

ANTON SYNDROME : THE PARADOXICAL BLINDNESS

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ABSTRACT :

Anton Syndrome (AS) is a rare neurological disorder marked by cortical blindness and visual anosognosia, where patients are unaware of their blindness. It typically occurs in elderly individuals following strokes but can also result from trauma, tumors, infections, or metabolic issues—especially in younger patients. Diagnosing AS is challenging due to the patient's denial of visual loss. Neuroimaging (MRI/CT) is key for confirming occipital lobe damage and ruling out other conditions. Treatment focuses on the underlying cause, with supportive care, cognitive therapy, and visual training aiding rehabilitation. Prognosis varies by age, cause, and severity. Some reversible cases show visual recovery, while others may lead to permanent blindness. Given its rarity, more research and clinician awareness are needed to improve diagnosis and patient outcomes.

INTRODUCTION :

Cortical blindness is an uncommon neurological condition characterized by a bilateral loss of vision due to damage in the occipital cortex. Anton-Babinski Syndrome (ABS), also referred to as Anton Syndrome, is a rare condition in which individuals experience visual anosognosia, meaning they are unaware of or deny their blindness. This syndrome is often accompanied by confabulation, where individuals create false memories despite clear evidence of visual impairment and cortical blindness. Unlike disorders arising from ocular issues, ABS stems from brain damage, making it a form of neurological visual dysfunction.

The earliest known account of ABS dates back to ancient Rome. Seneca, a philosopher and politician, described **Harpaste**, a blind slave who refused to accept her condition and blamed her inability to see on the darkness of her surroundings, persistently asking to be moved elsewhere. This account outlines key features of ABS, such as sudden blindness and **anosognosia**, while cognitive functions remain largely intact. Centuries later, during the Renaissance, Michel de Montaigne, a French writer, described a nobleman who similarly denied his blindness, providing another historical reference to visual anosognosia. Modern exploration of ABS began in 1895 when Gabriel Anton, an Austrian psychiatrist and

neurologist, studied Juliane Hochriehser, a 69-year-old dairymaid who exhibited cortical deafness and anosognosia as a result of damage to both temporal lobes.

Anton documented other cases where patients were either blind or deaf but unaware of their deficits. In 1914, Joseph François Babinski, a French-Polish neurologist, introduced the term "anosognosia" to describe a lack of recognition of one's impairments, initially focusing on individuals with hemiplegia. By 1920, a neurologist named Meyer linked ABS to infarction in the occipital lobe, proposing that the syndrome was caused by the compression of branches of the posterior cerebral artery. His work provided a foundational understanding of the vascular origins of this condition.(1)(15)(16)(18)

ETIOLOGY :

Anton-Babinski Syndrome (ABS) is a rare and fascinating neurological disorder defined by cortical blindness alongside anosognosia—a condition where individuals either fail to recognize or outright deny their blindness, even when their visual impairment is undeniable. While the most frequent cause of ABS is an ischemic stroke affecting both occipital lobes due to a blockage in the posterior cerebral artery, a number of other medical conditions have been associated with its development. These include complications from cardiac surgeries, cerebral angiography, MELAS syndrome (a condition involving mitochondrial dysfunction, encephalopathy, lactic acidosis, and stroke-like episodes), preeclampsia, significant obstetric hemorrhage, head trauma, and adrenoleukodystrophy.

The syndrome has also been linked to conditions such as hypertensive encephalopathy, central nervous system vasculitis, progressive multifocal leukoencephalopathy linked to HIV infection, multiple sclerosis, and posterior reversible encephalopathy syndrome (PRES), which may sometimes emerge after SARS-CoV-2 (COVID-19) pneumonia. Additionally, fat embolism syndrome and Trousseau syndrome have been identified as potential contributors to the condition. These underlying issues predominantly target the occipital lobes, causing visual dysfunction and giving rise to the hallmark symptom of ABS—the striking denial of blindness, which makes this syndrome a unique and intriguing area of neurological study.(1)(5)(10)(11)(13)(14)

EPIDEMIOLOGY :

Anton Syndrome is most frequently caused by a cerebrovascular accident and is predominantly seen in elderly individuals with multiple vascular risk factors. However, while existing literature on the condition is limited, cases have also been documented in younger individuals when the underlying cause is non-vascular. **Anton-Babinski Syndrome remains an exceptionally rare condition, with only 28 reported cases between 1965 and 2016.**(1)(2)

PATHOPHYSIOLOGY :

Progression of ABS :-Despite the anterior visual pathways being unaffected, bilateral damage to the occipital lobes results in total blindness. In some cases, however, small areas of the occipital cortex remain functional, allowing for intermittent visual perception when images are processed in these preserved regions. Occasionally, central vision may remain intact while peripheral vision is lost, a rare occurrence referred to as "gun-barrel vision."Interestingly, certain individuals maintain the ability to perceive motion under specific circumstances. This can happen consciously, as seen in Riddoch syndrome, where patients can detect motion but not static objects, or unconsciously, in a phenomenon called blindsight, where individuals respond to visual stimuli without being consciously aware of them. Conversely, some patients experience motion blindness, a condition where stationary objects are visible, but moving objects cannot be detected. This is believed to arise from damage to the neural pathways linking the lateral geniculate nucleus, the primary visual cortex, and the motion-sensitive middle temporal area (MT or V5).Another intriguing condition associated with vision loss is Charles Bonnet syndrome. In this disorder, individuals who are fully aware of their visual impairment experience vivid, detailed visual hallucinations. These hallucinations may involve unfamiliar objects, scenes, or people, providing a unique yet often unsettling insight into the brain's ability to generate imagery in the absence of normal visual input.(1)

Blindness Pathophysiology :-The recognition of visually presented objects relies on the proper functioning of several key neural structures. The visual pathways serve as the primary conduit for transmitting visual information from the eyes to the brain. The primary visual cortex, located in Brodmann area 17, is essential for processing basic visual stimuli. Adjacent to this region, the secondary visual cortex, encompassing Brodmann areas 18 and 19 in the occipital lobe, plays a crucial role in the further analysis and interpretation of visual input. Additionally, the angular gyrus in the dominant hemisphere, corresponding to Brodmann area 39, functions as a visual association area, integrating visual information with higher cognitive processes. Damage to any of these areas can significantly impair visual recognition, leading to deficits in object identification and overall visual perception.(1)

Anosognosia :-Multiple theories have been proposed to explain the lack of awareness of blindness in individuals with Anton-Babinski Syndrome (ABS). A widely accepted explanation revolves around disconnection syndromes. The Conscious Awareness System (CAS), responsible for processing sensory input, is located in both parietal lobes and works in conjunction with a network in the frontal lobes that governs higher cognitive functions. In cases of ABS, damage to the neural pathways linking the visual cortex and CAS may result in an inability to recognize blindness. Additionally, disruption of communication between the visual centers and language processing areas can lead individuals to fabricate visual descriptions, as they are unable to perceive actual stimuli.

Another theory suggests that excessive activation of the secondary visual pathway, involving the superior colliculus, pulvinar, and temporoparietal regions, plays a key

role. Under normal conditions, this pathway assists in relaying visual information to the association cortex. However, when the primary geniculo-calcarine pathway is compromised, the secondary pathway may take over, creating confabulated visual experiences in individuals who are cortically blind. Neuropsychological factors may also contribute to this phenomenon. A malfunctioning visual monitoring system can lead to misinterpretation of visual information, while false-positive feedback from the visual system can trick individuals into perceiving visuals that do not exist. As a result, their speech centers generate responses based on these incorrect perceptions, further reinforcing the denial of blindness. This complex interplay of neurological and psychological mechanisms makes ABS a particularly intriguing condition within the field of neuroscience.(1)(17)(21)(22)(25)

Confabulations :-Individuals with Anton syndrome often engage in confabulation as a means to compensate for the absence of visual input. Anton theorized that while the speech and language centers of the brain remain fully operational, they become disconnected from the impaired visual pathways. Without receiving accurate sensory information from the eyes, the active speech centers may generate fabricated perceptions, leading to confabulated accounts. As a result, patients steadfastly insist that they can see, despite evidence to the contrary. In some instances, these individuals may even experience detailed and vivid visual hallucinations, further solidifying their false convictions and reinforcing their unwavering belief in their ability to see. This fascinating interaction between disconnected neural systems underscores the complexity of the condition.(1)(4)

CRITERIA FOR DIAGNOSIS OF CORTICAL BLINDNESS :

Cortical blindness, a defining feature of Anton syndrome, is characterized by a complete loss of visual perception, including the inability to distinguish light from darkness. Despite this profound visual impairment, patients often remain unaware of their condition and may confabulate visual experiences due to dysfunction in the visual association areas of the brain. Clinically, the menace reflex, or the instinctive blink response to a perceived threat, is absent, yet the pupillary light reflex and accommodation reflex remain intact. Fundoscopic examination typically reveals no abnormalities, and ocular movements are preserved, further complicating diagnosis. This paradoxical denial of blindness, despite clear evidence of visual impairment, is a hallmark of Anton syndrome and underscores the role of cortical dysfunction in visual awareness.(1)

COMMON CAUSES OF ANTON SYNDROME :

Anton Syndrome is most commonly caused by ischemic cerebrovascular events affecting both posterior cerebral arteries, leading to bilateral occipital cortex damage. This disruption to the brain's visual processing centers results in cortical blindness, a defining feature of the syndrome. While ischemic strokes are the primary cause, other medical conditions have also

been implicated. Cardiovascular instability during invasive medical procedures can lead to hypoxic brain injury, increasing the risk of bilateral occipital damage. Additionally, adrenoleukodystrophy, a rare metabolic disorder affecting the nervous system, has been identified as a potential contributor. In some cases, obstetric hemorrhage has been associated with hypoxic-ischemic injury, which may lead to similar cortical damage.

Notably, all reported cases of Anton Syndrome have involved bilateral lesions of the occipital cortex, reinforcing the critical role of this region in visual perception. Without functional occipital lobes, patients lose the ability to process visual information, yet they paradoxically remain unaware of their blindness due to associated anosognosia.(3)

KEY CHARACTERISTICS OF ANTON SYNDROME :

Anton syndrome is primarily characterized by visual anosognosia, where the patient lacks awareness of their blindness, often accompanied by confabulation, leading to the creation of false visual descriptions to compensate for their loss of sight. Despite being completely blind, individuals with this condition adamantly deny any visual impairment. In some cases, nihilistic delusions, commonly associated with Cotard syndrome, may also be present, causing the patient to believe they do not exist or that parts of their body are missing. These symptoms result from bilateral occipital cortex damage, most frequently due to ischemic strokes or other severe neurological insults, disrupting visual processing and awareness.(1)

CONFABULATIONS IN ANTON SYNDROME :

Confabulations are false or inaccurate statements that individuals present as genuine accounts of their experiences or surroundings. Traditionally, they have been understood as an attempt to compensate for an underlying memory impairment. However, their exact prevalence remains uncertain, and their causes are still debated. Possible explanations include amnesia, embarrassment, frontal lobe dysfunction, personality traits, dream-like states, or disruptions in self-perception. Historically, the concept of confabulation emerged in the early 20th century as part of a broader psychological framework that included terms like delusions and fixed ideas, all aimed at describing narratives with questionable accuracy. This paper explores the historical development of the term and concept of confabulation, along with early observations of the behaviors it encompasses, and proposes a model based on these historical insights. Confabulations generally fall into two categories: false statements produced by individuals with memory impairments and elaborate, fantastical claims made with conviction by individuals with psychosis who do not have memory deficits. The notion that confabulations serve as a way to "fill in gaps" or "cover up" missing information remains widely accepted.(4)

DIFFERENTIAL DIAGNOSIS :

Anton-Babinski Syndrome (ABS) must be distinguished from several other neurological and visual conditions, including:

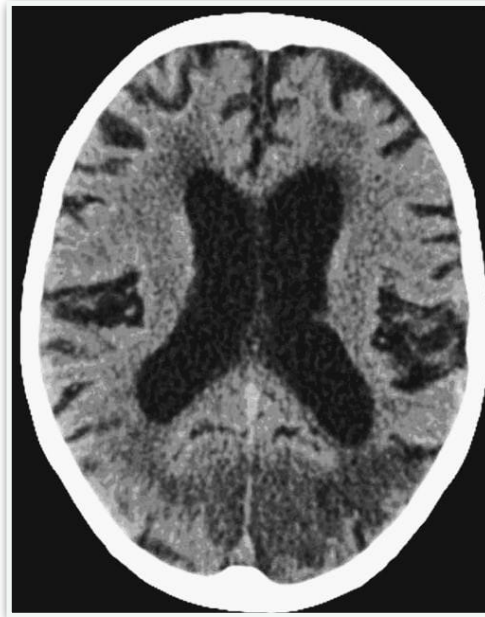
- **Cerebral visual impairment** – A condition where visual dysfunction results from brain damage rather than abnormalities in the eyes.
- **Delayed visual development** – A slower-than-expected maturation of visual abilities, often observed in infants.
- **Homonymous hemianopia** – The loss of vision in the same visual field of both eyes due to damage along the visual pathways.
- **Lack of facial recognition (prosopagnosia)** – The inability to recognize faces despite having normal vision.
- **Visual agnosia** – The failure to identify objects despite intact eyesight.
- **Visual neglect** – A deficit in attention to one side of the visual field, typically caused by damage to the parietal lobe.
- **Visual perceptual disorders** – Impairments in interpreting and processing visual information, despite having normal eyesight.(1)

EXPLORING THE DIVERSE CAUSES OF ANTON SYNDROME : CASE-BASED INSIGHTS :-

ANTON SYNDROME RESULTING FROM CEREBROVASCULAR DISEASE :

Cortical blindness and Anton-Babinski Syndrome (ABS) should be considered in patients who exhibit unusual patterns of vision loss along with clinical signs indicative of occipital lobe damage. Although ischemic strokes affecting both occipital lobes remain the most common cause, ABS may also develop due to various other conditions, including metabolic abnormalities, infections, autoimmune disorders, or traumatic injuries to the brain. The likelihood of visual recovery depends heavily on the underlying cause. Recovery is often limited in cases involving occipital lobe infarctions, which significantly reduce the chances of full vision restoration. Treatment should focus on secondary prevention measures to minimize the risk of additional vascular incidents. Simultaneously, rehabilitation programs play a critical role in helping affected individuals adapt to their visual impairment, enhancing their quality of life through tailored support and strategies for managing day-to-day challenges. Early diagnosis and a multidisciplinary approach are essential to improving outcomes for patients with ABS.(5)(7)(19)

Figure 1. A computed tomography scan of the patient's brain at initial presentation, demonstrating acute infarction in the right occipital and left occipito-parietal lobes.



General Case Discussion :-

A patient with a history of mild cognitive impairment and other comorbidities was found collapsed at home. Upon examination, there was initial suspicion of a transient neurological event, but by the time of hospital admission, motor function had normalized. The most notable clinical finding was severe visual impairment, despite the patient insisting they could still see. Pupillary reflexes were intact, and fundoscopy was unremarkable, suggesting an intact anterior visual pathway. A brain imaging study revealed acute infarctions in the occipital lobes, leading to a diagnosis of cortical blindness. The patient also exhibited anosognosia, denying visual loss and displaying signs of confabulation.

The treatment approach followed in this case focused on acute stabilization, rehabilitation, and long-term support to help the patient adapt to visual impairment. Initially, acute management aimed at stabilizing the patient's condition and preventing further neurological deterioration. This included supportive care, secondary stroke prevention with antiplatelet or anticoagulant therapy if indicated, and careful management of cardiovascular risk factors such as blood pressure and blood glucose levels. Following stabilization, rehabilitation efforts were implemented to improve functional independence.

Occupational and physical therapy played a crucial role in assisting the patient with mobility and daily activities, while vision rehabilitation helped in adapting to the loss of sight. Psychological support was also essential, particularly in addressing anosognosia and the associated confabulation, ensuring the patient received appropriate guidance in recognizing

and compensating for their visual deficit. Long-term management emphasized continued support and adaptation strategies.

Home modifications were introduced to enhance safety, and caregivers were involved in assisting with activities of daily living such as dressing, eating, and mobility. Regular follow-ups were scheduled to assess neurological recovery and functional progress. Although some improvement in light perception was observed over time, significant recovery of visual acuity remained unlikely, making ongoing support and rehabilitation essential for maintaining quality of life.(5)(24)

ACUTE CORTICAL BLINDNESS ASSOCIATED WITH POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME (PRES) :

A middle-aged man with a history of acute hypertension presented with seizures and cortical blindness during the recovery phase following surgery. Neuroimaging revealed notable abnormalities; CT scans demonstrated hypodensities in the occipital lobes, while MRI scans showed symmetrical hyperintense signals involving both the gray and white matter within these regions.

Further thromboembolic assessments, such as vertebral artery Doppler studies, yielded normal results, and echocardiography indicated only borderline left ventricular hypertrophy. Based on these clinical and imaging findings, the patient was diagnosed with posterior reversible encephalopathy syndrome (PRES).

PRES is characterized by posterior subcortical edema, which is readily identifiable on MRI. Clinically, the syndrome manifests with a range of symptoms, including visual disturbances, severe headaches, seizures, and altered levels of consciousness. In this particular case, treatment primarily targeted effective blood pressure management, ultimately leading to the complete resolution of the patient's blindness. This outcome aligns with the vasogenic theory underlying PRES, which proposes that sustained fluctuations in elevated blood pressure compromise the brain's vascular autoregulatory mechanisms. This failure in compensatory vasoconstriction results in hyperperfusion, causing fluid leakage into the occipital lobes and triggering temporary cortical blindness.(6)(14)

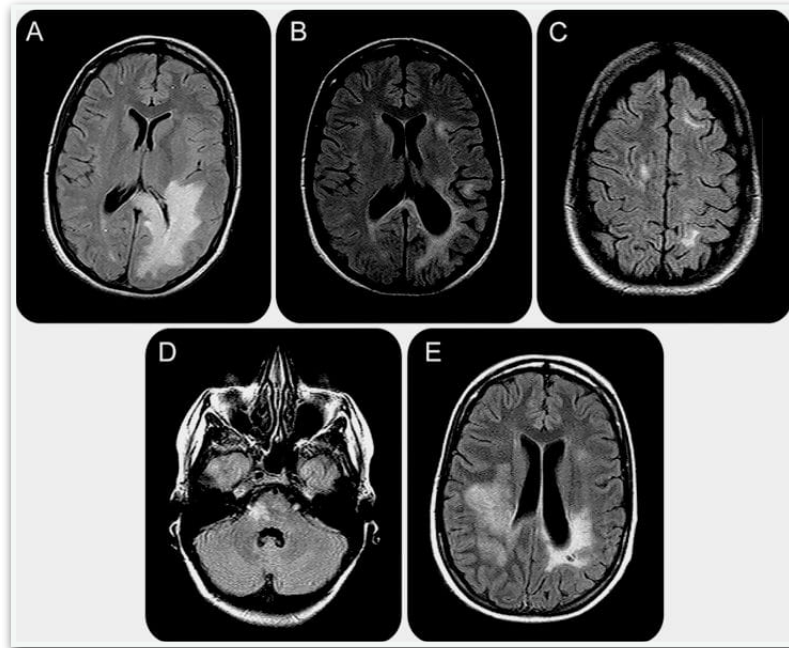
ANTON SYNDROME DUE TO MULTIPLE SCLEROSIS :

A young adult initially presented with right-sided hemiparesis and homonymous hemianopia. Neuroimaging revealed a subcortical lesion in the left temporo-parieto-occipital white matter, characterized by high T2/FLAIR signal intensity, restricted diffusion, and a mild mass effect, though no contrast enhancement was observed. Comprehensive serologic and cerebrospinal fluid (CSF) analysis yielded largely normal results, except for elevated CSF immunoglobulin G levels and the presence of oligoclonal bands. Suspecting a demyelinating disorder, the patient was treated with intravenous methylprednisolone, which led to partial, though incomplete, recovery. In the year following the initial episode, follow-up MRI scans showed

the emergence of small, asymptomatic juxtacortical and periventricular lesions. Meanwhile, the initial tumefactive lesion on the left side remained stable, displaying adjacent gliosis and ex vacuo dilation of the lateral ventricle. Sixteen months after the initial presentation, the patient experienced a severe relapse, presenting with global aphasia, left-sided sensorimotor deficits, and cortical blindness.

The T2/FLAIR-weighted MRI brain sequences revealed the following findings:

Figure 2 . MRI brain sequences



Initial episode (A): A prominent hyperintense lesion was detected in the subcortical and periventricular white matter of the left frontal, parietal, and occipital lobes. This was accompanied by a mild mass effect on the posterior horn of the left lateral ventricle.

Between episodes (B-D): Multiple asymptomatic hyperintense lesions were observed bilaterally, affecting the periventricular, juxtacortical, and infratentorial regions.

Second episode (E): A new hyperintense lesion emerged in the subcortical and periventricular white matter of the right fronto-parieto-occipital region. Additionally, an area of encephalomalacia with surrounding gliosis in the left posterior region persisted, leading to ex vacuo dilatation of the left posterior lateral ventricle.

Neuroimaging revealed a new lesion in the right parieto-occipital white matter, which demonstrated radiologic features similar to the original lesion. With the absence of spinal lesions and the detection of multiple T2 lesions consistent with dissemination in time and space, the patient was diagnosed with multiple sclerosis (MS) based on the revised McDonald criteria. Given the aggressive course of tumefactive relapsing-remitting MS, the treatment regimen included high-dose methylprednisolone, plasmapheresis, and initiation of alemtuzumab. Although most neurological deficits improved gradually, cortical blindness

persisted. Despite an inability to perceive light or respond to visual stimuli, the patient remained unaware of the visual impairment and confabulated descriptions when questioned. Over the span of two years, vision recovery occurred in stages. The patient initially developed an awareness of the blindness, followed by regaining light perception and later recognizing objects, although achromatopsia remained. Eventually, color perception improved to a functional level, allowing the patient to navigate surroundings, describe images in magazines, and read with moderate difficulty. Visual recovery ultimately plateaued, reflecting significant progress despite the severity of the condition.(8)

HEAD TRAUMA WITH BIFRONTAL CONTUSIONS LEADING TO ANTON SYNDROME :

Anton's syndrome can arise following head trauma that results in optic nerve damage and bifrontal contusions. In such cases, prominent neurobehavioral features include denial of blindness, generalized anosognosia, and confabulation. While the syndrome is most frequently associated with bilateral occipital cortex damage, it may also stem from peripheral visual impairment, even in the absence of delirium or significant cognitive decline. The role of bifrontal dysfunction in Anton's syndrome is particularly noteworthy, as it appears to significantly contribute to the development of anosognosia and the fabrication of false perceptions.

This observation highlights the importance of frontal lobe involvement as a key factor underlying these symptoms. Understanding this connection provides valuable insights into the neuroanatomical basis of anosognosia and confabulation, offering a deeper understanding of the mechanisms driving this rare and complex condition.(9)

ANTON SYNDROME PRESENTING AS A FEATURE OF TROUSSEAU SYNDROME INVOLVING BILATERAL OPTIC RADIATIONS :

Anton's syndrome is an uncommon neuropsychiatric condition marked by cortical blindness, anosognosia, and visual confabulation, occurring in the absence of significant cognitive decline. A rare case of Anton's syndrome was identified as a manifestation of Trousseau syndrome affecting the bilateral optic radiations.

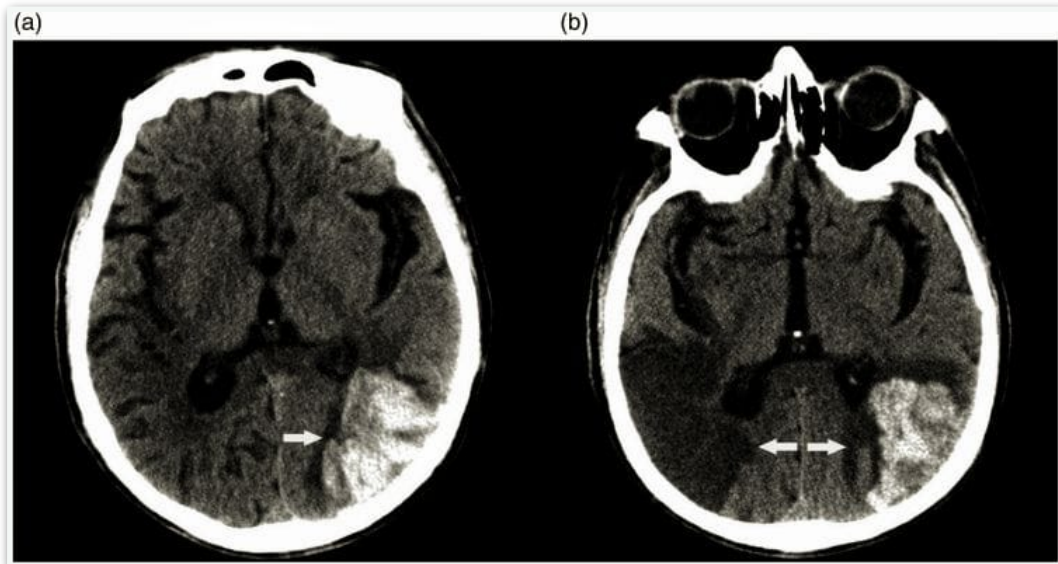
The patient, previously diagnosed with gallbladder cancer two months earlier, presented to the hospital with confusion and quadriplegia. Despite being completely blind, the patient adamantly denied their visual impairment and exhibited vivid visual confabulation, consistent with the hallmark features of Anton's syndrome.

Neuroimaging studies, including CT and MRI scans, showed infarcts in the bilateral temporo-parieto-occipital junctions with evidence of hemorrhagic transformation, primarily involving the optic radiations. Additional clinical findings included gallbladder cancer with peripheral metastases, significantly elevated tumor markers, and high D-dimer levels, all of which suggested a cancer-associated hypercoagulable state. This constellation of findings confirmed the diagnosis of Trousseau syndrome, shedding light on the underlying cause of Anton's syndrome in this patient.(10)

Figure 3 . Brain computed tomography (CT) images

a) Initial CT scan (on admission): A hemorrhage was detected at the left temporo-occipital junction (marked by the arrow).

b) Follow-up CT scan (next day): Infarcts were observed at the bilateral temporo-parieto-occipital junctions (indicated by arrows), with evidence of hemorrhagic transformation.



FAT EMBOLISM SYNDROME :

Fat embolism syndrome (FES) is a serious and potentially life-threatening complication resulting from fat embolization. It is classically associated with Bergman's triad, which includes respiratory distress, a petechial rash, and altered mental status. While fat emboli frequently enter the systemic circulation, FES develops in only a small percentage of cases—approximately 0.05%–3%—involving isolated long bone fractures. Visual symptoms in FES are most commonly linked to fat embolism retinopathy and generally emerge later in the disease. However, in rare instances, cortical blindness may present as an early symptom. Remarkably, no previous cases have documented cortical blindness as a consequence of an isolated tibial fracture.

One notable case involved a 20-year-old male who sustained an isolated fracture of the right tibial shaft. Within 24 hours, the patient experienced sudden bilateral vision loss, despite showing no other initial symptoms. The absence of fundoscopic abnormalities and other overt clinical signs initially complicated the diagnostic process. As the disease progressed, the patient began exhibiting confusion, hypoxia, and generalized tonic-clonic seizures. MRI scans ultimately revealed multiple cerebral fat emboli, notably affecting the bilateral occipital lobes, leading to a conclusive diagnosis of FES. Following supportive treatment, the patient gradually recovered both vision and neurological function.

The tibial fracture was stabilized using plate fixation under spinal anesthesia, and the perioperative period was uneventful. The patient was discharged after staple removal, and at a one-month follow-up, no residual visual field deficits or neurological impairments were observed. Although FES is a rare complication of isolated tibial fractures, this case underscores the critical importance of early recognition. Prompt diagnosis and timely intervention are crucial for significantly improving patient outcomes in such cases.(11)

ANTON SYNDROME RESULTING FROM BILATERAL OCCIPITAL LOBE DAMAGE :

Anton syndrome, characterized by visual anosognosia and confabulation, arises from bilateral occipital lobe damage, traumatic brain injury, leading to cortical blindness. Patients with this condition deny their visual impairment and fabricate descriptions to compensate for the lack of sensory input. In this case, the patient exhibited signs of Anton syndrome following bilateral occipital ischemia due to severe stenosis of the head and neck arteries.

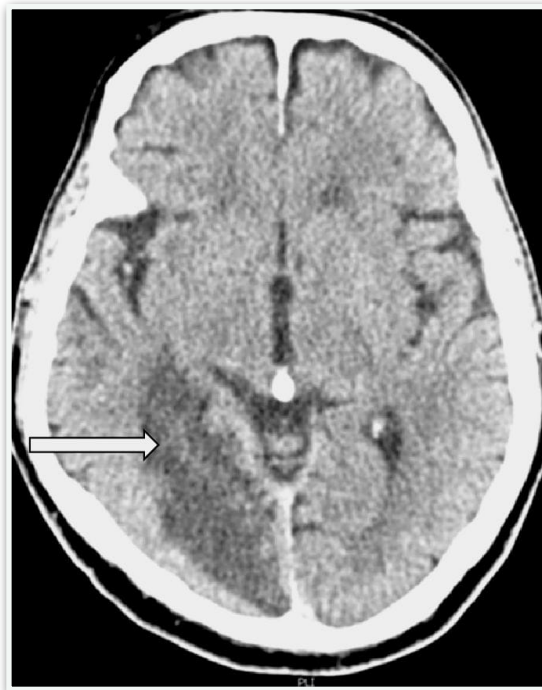


Figure 4 . An axial brain CT scan reveals a characteristic nonhemorrhagic infarct located within the region supplied by the right posterior cerebral artery (marked by the white arrow).

Additionally, episodes of agitation and self-directed speech suggested the possibility of Charles Bonnet syndrome, which manifests as visual hallucinations in individuals with significant vision loss. Ischemic strokes remain the most frequent cause of Anton syndrome, though its occurrence is relatively rare.(12)(20)(22)(23)

ANTON SYNDROME AS A RESULT OF DECOMPRESSION ILLNESS :

Anton's syndrome, also referred to as Anton-Babinski syndrome, is a rare condition in which individuals with cortical blindness fail to recognize their vision loss. This case involves a patient who developed Anton's syndrome following decompression illness (DCI) after recreational scuba diving. The patient exhibited progressive neurological symptoms, which were treated with multiple recompression sessions. Within 48 hours, the visual anosognosia resolved. Further evaluation through echocardiography later confirmed the presence of a patent foramen ovale.(15)

GENERALIZED TREATMENT APPROACHES :

Anosognosia is a neurological condition where individuals lack awareness of their own neurological or psychiatric impairments. Anton Syndrome (AS), a rare variant, involves bilateral vision loss accompanied by a denial of blindness. Management of AS varies depending on the underlying cause and the severity of symptoms.

A systematic review of 64 studies, analyzing 72 cases of AS, highlights different treatment approaches. Supportive care was implemented in 30.6% of cases, focusing on symptom management and rehabilitation. Another 30.6% received treatment aimed at addressing the root cause, such as stroke or other neurological conditions. Among patients, 45.8% showed improvement, while 22.2% remained stable, and 11.1% experienced further deterioration. The review also indicates that anticoagulation therapy, commonly used for stroke management, was linked to an increased mortality rate ($p < 0.05$), suggesting the need for a cautious and individualized approach. Since AS often results from occipital lobe damage, accurate diagnosis through clinical evaluation and neuroimaging is essential to distinguish it from other conditions with similar presentations. Tailoring treatment to the specific etiology is crucial for better outcomes. While some cases respond well to supportive and rehabilitative measures, others require targeted interventions to manage the underlying cause. Further research is necessary to refine treatment strategies and improve the overall care of individuals affected by AS.(16)

PROGNOSIS :

Vision recovery has been observed in cases of Anton-Babinski Syndrome (ABS) when the condition is associated with treatable causes such as hypertensive encephalopathy and cortical hypoperfusion. In such instances, early identification, combined with timely and appropriate treatment of the underlying cause, can lead to significant improvement and, in some cases, even complete restoration of vision. However, the overall prognosis for cortical blindness varies and is influenced by multiple factors, including the patient's age, the nature and severity of the underlying pathology, the duration of blindness before intervention, and the individual's overall medical history and neurological resilience. Timely intervention plays a crucial role in improving patient outcomes. Additionally, interdisciplinary collaboration

among neurologists, ophthalmologists, and rehabilitation specialists is vital in optimizing treatment approaches, ensuring that both the visual and cognitive aspects of Anton syndrome are adequately addressed. These insights underscore the necessity of increasing awareness about Anton syndrome within the medical community and beyond. Furthermore, raising awareness among healthcare providers and the general public may facilitate earlier recognition and intervention, ultimately improving prognosis and quality of life for affected individuals.(1)(2)

ENHANCING PATIENT CARE :

Recovery of visual function has been observed in cases of Anton-Babinski Syndrome (ABS) linked to treatable conditions such as hypertensive encephalopathy and cortical hypoperfusion. Timely recognition and appropriate management of the underlying cause are critical in these cases, often resulting in substantial improvement or even complete resolution of symptoms. The prognosis for cortical blindness is influenced by several factors, including the patient's age, the specific underlying cause, the duration and severity of the condition, the pace of initial recovery, and the individual's overall medical history.

Early diagnosis and prompt intervention, combined with targeted therapeutic strategies, play a pivotal role in maximizing outcomes. These measures not only enhance the likelihood of restoring visual function but also improve overall neurological recovery, emphasizing the importance of a proactive and multidisciplinary approach to treatment. This highlights the potential for positive outcomes, even in cases where symptoms may initially appear severe or persistent.(1)

Public Perception and Awareness of Anton Syndrome: A Survey-Based Justification

RATIONALE FOR THE SURVEY :

A preliminary survey indicated that while some respondents were familiar with Anton syndrome, many demonstrated a limited or incomplete understanding of its neurological foundation. A significant portion of respondents associated the condition primarily with blindness, often failing to recognize anosognosia as a defining characteristic. Furthermore, uncertainty regarding the etiology, clinical manifestations, and broader implications of the syndrome was prevalent, underscoring notable gaps in both public awareness and professional medical discourse.

These misconceptions highlight the necessity of a thorough and evidence-based review that elucidates the neurological mechanisms underlying Anton syndrome, its complex symptomatology, and its profound impact on affected individuals. By systematically addressing these deficiencies in knowledge, this review aims to enhance awareness, promote a more precise understanding of the condition, and contribute to more informed discussions within both medical and non-medical communities.

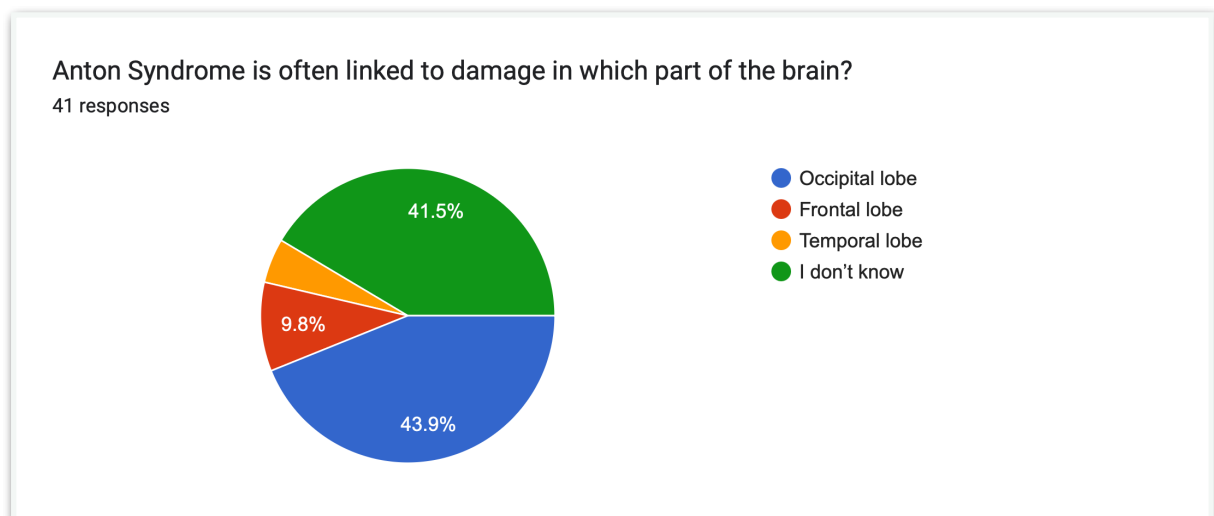
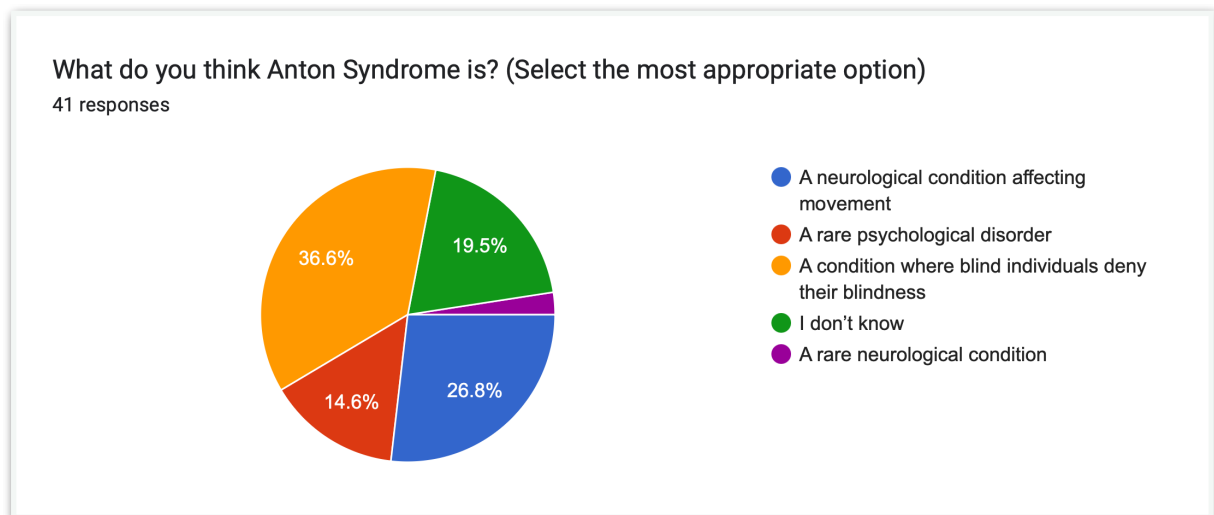
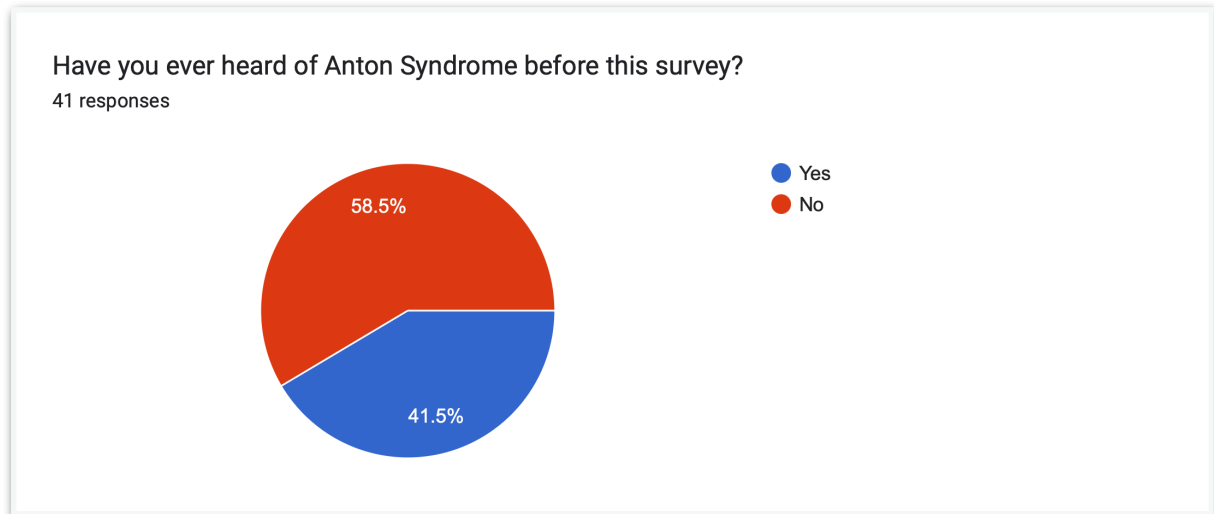
SURVEY METHODOLOGY :

A Google Forms survey was utilized to collect responses regarding Anton syndrome, aiming to gauge public awareness, understanding, and perceptions of the condition. The survey featured a combination of multiple-choice and open-ended questions designed to assess the extent of respondents' knowledge, their familiarity with its neurological basis, and their opinions on the need for further awareness. In addition to evaluating general awareness, the survey also gathered information about participants' professional backgrounds to determine whether knowledge of Anton syndrome varied across different fields of study and professions. Respondents came from diverse academic and professional disciplines, including pharmacy, MBBS, physiotherapy, law, computer science, business administration, Doctor of Pharmacy, electronic engineering, IT, and other related fields.

The majority of responses were contributed by students, particularly those pursuing pharmacy and medical studies, suggesting a greater interest or exposure to neurological and medical conditions within these fields. Additionally, individual responses were received from professionals across various sectors, including engineering, physiotherapy, and business administration. This diverse participant base provided valuable insights into the differences in awareness levels across medical and non-medical domains, highlighting the need for broader dissemination of information on Anton syndrome.

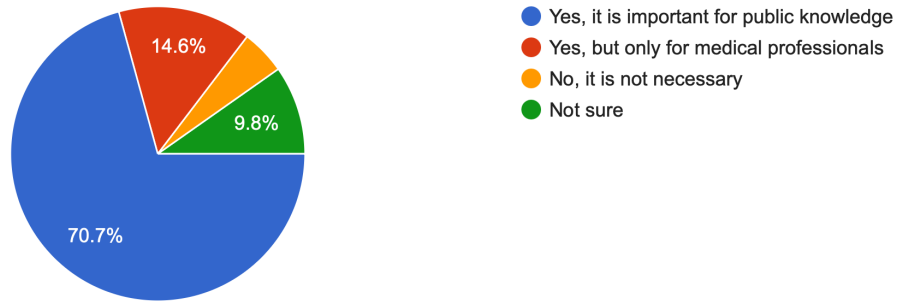
SURVEY ANALYSIS :

The following section presents an analysis of the survey responses, including key insights derived from participant feedback.



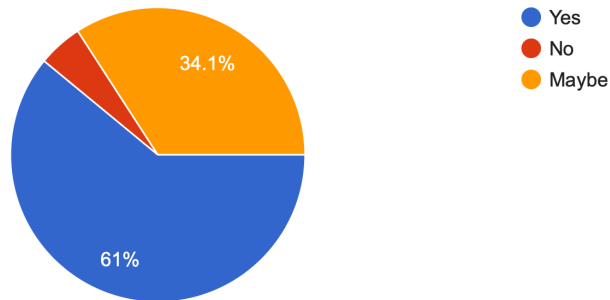
Do you think awareness about rare neurological conditions like Anton Syndrome should be increased?

41 responses



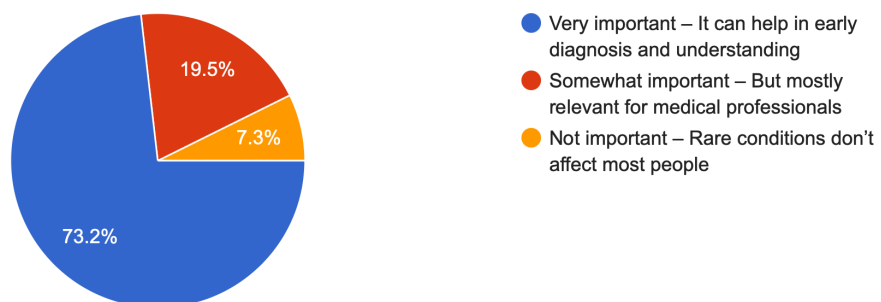
Would you be interested in learning more about Anton Syndrome through seminars, articles, or videos?

41 responses



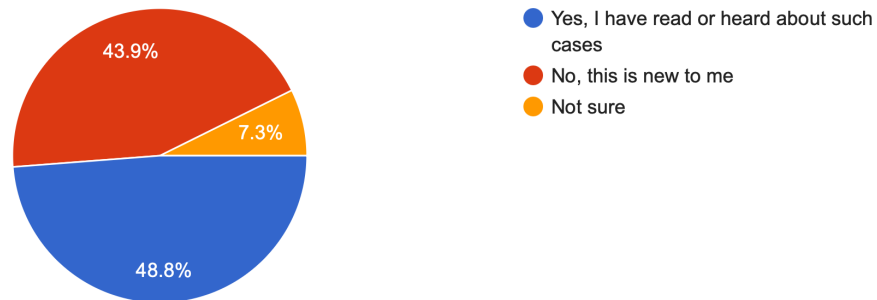
How important do you think awareness of rare neurological conditions is in everyday life?

41 responses



Have you ever come across a medical condition where a person is unaware of their own disability?

41 responses



If someone insists they can see despite medical evidence of blindness, how do you think they would explain their experiences?

41 responses



- Nearly 58% of respondents had heard about Anton syndrome; however, their understanding varied significantly.
- When asked about the specifics of the condition, responses were inconsistent, reflecting gaps in knowledge.
- Only 43% of participants correctly identified occipital lobe damage as being associated with Anton syndrome.
- Approximately 70% of respondents agreed that raising awareness about the syndrome is necessary.
- Around 61% expressed interest in learning more about the condition.
- A significant 73.2% believed that early diagnosis is crucial for better management of Anton syndrome.

- Nearly 48% of respondents had never discussed the condition or similar situations before.
- About 51% suggested that patients may experience confusion due to past memories, which could contribute to the denial of their blindness.

Overall, the responses indicate that while some level of awareness exists, misconceptions and knowledge gaps remain prevalent. The significant agreement on the need for awareness and early diagnosis further reinforces the importance of continued education and discussion about Anton syndrome.

CONCLUSION :

The survey responses provided supplementary insights to the existing review article, emphasizing the need for increased awareness and education on Anton syndrome. The data revealed a general lack of in-depth understanding, even among those aware of the condition, highlighting the necessity for further informational efforts. The findings suggest that targeted educational initiatives could bridge the knowledge gap and foster a more accurate perception of Anton syndrome within both medical and non-medical communities. While not a large-scale study, this survey underscores the importance of ongoing research and awareness campaigns to improve recognition and management of the condition.

DISCUSSION :

Anton syndrome, involving cortical blindness and anosognosia, remains underrecognized even within medical and allied health fields. Though documented in literature, it lacks widespread understanding, making early diagnosis and management difficult. The condition is most often linked to occipital lobe damage, yet this connection is often overlooked.

A survey conducted as part of this study found that 58% of respondents had heard of Anton syndrome, but many associated it only with blindness, neglecting the crucial element of anosognosia. Only 43% correctly identified occipital lobe damage as its neurological basis. These results reflect a larger trend of simplifying neurological disorders to their visible symptoms, ignoring their cognitive and psychological dimensions.

There was strong agreement on the need for increased awareness—70% emphasized its importance, and 61% expressed interest in learning more. Furthermore, 73.2% recognized the value of early diagnosis. Notably, 48% had never discussed the condition before, and 51% suggested that patients' past visual memories may contribute to denial of blindness—a perspective supported by current literature.

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