Case report

Successful therapeutic response of intravenous immunoglobulin in Guillain-Barre syndrome with onset more than two weeks: a case report

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Abstract

Guillain-Barré syndrome (GBS) has a global incidence of 1–2 cases per 100,000 individuals annually and remains the most common cause of acute flaccid paralysis. Standard treatment includes plasma exchange within 4 weeks or intravenous immunoglobulin (IVIg) within 2 weeks of symptom onset. However, select late-presenting cases may still benefit from IVIg treatment. Here, we report a case of a 17-year-old male who presented with progressive bilateral limb weakness and paresthesia that began approximately 3 weeks prior to admission. Furthermore, he experienced a brief syncope episode and defecation syncope, thus indicating possible autonomic involvement. On hospital day 3, after shared decision-making, the family opted for IVIg over plasma exchange. The laboratory tests on admission and before IVIg treatment showed normal albumin and elevated c-reactive protein (CRP) levels, which resulted in a low CRP-albumin ratio (CAR = 0.12). Although the value of CAR has not been elucidated in GBS, studies on Kawasaki disease suggest that a lower CAR may be associated with a favorable response to IVIg treatment. The patient received IVIg at 0.4 g/kg/day for 5 days, starting on hospital day 4 (approximately day 24 of symptom onset). By day 3 of IVIg therapy, the patient exhibited improved limb strength and reduced paresthesia. Moreover, the GBS disability score improved from 4 to 2 upon completion of treatment. This case supports the potential benefit of IVIg beyond the conventional 2-week window in GBS, particularly in patients exhibiting autonomic symptoms. The use of CAR as a potential supportive marker for predicting the response of patients with GBS to IVIg treatment warrants further investigation.

Keywords: Albumin, c-reactive protein, Guillain-Barre syndrome, immunoglobulins, intravenous, polyradiculoneuropathy.

Guillain-Barré syndrome (GBS) is a syndrome that manifests as acute inflammatory demyelinating polyradiculoneuropathy, making it the most common cause of acute paralysis, with an annual global incidence of approximately 1–2 per 100,000 people.[1,2] GBS occurs more frequently in males than in females, and its incidence increases with age, although individuals from all age groups can be affected.[3, 4] Patients with GBS generally present with symptoms of sensory weakness in the legs, which progresses to the arms and cranial muscles. However, the clinical presentation of the disease is heterogeneous and includes several distinct clinical variants.[2] The exact cause of GBS is still unknown, but an immune response to a preceding infection, such as gastroenteritis or respiratory tract infections, often triggers its onset. Several pathogens have been associated with the onset of GBS, including *Campylobacter jejuni*, cytomegalovirus, hepatitis E virus, *Mycoplasma pneumoniae*, Epstein-Barr virus, Zika virus, and

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coronavirus.[2, 4-7] GBS occurs because of an immune response that cross-reacts with peripheral nerve components, involving molecular mimicry. This immune response targets either the myelin or axonal components of the peripheral nerves, which leads to the demyelinating or axonal forms of GBS.[1,3] In some cases, there is evidence that suggests a potential genetic component involved in the development of GBS.[6]

Approximately 70.0% of patients with GBS experience dysautonomia, characterized by tachycardia or bradycardia, facial flushing, alternating hypertension and hypotension, orthostatic hypotension, anhidrosis or diaphoresis, and urinary retention, whereas gastrointestinal autonomic manifestations include diarrhea or constipation. Dysautonomia is critical to treat, as it is associated with a 5.0%–7.0% risk of sudden death.[1, 2, 8]

The first therapy proven effective for GBS was plasmapheresis, or plasma exchange (PE). PE is administered within 4 weeks of the onset of weakness for non-ambulant patients and 2 weeks for ambulant patients.[2, 3, 9-11] Subsequent studies have revealed that intravenous immunoglobulin (IVIg) therapy has nearly the same efficacy as PE for non-ambulant patients if started within 2 weeks of weakness onset.[9, 12] Based on these studies, IVIg is recommended within 2 weeks of weakness onset; however, the effectiveness of IVIg administration beyond this period remains uncertain.[2, 9, 10] Another report suggested that IVIg should be given within 2 weeks of symptom onset in patients with GBS who require assistance with walking or within 4 weeks for those with neuropathic symptoms.[11] Further assessment is also required to evaluate the potential risk of IVIg treatment failure to achieve optimal and efficient therapy outcomes for the patient. This case report aims to highlight that the IVIg administration, more than 2 weeks after weakness onset, supplemented with albumin and c-reactive protein (CRP) testing, as well as the calculation of the CRP-albumin ratio (CAR) to predict the potential risk of IVIg treatment failure, demonstrates therapeutic improvement in patients with GBS.

Case presentation

A 17-year-old male was referred for hospitalization in January 2024 with progressive bilateral leg weakness, an unsteady gait, and tingling in his hands and feet for the past 3 weeks, which had worsened over the preceding 2 weeks and was preceded by flu-like symptoms. He had a history of allergy to sulfonamide antibiotics. The patient weighed 48.5 kg, with a height of 170 cm.

His vital sign examination revealed that his blood pressure was 120/70 mmHg, pulse rate was 102 beats per minute, temperature was 36.5°C, oxygen saturation was 97.0%, and he was fully conscious. Furthermore, his cranial nerve examination was within normal limits, but his motor examination revealed that upper and lower motor strength was decreased. There was sensory paresthesia in his hands and feet, and hypesthesia from T_{12} to L_1. All deep tendon reflexes were decreased.

Laboratory tests performed on the first day of hospitalization showed that leukocytes, neutrophils, lymphocytes, monocytes, eosinophils, basophils, red blood cell count, hemoglobin, urea, and blood urea nitrogen levels were within normal range; immunoglobulin% was 0.004; albumin was 4.7 g/dL; CRP was 5.8 mg/L; aspartate aminotransferase was 27 U/L; and alanine aminotransferase was 73 U/L. Moreover, the rheumatoid factor, Cytobead ANA 2, HBsAg (CMIA), and total anti-HCV tests were negative. Three days later, before IVIg administration, further laboratory tests revealed the patient's albumin was normal and CRP was slightly increased. Furthermore, electromyography and nerve conduction velocity tests revealed axonal and demyelinating sensorimotor polyneuropathy, but magnetic resonance imaging and magnetic resonance angiography of the head were within normal limits. The patient and family declined a lumbar puncture after being informed about the procedure and its purpose. On the third day of hospitalization, following a discussion of the available treatment options, the patient's family opted for IVIg over PE, and IVIg therapy was initiated on hospital day 4 at a dose of 0.4 g/kg/day**.** The patient was administered 19.4 g/day (rounded up to 20 g/day) for 5 consecutive days. On the first day of IVIg administration, 30 min after the infusion, the patient complained of nausea, burped several times, experienced shortness of breath, had difficulty breathing comfortably due to coughing, and had an itchy throat. There was no itching in other areas, as well as no swelling or sensation of choking. The patient was given codeine as needed for the cough.

On the second day, the patient was premedicated with diphenhydramine before IVIg administration. The patient reported a reduction in coughing but had not

yet had a bowel movement. On the third day, the patient experienced headaches, nausea, epigastric pain, and constipation**.** However, there was an improvement in hand weakness, and the weakness in both legs exhibited slight improvement. The unsteadiness of the patient's walking has decreased. However, tingling in the ring and little fingers of both hands, as well as the soles of both feet, persisted.

On the fourth day, the patient experienced a vasovagal response, specifically defecation syncope. The patient initially reported abdominal cramps and the urge to defecate. While straining, the patient became pale, had cold sweats, and a non-palpable pulse. The patient was transferred to the bed and soon thereafter opened his eyes as he regained consciousness. The patient reported ringing in his ears but no dizziness. Vital sign assessment and electrocardiography were within normal limits, with a random blood glucose level of 92 mg/dL. On the same day, the patient complained of slight dizziness and vomited once, with the vomitus containing milk.

On the fifth day, the patient reported a 6-day episode of constipation. Weaknesses in both legs persisted, and the patient continued to walk with an unsteady gait, while the weakness in both hands improved. Tingling in the ring and little fingers of both hands and the soles of both feet remained. On the following day, the patient was able to defecate after receiving a glycerin enema.

Two days after IVIg treatment completion, the weakness in the hands and feet improved, and the tingling in the ring and little fingers of both hands and the soles of both feet lessened. The patient was discharged and approved for outpatient care. A week later, the patient experienced further improvement in motor and sensory symptoms. One month later, during a follow-up visit, the patient exhibited significant improvement in hand and leg weakness, and the tingling in the hands and feet had become minimal. A visual summary of the patient's clinical presentation is shown in **Figure 1.**

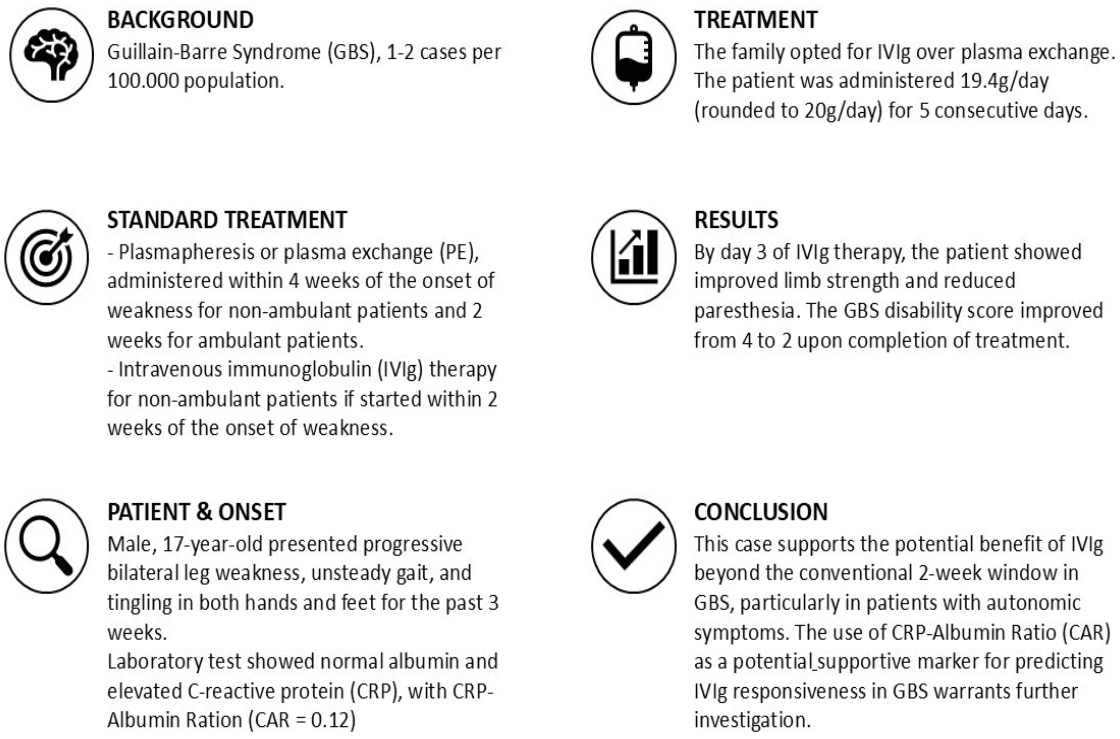

Figure 1. Summary of a GBS case in a 17-year-old patient, outlining clinical presentation, treatment with IVIg, outcomes, and the potential role of CAR as a predictive marker. CAR, c-reactive protein albumin ratio; GBS, Guillain-Barré syndrome; IVIg, intravenous immunoglobulin.

Discussion

GBS is characterized by inflammation that manifests as demyelinating polyradiculopathy, which leads to symmetrical ascending weakness of the extremities, sensory disturbances, and the absence or reduction of deep tendon reflexes.[13] Recommended treatment options for GBS include IVIg at a dosage of 0.4 g/kg body weight/day for 5 days and PE at a rate of 200–250 mL plasma/kg body weight over five sessions. Both treatment options demonstrate comparable effectiveness.[2, 3, 9, 14, 15] The mechanism of action for PE involves the non-selective removal of immunoglobulins, complement proteins, and cytokines, all of which may play a role in the pathogenesis of GBS. In contrast, IVIg acts via pleiotropic immunomodulatory effects, including the neutralization of autoantibodies or cytokines, saturation of macrophage Fc receptors, or inhibition of complement activation that induces further nerve damage.[10, 14, 16, 17] According to a Cochrane review comparing the effectiveness of PE and IVIg, the risk ratio (RR) for the improvement in disability grade was 1.1 (95% confidence interval (CI) 0.94–1.23), indicating that the effectiveness of both therapies is not significantly different.[3]

In addition to comparing their effectiveness, the literature indicates that the side effects associated with the use of IVIg and PE do not differ significantly, with an RR of 0.84 (95% CI 0.54–1.30), and that there were fewer side effects in the IVIg group compared to those of the PE group.[3, 15, 18] The incidence of side effects, such as pneumonia, atelectasis, thrombosis, and hemodynamic difficulties, was more frequent in patients receiving PE than in those receiving IVIg. Among patients who received PE, 16 out of 73 (22.0%) experienced multiple complications compared to the 5 out of 74 (7.0%) patients who received IVIg.[11,19] Although IVIg is more expensive, it is easier and more comfortable to administer, and it is also considered safer for patients with dysautonomia.[3, 15]

Other outcome measurements include the duration of stay in the intensive care unit (ICU) and the ability to walk unaided within 4 weeks after discharge from the ICU, which did not demonstrate significant differences between the therapies.[20] A review of the ease and comfort of the administration of these therapies was conducted by comparing the proportion of participants who discontinued treatment. The likelihood of treatment cessation in the IVIg group was significantly lower compared to that of patients treated with PE, with an RR of 0.11 (95% CI 0.04–0.32).[11] A similar finding was reported in the Cochrane review, which found an RR for treatment discontinuation of 0.14, which indicates that treatment discontinuation was less likely to occur in the IVIg group (95% CI 0.05–0.36).[3] Therefore, it can be concluded that there is a significant difference between the two therapies. This difference may be attributed to the simpler administration of IVIg compared to that of PE, which requires two venous accesses, a PE machine, and specially trained personnel. In contrast, IVIg administration only requires a single peripheral vein and does not necessitate specialized machinery or personnel for the therapy.[21]

Various literature sources indicate that the use of PE is effective within 4 weeks of neuromotor onset, while IVIg is effective within 2 weeks of onset.[16, 22] The efficacy of IVIg therapy initiated beyond 2 weeks post-symptom onset in patients with GBS has not been extensively documented. The selection of therapy is determined based on the availability of medications, adequate personnel, facilities, comorbidities or contraindications in the patient, and patient-related socioeconomic factors.[10, 17] In this case, after the patient's parents considered the side effects of PE and IVIg, they decided to use IVIg therapy despite the symptom onset of GBS being longer than 2 weeks, and a significant improvement in the disability grade was observed.

The choice of IVIg therapy after 2 weeks of GBS symptom onset requires further consideration. Therefore, it is essential to assess the potential for IVIg treatment failure by calculating the patient's CAR. IVIg treatment failure can be attributed to several factors, such as incorrect dosing, inappropriate therapy duration, or IVIg resistance. The term "IVIg resistance" is recognized in patients with Kawasaki disease (KD). IVIg resistance in patients with KD is defined as the persistence of fever for at least 36 h after the completion of IVIg therapy. This occurs in approximately 10%–20% of patients with KD.[23–25] A retrospective cohort study in patients with KD indicated that albumin testing is a potential predictive marker for estimating IVIg resistance.[23, 24, 26] IVIg resistance results in a higher risk of coronary artery lesions in patients with KD and increases the therapeutic burden. In that study, it was found that patients who responded to IVIg therapy had a higher percentage of neutrophils (n %), CRP level, and CAR value compared to those who were resistant to IVIg. The results of multivariate regression analysis indicated that an albumin level of ≤33.4 g/L

(equivalent to ≤ 3.3 g/dL) before IVIg therapy was an independent risk factor for IVIg resistance.[23] Nevertheless, albumin should not be used as a sole factor for determining IVIg resistance in a clinical setting because of its low sensitivity (40.9%); thus, other factors, including the patient's genetics, must be considered.[23]

In addition to predicting the possibility of IVIg treatment failure, CAR calculation and evaluation in patients with GBS may serve as a new inflammatory biomarker. A CAR > 0.21 was independently associated with the occurrence of respiratory failure in patients with GBS, while a CAR > 0.19 was independently linked to poor short-term outcomes. CAR can help identify patients with GBS at high risk for poor prognosis.[27] A study conducted between 2015 and 2019 indicated that CAR assessment in patients with GBS can be employed as a new inflammatory marker to predict neurological outcomes.[27] In this case, IVIg was selected over PE following shared decision-making with the patient's family. The decision was influenced by the presence of autonomic symptoms, including defecation syncope and prolonged constipation, which may indicate more severe clinical involvement. In addition, a low CAR = 0.12, measured before IVIg initiation, may have suggested a favorable response. The patient exhibited improvements in limb strength and paresthesia by the third day of IVIg therapy, with continued progress noted during their 1-week and 1-month follow-ups.

Conclusion

GBS is a rare syndrome that requires effective and immediate therapy selection to prevent the deterioration of the patient's condition. IVIg therapy in patients with GBS has been shown to be effective when used within 2 weeks after GBS symptom onset, while the use of IVIg more than 2 weeks post-onset has not been widely discussed. The possibility of failure and prognosis of IVIg treatment needs to be considered before administration, which can be assessed by evaluating the CRP, albumin, and calculated CAR levels. In this case, IVIg therapy administered more than 2 weeks post-onset resulted in improvements in motor and sensory functions, with minimal side effects. However, further research is required regarding the risk factors for IVIg treatment failure, specifically in GBS.

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Conflict of interest statement

Each of the authors has completed an ICMJE disclosure form. None of the authors declare any potential or actual relationship, activity, or interest related to the content of this article.

Data sharing statement

All data generated or analyzed for the present report are included in this published article. Further details are available from the corresponding author on reasonable request after deidentification of the patient whose data are included in the report.

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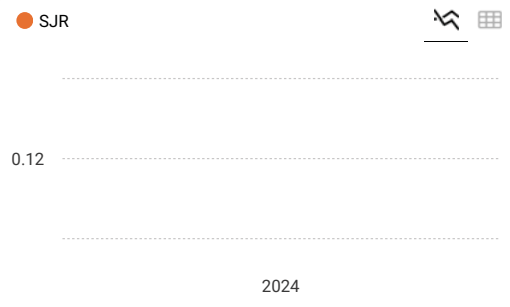
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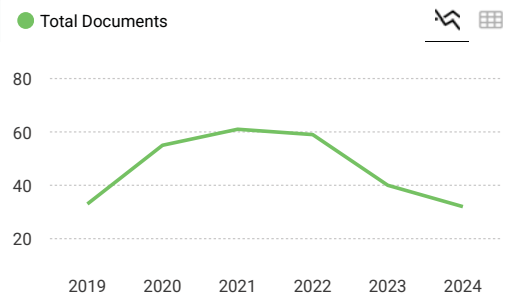
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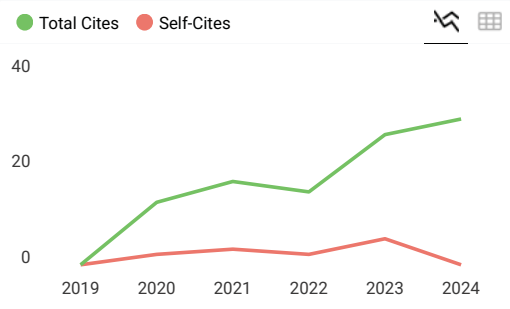
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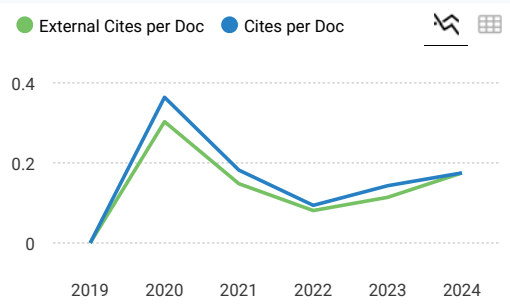
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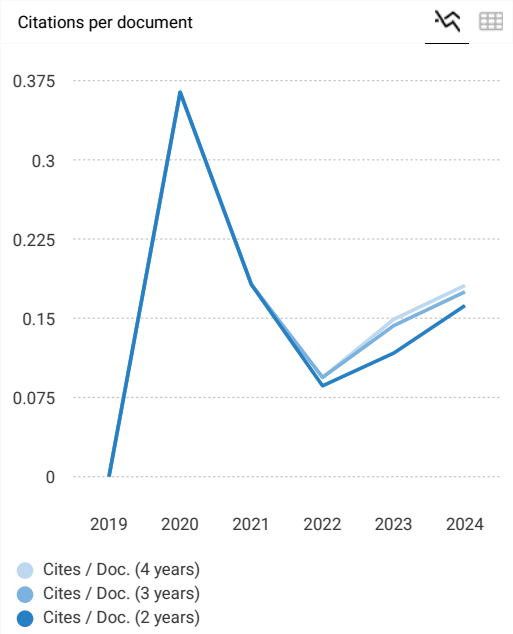
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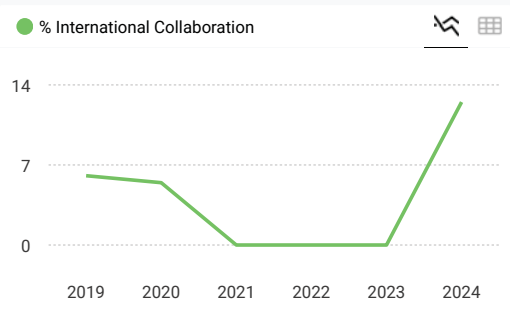
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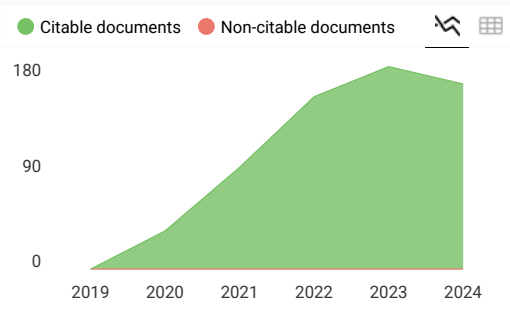
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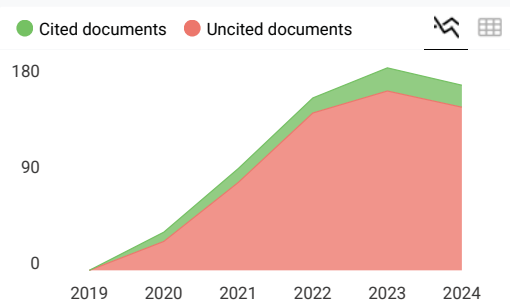
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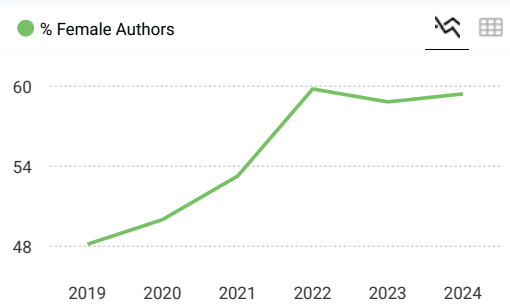
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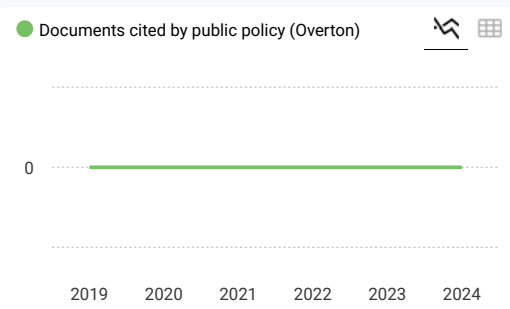
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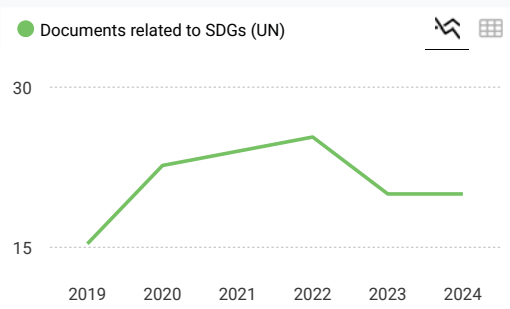
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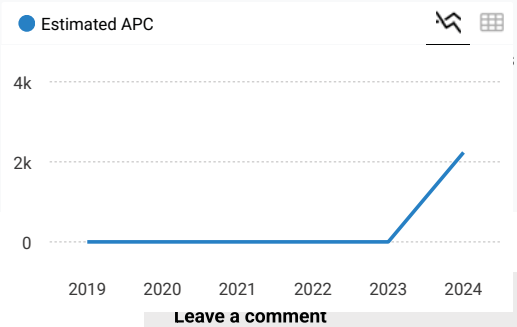

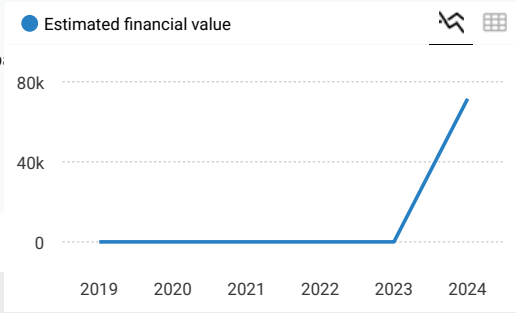

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Source details

Chulalongkorn Medical Journal

Years currently covered by Scopus: from 2019 to 2025

Publisher: Chulalongkorn University Printing House

ISSN: 2651-2343 E-ISSN: 2673-060X

Subject area: Medicine: General Medicine

Source type: Journal

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0.3

SJR 2024
0.115

SNIP 2024
0.085

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CiteScore 2024

$$0.3 = \frac{51 \text{ Citations } 2021 - 2024}{192 \text{ Documents } 2021 - 2024}$$

Calculated on 05 May, 2025

CiteScoreTracker 2025

$$0.3 = \frac{49 \text{ Citations to date}}{162 \text{ Documents to date}}$$

Last updated on 05 October, 2025 • Updated monthly

CiteScore rank 2024

Category	Rank	Percentile
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