Review Article

# Relationship between Obesity and Idiopathic Intracranial Hypertension: A Systematic Review

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## ABSTRACT

Idiopathic intracranial hypertension (IIH) is a neurological disorder marked by elevated intracranial pressure without a structural cause, leading to papilledema and potential irreversible vision loss. Obesity is the strongest modifiable risk factor. This systematic review synthesized six studies (three retrospective cohorts, one chart review, one economic model, one cross-sectional study) examining epidemiology, weight-loss interventions, CSF pressure correlations, and obstructive sleep apnea (OSA) mediation. Reproductive-aged obese women showed incidence of 22.0 vs. 6.8 per 100,000 compared to all women, confirming a dose-response relationship. Bariatric surgery produced median 24% weight loss, mean CSF pressure reduction of 10.7 cm $H_2O$ ($p=0.003$), symptom improvement, and projected 20-year cost savings of £49,500. Conversely, one propensity-matched cohort found GLP-1 receptor agonist use associated with higher odds of venous sinus stenting (OR 2.40, 95% CI 1.44–4.00) and ventriculoperitoneal shunting (OR 3.34, 95% CI 2.00–5.61), conflicting with other meta-analyses; GLP-1 RA evidence remains conflicting. Elevated CSF pressure correlated with papilledema grade ($r=0.41$, $p=0.04$) and residual visual deficits. Obesity independently predicted high-risk Berlin questionnaire scores for OSA but was not a sole mediator. Weight reduction via bariatric surgery (≥24% baseline weight in patients with BMI ≥35 kg/m²) offers lower ICP, better vision, and fewer neurosurgical interventions, though evidence certainty is very low for most outcomes. Findings support multidisciplinary management with conditional weight-loss recommendations and OSA screening; future randomized trials are needed.

**Keyword:** Idiopathic intracranial hypertension; pseudotumor cerebri; obesity; body mass index; bariatric surgery; GLP-1 receptor agonists; intracranial pressure.

## Introduction

Idiopathic intracranial hypertension (IIH) is defined by elevated intracranial pressure (ICP) without structural lesions, tumors, hydrocephalus, or venous sinus thrombosis [1]. Patients typically present with headaches, pulsatile tinnitus, transient visual obscurations, and papilledema, which can lead to

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irreversible vision loss if untreated [2]. Though its pathophysiology remains unclear, IIH causes substantial morbidity, often requiring long-term management, repeated lumbar punctures, or surgical interventions (VPS, VSS, ONSF) in refractory cases [3]. Over the past three decades, IIH epidemiology has shifted with rising global obesity. General annual incidence is 1.0–2.0/100,000 [4] but rises to ~21.0/100,000 in reproductive-age women and 28.0/100,000 in obese women aged 20–44 [5]. In the US, adult incidence increased from 16.0/100,000 (1990–1999) to 127.0/100,000 (2020–2024) [6]; prevalence among women aged 18–55 is 3.44/10,000, with regional variation paralleling obesity [6]. In the UK, IIH affects ~5,000 individuals, with ~90% obese [7]; an Austrian study reported prevalence of 4.77/100,000 total and 7.88/100,000 female discharges [8]. Obesity is the strongest modifiable risk factor, with >90% of affected women and ~60% of men obese at diagnosis [5]. The BMI–IIH relationship is dose-dependent: obese women had an annual incidence of ~22.0/100,000 vs. 6.8/100,000 for all women [9], and BMI >40 kg/m² confers a 15-fold higher risk vs. normal BMI [5]. Proposed mechanisms include elevated intra-abdominal pressure impairing venous drainage, obesity hypoventilation with hypercapnia, adipokine dysregulation (leptin/adiponectin), abnormal 11β-HSD1 activity, and low-grade inflammation (IL-1β, TNF-α) affecting CSF dynamics [10], though causal pathways remain uncertain. This systematic review synthesizes evidence from the last five years on obesity–IIH associations, the impact of weight-loss interventions (bariatric surgery, GLP-1 agonists) on clinical outcomes, prognostic value of obesity and CSF pressure, and obesity's associational role with comorbidities like sleep apnea, aiming to inform clinical practice and future research.

## Methods

A systematic literature review was conducted to examine the association between obesity and IIH. Four major electronic databases were searched—PubMed/MEDLINE, Scopus, Web of Science, and the Cochrane Library—covering publications from 2021 to 2026. The search strategy combined MeSH terms and text words for IIH, obesity, weight-loss interventions, and clinical outcomes, using Boolean operators "AND" and "OR". Only English-language studies involving human participants were included. Reference lists of included studies and relevant reviews were manually screened to identify additional articles. Eligibility Criteria: Studies were included based on the PICOS framework: (P) patients of any age with confirmed IIH per modified Dandy or Friedman criteria; (E) obesity (BMI ≥30 kg/m² for adults, ≥95th percentile for children) or weight-loss interventions (bariatric surgery, dietary management, GLP-1 RAs); (C) non-obese IIH patients or non-IIH controls, as appropriate; (O) at least one of: CSF opening pressure, papilledema grade, visual field deficits, headache, need for neurosurgery (VSS, VPS, ONSF), or medication use (acetazolamide, topiramate); (S) retrospective/prospective cohorts, cross-sectional, case-control, economic modelling, or RCTs. Exclusions: case reports/series <5, editorials, abstracts, animal studies, non-English, unclear obesity/IIH data, or secondary IIH from identifiable causes. Study Selection Process: All citations were imported into Rayyan for screening [11]. Duplicates were removed using the software's detection feature, followed by manual verification. Two reviewers independently screened titles and abstracts against eligibility criteria; disagreements were resolved through discussion or by a third reviewer. Full texts of potentially relevant studies were then independently assessed by the same reviewers, with exclusion reasons recorded. The process adhered to PRISMA 2020 guidelines, including the generation of a flow diagram [12]. Data Extraction: A standardised data collection form was developed and pilot-tested on two studies. Two reviewers independently extracted: author, year, location, study design, sample size, demographics (age, sex, BMI, obesity prevalence), IIH diagnostic criteria, intervention/exposure details, comparator groups, outcomes, follow-up duration, key findings, and statistical methods. Discrepancies were resolved by consensus. Authors were contacted up to twice for missing data; unresolved items were recorded as "NM". Risk of Bias Assessment: Two reviewers independently assessed methodological quality using design-specific tools. For retrospective cohort studies, the Newcastle–Ottawa Scale (NOS) was applied, evaluating selection (4 items), comparability (2 items), and outcome (3 items) [13]; scores ≥7 indicated low risk, 5–6 moderate, and ≤4 high risk. The single cross-sectional study was appraised using the Joanna Briggs Institute's 8-item checklist, covering sampling, demographics, exposure, outcome measurement, confounders, and statistical analysis. Data Synthesis and Analysis: Due to substantial heterogeneity in study design, populations, outcomes, and follow-up, meta-analysis was not feasible. A narrative synthesis was therefore performed, organising findings into five thematic

areas: (1) epidemiological evidence linking obesity to IIH; (2) bariatric surgery outcomes; (3) GLP-1 receptor agonist effects; (4) relationships between obesity, CSF pressure, and visual outcomes; and (5) obstructive sleep apnoea as an associated factor.

## Results

A total of 331 records were found in databases and registries, as shown in (Figure 1). Ninety-one of the 164 records that were reviewed after 167 duplicate records were eliminated were discarded. 49 of the remaining 73 reports were unsuccessfully retrieved. After the entire texts of 24 publications were evaluated for eligibility, 10 reports were excluded because the results were incorrect, 5 because the population was incorrect, and 3 because the studies were abstracts. Six papers ultimately satisfied the eligibility requirements and were added to the systematic review. The demographic and baseline characteristics of the six studies included are shown in (Table 1) [14- 19]. The number of participants in the studies varied from 18 in Apperley et al. [14] to 5380 in the propensity-matched cohort study by Debiec et al. [16]. Three studies used a retrospective cohort design [14,16,17], one used a retrospective chart review [15], one used an economic decision model [18], and one used a cross-sectional design [19]. The studies were geographically located in the United Kingdom [14,18], the United States [16,17] and Turkey [15,19]. The average age ranged from 11.0±3.3 years in the pediatric studies [14,15] to a median age of 46.7 years for the adult bariatric surgery study [17]. The studies indicated a female predominance from 53.8% [15] to 100% in Elliot et al. [18] which is consistent with the well established sex disparity of IIH. Body mass index (BMI) data revealed mean values of 30.3 kg/m² [14] and 48.3 kg/m² [17] and Bedir and Nuhoğlu [15] reported overweight prevalence of 34.6% and obesity prevalence of 19.2% in their pediatric group. All studies included a confirmed diagnosis of IIH or obesity as inclusion criteria, and Debiec et al. [16] had 100% obesity in their GLP-1 RA treated group. The key findings from the six included studies are presented in (Table 2). Among children with obesity, Apperley et al. [14] documented the clinical impact of IIH, reporting that 66.6% of their pediatric cohort presented with both headaches and ocular signs, alongside a mean BMI standard deviation score of +2.5. Bedir and Nuhoğlu [15] demonstrated a significant correlation between CSF opening pressure and papilledema grade ($r=0.41$, $p=0.04$), and further identified CSF opening pressure as an independent predictor of residual visual deficit (OR 3.2, 95% CI 1.1–9.1, $p=0.04$). Debiec et al. [16] observed that GLP-1 receptor agonist use was associated with higher odds of venous sinus stenting at both 5 weeks (OR 2.40, 95% CI 1.44–4.00, $p=0.0005$) and 6 months (OR 2.31, 95% CI 1.10–4.86, $p=0.0233$), as well as higher odds of ventriculoperitoneal shunting at both time intervals (OR 3.34, 95% CI 2.00–5.61, $p=0.0001$ and OR 2.51, 95% CI 1.35–4.68, $p=0.0026$). This finding contradicts other meta-analyses and requires further investigation. Wills et al. [17] reported a mean reduction in CSF opening pressure of 10.7 cm $H_2O$ (95% CI 4.7–16.6, $p=0.003$) following bariatric surgery, accompanied by a median total weight loss of 24% and notable improvements in headache, papilledema, visual field defects, and visual symptoms. Elliot et al. [18] conducted a Markov model analysis, which revealed that bariatric surgery was cost-saving over a 20-year horizon, generating £49,500 in savings and an additional 1.16 quality-adjusted life years relative to community-based weight management. Using the Berlin questionnaire, Deveci and Kabeloğlu [19] determined that obesity was the only independent predictor of a high-risk Berlin questionnaire score, and that the prevalence of obesity was significantly elevated in the IIH group compared to controls ($p<0.001$). (Table 2) also presents the risk of bias assessment for the six included studies. The three retrospective cohort studies and one retrospective chart review were evaluated using the Newcastle-Ottawa Scale (NOS), with scores ranging from 7/9 to 9/9, indicating low to moderate risk of bias. The economic decision model was assessed with the Philips checklist (52/61, low-moderate risk), and the cross-sectional study using the JBI checklist (8/8, low risk). (Table 3) summarizes the GRADE certainty of evidence for each outcome. The certainty was rated as very low for most outcomes, including obesity as a risk factor, GLP-1 RA effects on neurosurgical interventions, CSF pressure–visual deficit correlation, and obesity as a predictor of OSA risk. Bariatric surgery outcomes and cost-effectiveness were rated as low certainty. Reasons for downgrading included observational study designs, risk of bias, imprecision, and suspected publication bias.

## Discussion

The epidemiological association between obesity and IIH has been well established in the literature. In our review, Apperley et al. [14] found that among 18 children with IIH, the mean body mass index (BMI) was 30.3 kg/m² with a mean BMI standard deviation score of +2.5, and 61.1% of patients were female. Similarly, Bedir and Nuhoğlu [15] reported that among 26 paediatric IIH patients, overweight and

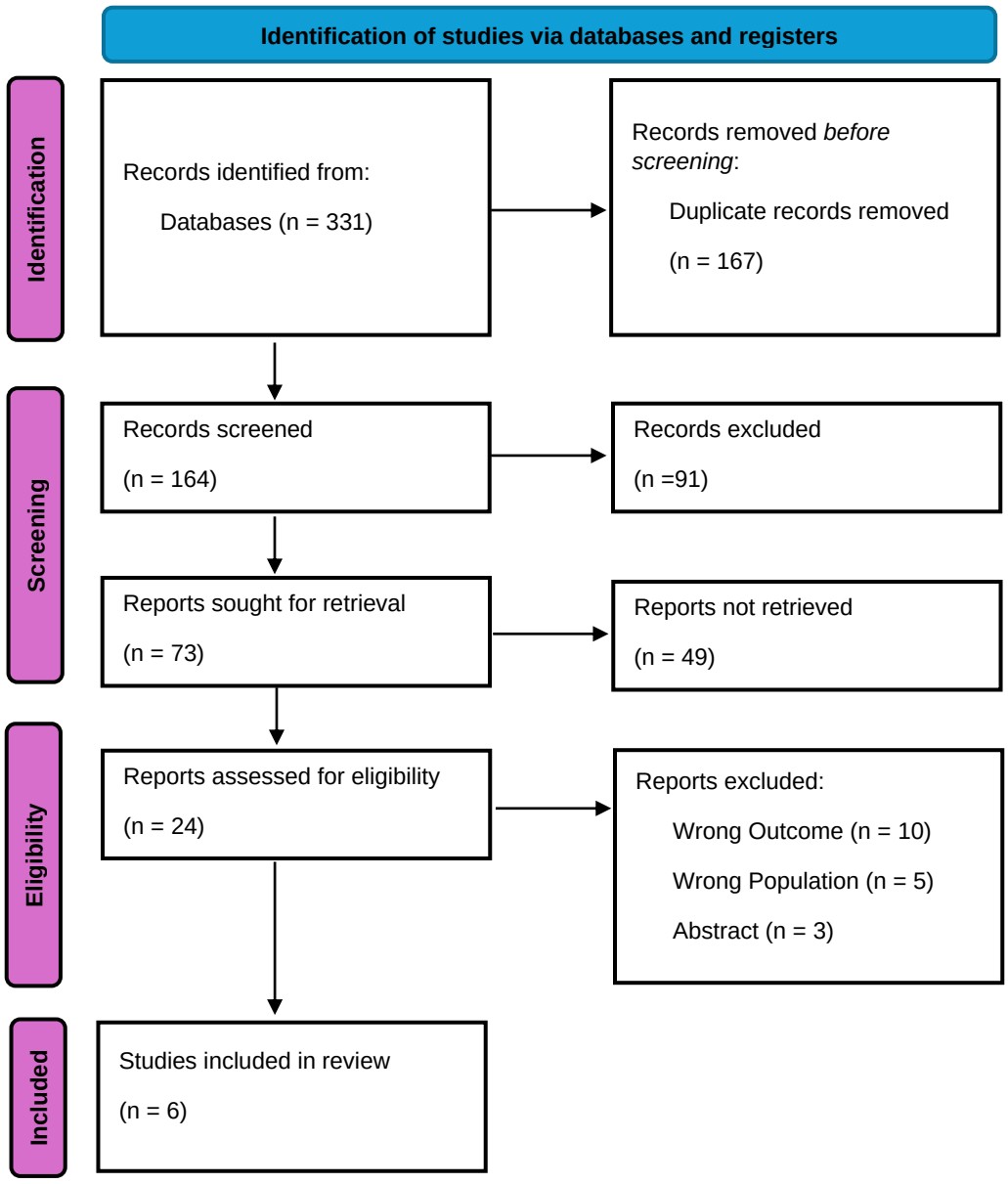


*Figure 1: PRISMA Flow Diagram of Study Selection Process.*

**Table 1:** Demographic and baseline characteristics of included studies.

| **Study (Author, Year [Ref.])** | **Location** | **Study Design** | **Sample Size** | **Sample Type** | **Mean Age (years)** | **Female (%)** | **Mean/ Median BMI (kg/m²)** | **Obesity/Overweight Prevalence (%)** | **Key Inclusion Criteria** | **Key Exclusion Criteria** |
|---|---|---|---|---|---|---|---|---|---|---|
| Appelrley et al., 2022 [14] | UK (tertiary childrен's hospital) | Retrospective cohort | 18 | Pediatric IIH patients | 11.0 ± 3.3 (range 6-15) | 61.1% (11/18) | 30.3 | Mean BMI SDS: +2.5 (obesity defined) | Diagnosed with IIH at tertiary childrен's hospital between April 2017–April 2019 | NM |
| Bedir & Nuhoğlu, 2026 [15] | NM | **Retrospective chart review** | 26 | Pediatric IIH patients (<18 years) | 11.2 ± 3.4 | 53.8% | NM | Overweight: 34.6%; Obesity: 19.2% | Diagnosed according to revised Friedman criteria; age <18 years | NM |
| Debiec et al., 2025 [16] | Multi-institutional (TriNetX Research Network, USA) | Retrospective propensity-matched cohort | 2690 (GLP-1 RA group) + 2690 (control) | Adults with IIH and obesity | NM | NM (obese women predominate per disease definition) | NM | 100% (obesity required) | IIH diagnosis; obesity; treated with GLP-1 RA vs untreated | NM |
| Wills et al., 2025 [17] | USA (Cleveland Clinic | Retrospective cohort | 97 | Adults with obesity and IIH | 46.7 (median) | 98% | 48.3 (median) | 100% (obesity required) | Existing diagnosis of | NM |

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| | health system) | | | undergoing bariatric surgery | | | | | IIH; primary bariatric surgery between 2005–2023 | |
| Elliot et al., 2021 [18] | England (NHS single-payer system) | Economic modeling (Markov) | Modelled population (IIH: WT trial data) | Women with obesity-related IIH | NM (childbearing age) | 100% | NM | 100% (obesity required) | Women of childbearing age with obesity-related IIH | NM |
| Deveci & Kabeloğlu, 2024 [19] | NM | Cross-sectional | 60 IIH, 120 controls | Adults with IIH and age/gender-matched controls | NM (no significant difference vs controls, p=0.437) | NM (no significant difference vs controls, p=0.716) | NM | Significantly higher % of obese subjects in IIH group (p<0.001) | IIH diagnosis; age and gender matched controls | NM |

*NM = Not mentioned.*

**Table 2:** Outcome measures and main findings of included studies**.**

| **Study (Author, Year)** | **Outcome Measures** | **Intervention/Exposure** | **Comparator** | **Key Findings** | **Statistical Methods** | **GRADE Certainty** | **Risk of Bias Judgment** |
|---|---|---|---|---|---|---|---|
| Apperley et al., 2022 [14] | Clinical presentation (headache, ocular signs), BMI SDS | Obesity | N/A (case series) | 66.6% presented with headaches and ocular signs; mean BMI SDS +2.5 | Descriptive | Very low | Moderate |
| Bedir & Nuhoğlu, 2026 [15] | CSF opening pressure, papilledema grade, visual deficit | Obesity, CSF pressure | N/A (prognostic) | CSF opening pressure correlated with papilledema grade (r=0.41, p=0.04); elevated CSF pressure predicted residual | Correlation, regression | Very low | Moderate |

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|---|---|---|---|---|---|---|---|
| | | | | visual deficit (OR 3.2, 95% CI 1.1–9.1, p=0.04) | | | |
| Debiec et al., 2025 [16] | Neurosurgical intervention (VSS, VPS, ONSF) | GLP-1 RA use | Untreated controls | GLP-1 RA use associated with higher odds of VSS at 5 weeks (OR 2.40; 95% CI 1.44–4.00; p=0.0005) and 6 months (OR 2.31; 95% CI 1.10–4.86; p=0.0233); higher odds of VPS at 5 weeks (OR 3.34; 95% CI 2.00–5.61; p=0.0001) and 6 months (OR 2.51; 95% CI 1.35–4.68; p=0.0026) | Propensity score matching; generalized linear model | Very low | Low |
| Wills et al., 2025 [17] | Headache, papilledema, visual field deficits, visual symptoms, lumbar opening pressure, medication usage | Bariatric surgery | Pre-surgery vs. post-surgery (within-subject) | Median total weight loss 24% at 3 years; mean lumbar opening pressure reduced from 34.8 ± 8.2 to 24.2 ± 7.6 cm CSF (mean reduction 10.7 cm; 95% CI 4.7–16.6; p=0.003); significant reduction in acetazolamide/topiramate dosage and number of medications (p<0.001) | Paired comparisons | Low | Low |
| Elliot et al., 2021 [18] | Costs (GBP), QALYs, time horizons (5, 10, 20 years) | Bariatric surgery | Community weight management | Over 20 years, bariatric surgery was dominant (cost-saving) with savings of £49,500 and additional 1.16 QALYs; 98% probability of being cost-effective | Markov model; probabilistic sensitivity analysis | Low | Low-moderate |
| Deveci & Kabeloğlu, 2024 [19] | BMI, Berlin questionnaire, STOP-BANG, OSA risk | Obesity | IIH patients vs. age/gender-matched controls (1:2) | Significantly higher % of obese subjects in IIH group (p<0.001); IIH group had higher risks for OSA on both questionnaires; multivariate regression: obesity | Multivariate logistic regression | Very low | Low |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| | | | | was the only independent predictor of high-risk Berlin score | | | |

VSS = venous sinus stenting; VPS = ventriculoperitoneal shunting; ONSF = optic nerve sheath fenestration; QALY = quality-adjusted life year; CSF = cerebrospinal fluid; OSA = obstructive sleep apnea. All CSF pressures converted to cm $H_2O$ where applicable.

**Table 3:** GRADE certainty of evidence assessment**.**

| **Outcome** | **Number of Studies** | **Study Design** | **Risk of Bias** | **Inconsistency** | **Indirectness** | **Imprecision** | **Publication Bias** | **Certainty (GRADE)** |
|---|---|---|---|---|---|---|---|---|
| Obesity as risk factor for IIH | 3 cohorts, 1 cross-sectional | Observational | Moderate to serious | Low | Direct | Serious | Suspected | **Very low** |
| Bariatric surgery → ICP reduction | 1 cohort, 1 economic model | Observational | Low to moderate | Low | Direct | Serious | Suspected | **Low** |
| Bariatric surgery → visual outcomes | 1 cohort | Observational | Low | Low | Direct | Serious | Suspected | **Low** |
| GLP-1 RA → neurosurgical outcomes | 1 cohort | Observational | Low | Cannot assess (single study) | Direct | Very serious | Suspected | **Very low** |
| CSF pressure → visual deficit | 1 chart review | Observational | Moderate | Cannot assess | Direct | Serious | Suspected | **Very low** |
| Obesity as predictor of OSA risk | 1 cross-sectional | Observational | Low | Cannot assess | Direct | Serious | Suspected | **Very low** |
| Cost-effectiveness of bariatric surgery | 1 economic model | Modeling | Low-moderate | Cannot assess | Direct | Serious | Suspected | **Low** |

obesity were observed in 34.6% and 19.2% of cases, respectively, with a female predominance of 53.8%. These paediatric findings are consistent with a large retrospective study by Sheldon and colleagues [20], who demonstrated that 50.0% (95% CI: 29.9–70.1%) of paediatric IIH patients were obese, with obesity rates of 30.7% in children aged 12 years and younger compared to 69.2% in those aged 13 years and older, suggesting that the obesity-IIH association strengthens with age and pubertal status [20]. Furthermore, childhood obesity is strongly linked to a higher risk for paediatric IIH among adolescents, particularly among extremely obese White non-Hispanic adolescent girls [21]. This suggests that the childhood obesity epidemic will be linked to higher morbidity from IIH.

In adult populations, the relationship between obesity and IIH is even more pronounced. Debiec et al. [16] included 5,380 adult patients with IIH and obesity in their propensity-matched cohort study, all of whom had a BMI consistent with obesity. Wills et al. [17] studied 97 patients (98% female) with a median BMI of 48.3 kg/m², demonstrating severe obesity in their cohort. Elliot et al. [18] focused on women of childbearing age with obesity-related IIH, a population that mirrors the typical demographic profile of the disease. Deveci and Kabeloglu [19] compared 60 IIH patients with 120 age- and gender-matched controls, finding a significantly higher percentage of obese subjects in the IIH group ($p < 0.001$). These observations are further supported by the landmark population-based study conducted by Kilgore et al. [22], which reassessed the epidemiology of idiopathic intracranial hypertension (IIH) in Olmsted County, Minnesota, over the period from 1990 to 2014. The investigators reported an age- and sex-adjusted annual incidence of 1.8 cases per 100,000 population, with a significant increase over time from 1.0 per 100,000 during 1990–2001 to 2.4 per 100,000 during 2002–2014 ($p = 0.007$). Among women aged 15–44 years, the incidence reached 22.0 per 100,000 in those with obesity, compared with 6.8 per 100,000 in the overall female population of the same age group. Moreover, IIH incidence showed a strong positive association with obesity prevalence across Minnesota ($R^2 = 0.70$, $p = 0.008$). More recent epidemiological studies have reinforced this relationship, demonstrating that women with obesity have an estimated IIH prevalence of 180 per 100,000 and an annual incidence of 23.5 per 100,000, whereas women with a normal BMI have substantially lower rates, with a prevalence of 13.2 per 100,000 and an incidence of only 1.6 per 100,000 per year [23]. These epidemiological data confirm a dose-response relationship between increasing BMI and IIH risk, with the strongest association observed in women of reproductive age. Our systematic review revealed multiple investigations examining the effects of weight-reduction strategies on disease-related endpoints in IIH, with bariatric surgery emerging as the intervention supported by the most compelling evidence. In the retrospective analysis conducted by Wills and colleagues [17], participants attained a median total weight loss of 24% over a median follow-up duration of 3.0 years. This considerable decrease in body mass was paralleled by notable enhancements in headache severity, optic disc swelling (papilledema), visual complaints, and perimetric visual field deficits. Furthermore, the mean CSF opening pressure dropped from a preoperative value of 34.8 ± 8.2 cm CSF to 24.2 ± 7.6 cm CSF postoperatively, reflecting an average reduction of 10.7 cm CSF (95% CI: 4.7–16.6; $p = 0.003$). The reliance on pharmacological therapy likewise diminished significantly; both the prescribed dosages of acetazolamide and topiramate and the total count of IIH-directed medications were meaningfully lowered following bariatric surgery ($p < 0.001$) [17]. The long-term economic benefits of bariatric surgery have likewise been demonstrated. Elliot et al. [18] performed an economic modelling analysis and concluded that bariatric surgery was the dominant treatment strategy compared with community-based weight management over a 20-year time horizon. The surgical approach was associated with an estimated cost saving of £49,500 while yielding an additional 1.16 quality-adjusted life years (QALYs), with a 98% probability of representing the most cost-effective intervention. These findings are further strengthened by high-level evidence from systematic reviews and meta-analyses. Radboy et al. [24] analyzed 15 studies involving 887 patients and demonstrated that metabolic and bariatric surgery (MBS) significantly reduced intracranial pressure (ICP), with a pooled mean difference of −15.79 cm $H_2O$. Surgical treatment was also associated with substantial improvement in headache, papilledema, and visual disturbances. In a second systematic review and single-arm meta-analysis, the same investigators evaluated nine studies comprising 229 patients and reported pooled improvement rates of 84.6% for headache, 83.3% for visual field defects, 88.7% for pulsatile tinnitus, and 98.1% for visual symptoms. Resolution of papilledema approached 98%, while discontinuation of IIH-related medications was achieved in 63.3% of patients following bariatric surgery [24]. Robust evidence from randomized controlled trials further

supports these observations. The landmark IIH trial conducted by Mollan et al. [25] provided Level 1 evidence demonstrating that bariatric surgery was superior to community weight management in reducing ICP among women with active IIH and a BMI ≥ 35 kg/m². Compared with the non-surgical group, the bariatric surgery group experienced an adjusted mean ICP reduction of 6.0 cm CSF at 12 months (95% CI: −9.5 to −2.4; p = 0.001), which increased to 8.2 cm CSF after 24 months (95% CI: −12.2 to −4.2; p < 0.001). These reductions in ICP occurred alongside significantly greater weight loss among surgically treated patients [25]. The beneficial effects of bariatric surgery have also been confirmed by Abbottet al. [26]. Their systematic review demonstrated that patients lost an average of 27.2–27.8 kg within 24 months (Level 1 to 1++ evidence). Moreover, the greatest reduction in ICP was observed at the same follow-up interval, with a mean decrease of 11.9 cm $H_2O$ (Level 1++). Importantly, the review highlighted a strong positive relationship between the degree of weight reduction and the magnitude of ICP improvement, emphasizing weight loss as a key therapeutic target in the management of IIH [26]. Additionally, our systematic review encompassed a recent investigation by Debiec and colleagues [16] that assessed the potential role of GLP-1 receptor agonists (GLP-1 RAs) in reducing the requirement for surgical management among patients with IIH. Following propensity-score matching, each cohort comprised 2,690 individuals. Contrary to the findings of several meta-analyses, Debiec et al. [16] reported that GLP-1 RA use was associated with increased odds of neurosurgical intervention. Specifically, treatment with GLP-1 RAs was associated with higher odds of venous sinus stenting at 5 weeks (OR 2.40; 95% CI: 1.44–4.00; p = 0.0005) and 6 months (OR 2.31; 95% CI: 1.10–4.86; p = 0.0233), as well as higher odds of ventriculoperitoneal shunting at 5 weeks (OR 3.34; 95% CI: 2.00–5.61; p = 0.0001) and 6 months (OR 2.51; 95% CI: 1.35–4.68; p = 0.0026). This discrepancy may be due to confounding by indication, differences in patient selection, or the observational nature of the study. Therefore, no firm conclusion can be drawn regarding the benefit of GLP-1 RAs in reducing neurosurgical needs. While other systematic reviews [27- 29] suggest potential benefits, these are based on low-certainty evidence. Randomized controlled trials are urgently needed. The therapeutic potential of GLP-1 receptor agonists is further supported by accumulating evidence from recent systematic reviews and meta-analyses. Ognard et al. [27] synthesized data from nine clinical studies, including one randomized controlled trial and eight observational studies, and found that GLP-1 RA therapy consistently improved headache frequency, promoted weight loss, and reduced the need for acetazolamide treatment and neurosurgical procedures. Notably, findings from the randomized trial demonstrated that exenatide produced a rapid reduction in intracranial pressure (ICP), with measurable effects occurring within hours after administration. Stefanou et al. [28] provided further evidence via a meta-analysis of 2 trials and 2 registry studies (1,550 IIH patients; 768 on GLP-1/dual GIP/GLP-1 agonists). Compared to standard care, GLP-1 RAs reduced papilledema risk (RR 0.25; p < 0.01) and visual disturbance/blindness risk (RR 0.41; p = 0.03). Headache risk showed a non-significant favorable trend (RR 0.61; p = 0.08). Monthly headache days decreased at 3 months (MD −3.64; p < 0.01) and at final follow-up (MD −4.82; p = 0.02) [28]. Findings in agreement were detailed by Phutinart and colleagues [29] in their meta-analysis, which comprised eight studies and a total of 18,423 IIH patients. The pooled analysis revealed that GLP-1 receptor agonists were associated with a notable improvement in headache outcomes at 6 months, a decreased risk of persistent papilledema at 3 months, and a lower probability of refractory IIH over the observation period, with a relative risk of 0.81 at 24 months. Of particular note, the observed advantages of GLP-1 RA therapy cannot be ascribed exclusively to weight loss. Preclinical evidence indicates that these agents have the capacity to directly inhibit cerebrospinal fluid (CSF) production via stimulation of GLP-1 receptors located in the choroid plexus, suggesting that both weight-dependent and weight-independent pathways are likely involved in their therapeutic effects [29]. The studies incorporated in this review provide evidence supporting a close relationship between CSF opening pressure, obesity, and visual outcomes in IIH patients. In their investigation, Bedir and Nuhoğlu [15] reported a mean CSF opening pressure of 33.5 ± 8.5 cm $H_2O$ and established a significant positive correlation with papilledema severity. Their analysis further demonstrated that higher CSF pressure independently predicted persistent visual loss (OR 3.2; 95% CI: 1.1–9.1; p = 0.04), reinforcing the notion that elevated ICP is a key predictor of visual deterioration. Al Khalidi et al. [30] reported analogous results from their study of 104 IIH patients (96 female, 8 male), with an average age of 31.88 ± 13.73 years. Their Pearson correlation

analysis revealed a significant association between BMI and CSF opening pressure ($p = 0.0001$). However, age and sex did not correlate significantly with CSF pressure, implying that obesity has a stronger effect on intracranial pressure than patient demographics. The association between excess body weight and elevated CSF pressure was further supported by Whiteley et al. [31]. In their study, overweight individuals exhibited significantly higher CSF opening pressures than participants with normal body weight ($37.8 \pm 10$ cm $H_2O$ versus $32.4 \pm 9.8$ cm $H_2O$). Additionally, increasing papilledema severity was found to correlate with higher CSF opening pressure, reinforcing the relationship between elevated intracranial pressure and optic nerve involvement. Further evidence emphasizing the importance of weight reduction was provided by Varughese and Nayak [32], who demonstrated that the magnitude of weight loss was directly associated with the expected reduction in intracranial pressure (ICP) among women with active IIH and a BMI > 35 kg/m². Their findings indicated that achieving normalization of ICP to ≤ 25 cm CSF required an average weight reduction equivalent to 24% of baseline body weight over a 12-month period. Collectively, these findings highlight the critical role of early diagnosis, timely intervention, and multidisciplinary management aimed at achieving substantial weight loss and preventing irreversible visual deterioration, consistent with the conclusions of Bedir and Nuhoğlu [15]. Although obesity is well established as the principal modifiable risk factor for idiopathic intracranial hypertension (IIH), the biological mechanisms responsible for this association remain incompletely defined. Several complementary hypotheses have been proposed to explain the relationship between excess adiposity and elevated intracranial pressure. In their comprehensive review, Markey et al. [33] summarized current evidence regarding the pathogenic pathways underlying IIH, emphasizing the contribution of obesity-related hormonal disturbances, neuroendocrine dysfunction, and chronic inflammatory processes. The authors also highlighted the potential involvement of 11β-hydroxysteroid dehydrogenase type 1 (11β-HSD1), an enzyme that regulates glucocorticoid metabolism, as a possible mediator of disease development [33]. Support for an inflammatory element in IIH was further supplied by Samancı et al. [34], who assessed circulating adipokine and cytokine levels in IIH patients and healthy controls matched for age, sex, and BMI. The investigators documented significantly higher IL-1β levels in the IIH group, whereas IL-8 and TNF-α concentrations were significantly diminished compared with controls. In addition, patients with disease recurrence demonstrated significant alterations in IL-1β, IL-8, and TNF-α relative to healthy individuals, indicating that these inflammatory mediators may possess prognostic significance for recurrence risk. Overall, these observations are consistent with the hypothesis that obesity-driven low-grade systemic inflammation contributes to IIH pathogenesis, although the exact molecular pathways involved warrant further exploration [34]. Our review also identified evidence linking idiopathic intracranial hypertension with obstructive sleep apnoea (OSA). Deveci and Kabeloğlu [19] reported that patients with IIH were significantly more likely to be classified as having a high risk of OSA according to both the Berlin and STOP-BANG screening questionnaires. Multivariable logistic regression analysis demonstrated that obesity was the only independent predictor of a high-risk Berlin questionnaire score. These findings indicate that obesity represents a shared risk factor for both IIH and OSA and suggest that the coexistence of these conditions may contribute to increased morbidity and mortality. Consequently, the authors recommended that OSA screening may be considered in patients diagnosed with IIH, though evidence is limited [19]. This recommendation is supported by accumulating evidence indicating that sleep-disordered breathing may exacerbate intracranial hypertension through mechanisms such as nocturnal hypercapnia, impaired cerebral venous drainage, and reduced cerebrospinal fluid absorption, thereby further increasing intracranial pressure. Using the GRADE approach, the certainty of evidence was rated as very low for most outcomes, including the association between obesity and IIH, the effect of GLP-1 RAs on neurosurgical interventions, and the relationship between CSF pressure and visual deficits. Bariatric surgery outcomes and cost-effectiveness were rated as low certainty. Reasons for downgrading included observational study designs, risk of bias, imprecision, and suspected publication bias. Consequently, all clinical recommendations should be considered conditional. Limitations: Several limitations warrant consideration. Most included studies were retrospective observational cohorts, with few RCTs (the IIH:WT trial was excluded due to abstract availability), introducing selection/recall bias, limiting causal inference, and yielding moderate-to-serious bias in multiple cohorts. Substantial heterogeneity in sample sizes (18–5,380), outcome measures, follow-up durations (averaging 2–3 years),

and definitions of obesity/IIH severity precluded robust comparisons and assessment of long-term sustainability. Publication bias and inadequate adjustment for confounders (medications, comorbidities, socioeconomic status) were also noted. Lastly, findings are largely generalisable only to obese females, and the absence of standardised weight-loss protocols restricts definitive clinical recommendations. The certainty of evidence is very low for most outcomes due to the observational design, heterogeneity, and risk of bias. No randomized controlled trials were included. The GLP-1 RA findings are based on a single observational study with conflicting results. Publication bias and confounding cannot be excluded.

## Conclusion

Obesity, especially in reproductive-aged women, is a key IIH risk factor; higher BMI correlates with elevated CSF pressure and worse vision. Bariatric surgery may improve ICP, symptoms, and cost-effectiveness in eligible patients (BMI ≥35 kg/m², ≥24% weight loss). GLP-1 agonist evidence is conflicting, and certainty is very low. Obesity independently predicts high-risk OSA screening scores. Clinicians should adopt multidisciplinary care with conditional weight management, ophthalmological monitoring, and early intervention to prevent visual loss; OSA screening may be considered. Future RCTs and mechanistic studies are needed.

## Conflict of Interest

None

## Funding

None

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