity	16 (45% of obese patients)
Oral contraceptive use	46 (48% of women)
Endocrine disorders	19 (18%)
Anemia	8 (7.6%)
Pregnancy	5 (4.7%)

Table 2. Risk Factors and Associated Conditions

Clinical Presentation

Headache was the presenting symptom in all patients . Headaches were typically diffuse, pulsatile, and more intense in the morning, often associated with neck pain. Visual disturbances were reported in 48% of cases, including blurred vision and transient visual obscurations. Diplopia was observed in 50% of patients, most commonly due to sixth cranial nerve palsy. Pulsatile tinnitus was reported by 48% of patients.

More than 60% of patients presented with an acute onset of symptoms, while 24% had a progressive course with delayed consultation.

Neurological and Ophthalmological Findings

All patients were conscious with normal mental status. Apart from cranial nerve involvement, no focal neurological deficits were observed. Cranial nerve VI palsy was the most frequent neurological finding.

Papilledema was present in 96% of patients (Table 3), predominantly bilateral and symmetric. Visual acuity was reduced in 48% of cases, with four patients presenting with bilateral blindness at admission.

Finding	n (%)
Papilledema	101 (96%)
Bilateral papilledema	97 (92%)
Reduced visual acuity	50 (48%)
Bilateral blindness at presentation	4 (3.8%)
Sixth cranial nerve palsy	53 (50%)

Table 3. Ophthalmological and Neurological Findings

Neuroimaging Findings

Brain CT scans were performed in 37 patients and were normal in all cases. Brain MRI revealed indirect signs of intracranial hypertension in the majority of patients, including empty sella, optic nerve sheath distension, posterior globe flattening, and optic nerve tortuosity. Transverse sinus stenosis was identified in a subset of patients on MR venography (Figure 1: a,b,c,d).

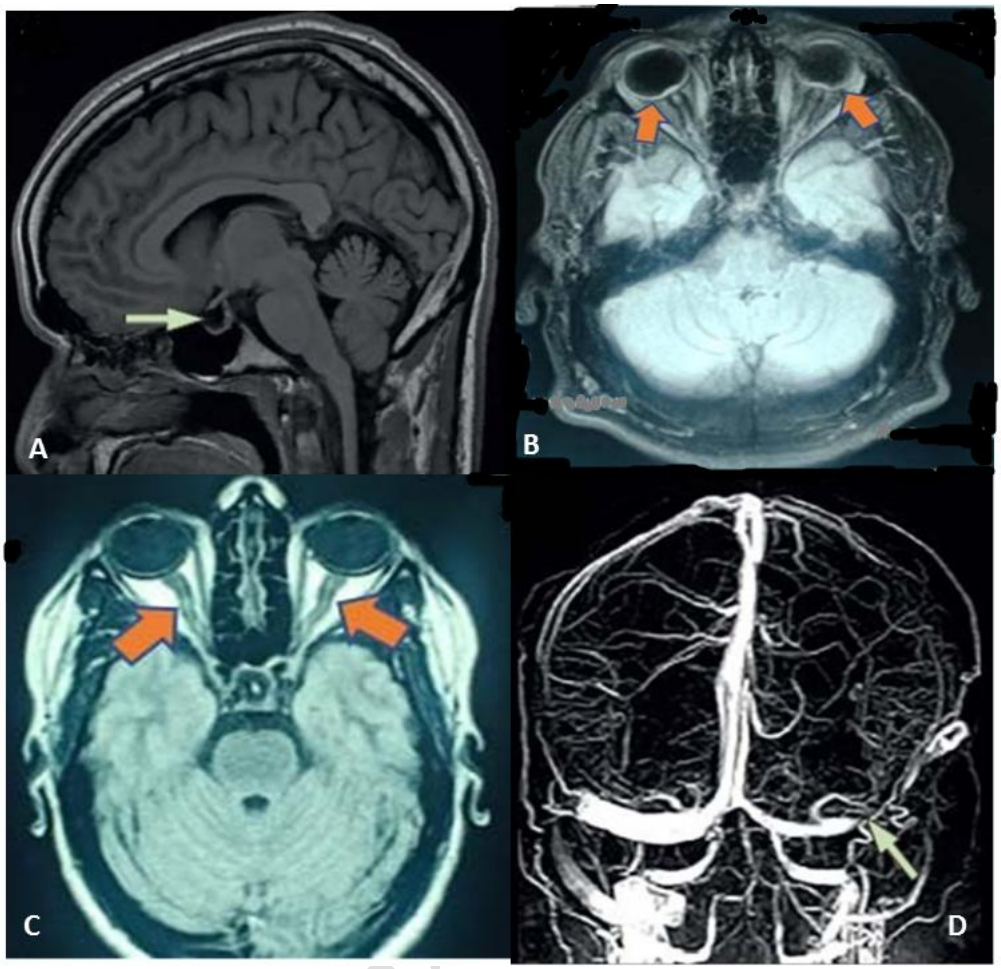

Figure 1. MRI indirect signs of intracranial hypertension:
(a) empty sella;
(b) posterior globe flattening and optic nerve sheath distension;
(c) optic nerve tortuosity;
(d) transverse sinus stenosis.

Cerebrospinal Fluid Findings

Elevated CSF opening pressure (>25 cmH_2O in adults and >28 cmH_2O in children) was documented in all patients. CSF composition was normal in all cases.

Treatment and Outcomes

All patients underwent therapeutic lumbar puncture. Medical treatment with acetazolamide was administered in nearly all cases, with potassium supplementation as needed (Table 4). Corticosteroids were used in selected cases, particularly before 2017. Surgical treatment, mainly ventriculoperitoneal shunting, was required in 21% of patients, primarily in fulminant forms with rapid visual deterioration.

Treatment/ outcome	n (%)
Therapeutic lumbar puncture	105 (100%)
Acetazolamide therapy	98 (93%)
Corticosteroid use	22 (21%)
Surgical treatment	22 (21%)
Visual improvement	61 (58%)
Recurrence	5 (4.7%)
Permanent blindness	5 (4.7%)

Table 4. Treatment Modalities and Outcomes

Short-term outcomes were favorable in most patients, with improvement in headache and visual symptoms. Visual acuity improved in 58% of patients with initial visual impairment . Long-term follow-up revealed complete recovery in the majority of patients within 3–6 months. Recurrence occurred in five patients. Permanent blindness was observed in 4.7% of cases.

Discussion

Idiopathic intracranial hypertension is a complex neurological disorder with heterogeneous clinical presentations and incompletely understood pathophysiological mechanisms. Our study provides a comprehensive overview of IIH in a large cohort of patients managed at a tertiary neurology center in Morocco and offers valuable insights into its epidemiological, clinical, radiological, and therapeutic characteristics.

Epidemiological Characteristics

The marked female predominance observed in our cohort (female-to-male ratio of 10.6:1) is consistent with previously published series, in which sex ratios range from 3:1 to 15:1 [1–4]. This predominance is classically attributed to hormonal, metabolic, and adipose tissue–related factors. The mean age of 29.7 years and the predominance of young adults between 18 and 30 years also align with international data, confirming that IIH is primarily a disease of women of childbearing age [5,6].

Although IIH is traditionally considered rare, its incidence has increased significantly over recent decades, largely paralleling the global rise in obesity [7]. In our series, IIH accounted for 2% of neurological hospitalizations, a proportion comparable to reports from other tertiary centers in both developed and developing countries [8,9].

Pathophysiological considerations

The exact pathophysiology of idiopathic intracranial hypertension remains incompletely understood. Several mechanisms have been proposed, including impaired cerebrospinal fluid (CSF) absorption at the level of the arachnoid granulations, increased intracranial venous pressure, and metabolic or hormonal factors associated with obesity. Recent studies have emphasized the potential role of transverse venous sinus stenosis, which may lead to increased venous pressure and impaired CSF drainage. In addition, obesity-related endocrine and inflammatory pathways may contribute to altered CSF dynamics and intracranial pressure regulation. These mechanisms are likely multifactorial and may vary among patients, reflecting the complex pathophysiology of the disease [5–8].

Risk Factors and Associated Conditions

Obesity remains the most consistently identified risk factor for IIH. In our cohort, one-third of patients were overweight or obese, with a high proportion presenting with morbid obesity. Several studies have demonstrated a strong association between elevated body mass index and poor visual outcomes, with a higher risk of severe visual loss as BMI increases [9,10,17]. These findings support the hypothesis that adipose tissue plays an active role in IIH pathophysiology through endocrine and metabolic mechanisms.

Endocrine disorders, including diabetes mellitus, thyroid dysfunction, and Cushing's disease, were observed in a subset of patients. These associations have been previously reported and suggest that metabolic dysregulation may contribute to altered cerebrospinal fluid (CSF) dynamics [11].

Iron-deficiency anemia was identified in eight patients in our series. An association between anemia and IIH has been increasingly recognized, with several studies reporting rapid clinical and visual improvement following correction of anemia [12,13]. Although the underlying mechanism remains unclear, impaired oxygen delivery and altered cerebral venous hemodynamics have been proposed.

Clinical Presentation

Headache was the most common presenting symptom, reported by all patients, consistent with the literature [14]. The typical characteristics—diffuse, pulsatile headaches with morning predominance—reflect raised intracranial pressure. Visual disturbances and diplopia were also frequent, highlighting the potential for severe visual morbidity.

Sixth cranial nerve palsy was the most common neurological deficit observed, a well-recognized manifestation of increased intracranial pressure [15]. Rare cranial nerve involvements, including third and fourth nerve palsies, were also noted, emphasizing the variability of neurological presentations in IIH.

Ophthalmological Findings and Visual Prognosis

Papilledema was present in 96% of patients, confirming its role as a cardinal diagnostic sign of IIH. However, a small proportion of patients presented without papilledema, a phenomenon increasingly described in the literature as "IIH without papilledema" [16]. These cases pose a diagnostic challenge and underscore the importance of integrating clinical, radiological, and CSF findings.

Visual impairment was common in our cohort, with nearly half of the patients presenting with reduced visual acuity. Permanent blindness occurred in 4.7% of cases, a rate comparable to that reported in large series [16,17]. Delayed diagnosis and delayed initiation of appropriate treatment were the main contributors to poor visual outcomes, reinforcing the need for early recognition and close ophthalmological monitoring.

Neuroimaging and Pathophysiological Considerations

Brain MRI played a crucial role in diagnosis, revealing indirect signs of raised intracranial pressure in most patients. Empty sella, optic nerve sheath distension, posterior globe flattening, and optic nerve tortuosity are well-established imaging markers of IIH [9,12].

Transverse sinus stenosis was identified in a subset of patients. Venous sinus stenosis is now recognized as a common finding in IIH and is thought to contribute to impaired CSF absorption by increasing venous pressure [18]. However, the causal relationship remains debated, as such stenoses may resolve after normalization of intracranial pressure or persist despite clinical improvement .

Therapeutic Management

Medical management remains the cornerstone of IIH treatment. In our series, acetazolamide was the first-line therapy and was well tolerated in most patients. Its efficacy in reducing CSF production and improving visual outcomes has been demonstrated in randomized controlled trials [19,20].

Therapeutic lumbar puncture was systematically performed and provided symptomatic relief in many patients. However, repeated lumbar punctures were required in a minority of cases and were insufficient in fulminant forms.

Surgical intervention was necessary in 21% of patients, primarily those with fulminant IIH and rapidly progressive visual loss. Ventriculoperitoneal shunting was the most commonly used procedure, with favorable outcomes in the majority of cases. These findings are consistent with current recommendations advocating early surgical intervention in patients with vision-threatening disease refractory to medical therapy [15,18].

Study Limitations

This study has several limitations. First, the retrospective design may introduce selection bias and limit the completeness of some clinical data. Second, body mass index and long-term ophthalmological follow-up were not available for all patients. Third, the study was conducted in a single tertiary referral center, which may limit the generalizability of the results to the general population. Nevertheless, the relatively large sample size and the long study period provide valuable insights into the characteristics of idiopathic intracranial hypertension in a North African population.

Study Strengths

Despite these limitations, our study represents one of the largest series of idiopathic intracranial hypertension reported from Morocco and North Africa. The relatively large number of patients, the long study period, and the comprehensive clinical, radiological, and therapeutic evaluation provide valuable data that contribute to the limited regional literature on this condition.

Clinical Implications

Early recognition of idiopathic intracranial hypertension is essential to prevent irreversible visual loss. Clinicians should maintain a high index of suspicion in young women presenting with chronic headache, visual disturbances, and papilledema, particularly in the presence of obesity or endocrine disorders. Prompt neuroimaging to exclude secondary causes, followed by lumbar puncture with measurement of CSF opening pressure, remains essential for diagnosis. Early initiation of medical therapy, particularly acetazolamide, combined with weight management and close ophthalmological monitoring, plays a key role in preserving visual function and preventing long-term complications [5,9,10].

Conclusion

Idiopathic intracranial hypertension is a potentially vision-threatening neurological disorder that predominantly affects young women. Our study highlights the clinical diversity and the significant risk of visual impairment associated with delayed diagnosis. Early recognition, appropriate neuroimaging, and prompt initiation of treatment are essential to improve outcomes. Greater awareness of this condition among clinicians may help reduce diagnostic delays and prevent permanent visual loss.

Declaration

Ethical Approval And Consent To Participate

It is a retrospective report and the authors respected the anonymity of the patients and his archived medical file. Consequently, only the agreement of the head of the department (KN) was required and obtained.

Consent For Publication

Not applicable.

Competing Interests

The authors declare no potential conflicts of interest.

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Authors' Contributions

All authors participated in patient care. They all contributed to the writing of the manuscript. CM, KN, and LN approved the final version.

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References

1. KapilG.Kapoor. MoreThanMeetsTheEyeRedefiningIdiopathicIntracranialHypertension. International Journal of Neuroscience, 120, 471–482, 2010. DOI10.3109/00207451003760098

2. CorbettJJ,Thompson HS. Therationalmanagementofidiopathicintracranialhypertension. Arch Neurol. 1989 ; 46 :1049–51. DOI10.1001/archneur.1989.00520460025008

3. Dandy,W.E. Intracranialpressurewithoutbraintumor:Diagnosisandtreatment. Annals of Surgery, 1937, 106, 492– 513. DOI 10.1097/00000658-193710000-00002

4. Foley,J. Benignformsofintracranialhypertension:"Toxic"and"otitic"hydrocephalus. Brain, 1955, 78, 1–41. DOI 10.1136/jnnp.19.1.28

5. Digre. Three Current Controversies in Idiopathic Intracranial Hypertension Neuro-Ophthalmology, 33, 93–99, 2009. DOI 10.1080/01658100902930537.

6. DeborahI.Friedman,GrantT. LiuandKathleenB.Digre.Reviseddiagnosticcriteriaforthepseudotumorcerebri syndrome in adults and children. Neurology 2013 ; 81 ;1159-1165. DOI 10.1212/WNL.0b013e3182a55f17.

7. Friedman DI,Jacobson:Diagnosticcriteria for idiopathicintracranial hypertension. Neurology 2002 ; 59 :1492–1495. DOI10.1212/01.wnl.0000029570.69134.1b.

8. DrMohamedAmineDERDOUR STÉNOSEDUSINUSTRANSVERSEETHYPERTENSIONINTRACRANIENNEIDIOPATHIQUE:aspectsdiagnosticsetvaleurpronostique2017 ; http://www.sfoonline.fr/session/media/stenose-du-sinus-transverse-et-hypertension-intracranienne-idiopathique-aspects.

9. BidotS,BruceBB.

Updateonthediagnosisandtreatmentofidiopathicintracranialhypertension.SeminNeurol 2015 ;35 (5) :527–38. DOI 10.1055/s-0035-1563569.

10. RiggealBD,BruceBB,SaindaneAM,RidhaMA,KellyLP,NewmanNJ,etal. Clinicalcourseofidiopathicintracranialhypertensionwithtransversesinusstenosis. Neurology 2013 ;80(3) :289–95. DOI 10.1212/WNL.0b013e31827debd6

11. PatsalidesA,OliveiraC,WilcoxJ,BrownK,GroverK,GobinYP,etal. Venoussinusstentinglowerstheintracranialpressureinpatientswithidiopathic intracranial hypertension. J NeurointervSurg 2019 ;11(2) :175–8. DOI 10.1136/neurintsurg-2018-014032.

12. LenckS,RadovanovicI,NicholsonP,HodaieM,KringsT,MendesPereiraV. Idiopathicintracranialhypertension:thevenoglymphaticconnections.Neurology2018 ;91(11):515–22. DOI 10.1136/neurintsurg-2018-014032. DOI10.1212/WNL.0000000000006166.

13. KanagalingamS,SubramanianP. Endoprothèsedessinusveineuxcérébrauxpourpseudotumeurcérébrale:unerevue. Saoudien J Ophthalmol.2015 ;29(1) :3-8. DOI 10.1016/j.sjopt.2014.09.007.

14. CorbettJJ,Thompson HS. Therationalmanagementofidiopathicintracranialhypertension.ArchNeurol.1989; 46:1049–51.

15. James H Manfield , Kenny K-H Yu , EvangelosEfthimiou , Ara Darzi , ThanosAthanasiou , HutanAshrafian : Chirurgie bariatrique ou perte de poids non chirurgicale pour l'hypertension intracrânienne idiopathique . Une revue systématique et une comparaison des méta- analyses. Surg obèse. 2017, 27:513-521. DOI 10.1007/s11695-016-2467-7.

16. SzewkaAJ,BruceBB,NewmanNJ,Biousse V. Hypertensionintracrânienneidiopathique:relationentrel'obésitéetlesrésultatsvisuels. J Neuroophtalmol.2013 ;33(1) :4-8.DOI 10.1097/WNO.0b013e31823f852d

17. KoMW,ChangSC,RidhaMA,et al. Prisedepoidsetrécidivedansl'hypertensionintracrânienneidiopathique:uneétude cas-témoins.Neurologie.2011 ;76(18) :1564-1567.

18. JensenR,RadojicicA,YriH. Le diagnostic et la prise en charge de l'hypertension intracrânienne idiopathique et desmaux de tête associés.Neurol Sci.2013 ;34(suppl 1) : S147-S149.DOI 10.1177/1756285616635987.

19. ValérieBiousse , Beau B Bruce , Nancy J Newman: Anemia and papilledema. Am J Ophthalmol. 2003,

135:437-46. DOI 10.1016/s0002-9394(02)02062-7

20. Saket S T, Jiwan K, Yatika C et al .
Navigating the Enigma: A Comprehensive Review of Idiopathic Intracranial Hypertension; Cureus. 2024 Mar 16;16(3):e56256. DOI 10.7759/cureus.56256. eCollection 2024 Mar.