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Does Ankylosing Spondylitis Affect the Volume of the Pineal Gland?

¿Afecta la Espondilitis Anquilosante el Volumen de la Glándula Pineal?

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URHAN, A. T.; DOGAN, B.; IRMAK SAPMAZ, H.; ASLAN BARAN, S.; KOKCU, F. & OZTURK, M. F. Does ankylosing spondylitis affect the volume of the pineal gland? *Int. J. Morphol., 44(3)*:1008-1013, 2026.

SUMMARY: It is thought that Ankylosing Spondylitis (AS), which primarily affects the axial skeleton, may cause structural and functional effects in the brain through chronic inflammation and neuroendocrine mechanisms. The aim of this study is to investigate the potential effects of AS on the pineal gland (gl) cerebrum, and intracranial volume using magnetic resonance imaging (MRI). In this study, images from 25 patients with AS and 25 healthy individuals of similar age and sex were retrospectively evaluated. The pineal gland volume was examined using manual segmentation with the 3D Slicer software (version 4.10.2) on high-resolution T1-weighted MRI, while cerebral and intracranial volumes were examined using the VolBrain automatic segmentation method. The T-test was used for statistical analysis. No statistically significant differences were found between the AS group and the control group in terms of pineal gland, cerebrum, and intracranial volume ($p > 0.05$). Similarly, no significant differences were found in the pineal gland/cerebrum and pineal gland/intracranial volume ratios ($p > 0.05$). Although neuroinflammatory and neuroendocrine mechanisms are thought to play a role in the pathophysiology of AS, our findings indicate that these processes do not lead to significant structural differences in brain volume. However, advanced studies with larger sample groups that also analyze melatonin levels biochemically will contribute to a more comprehensive understanding of this relationship.

KEY WORDS: Ankylosing spondylitis; Pineal gland; Cerebrum; VolBrain.

INTRODUCTION

The pathogenesis of ankylosing spondylitis (AS) involves a complex interaction of genetic, immunological, and environmental factors (Braun & Sieper, 2007). The process, which begins with postural changes and back pain, eventually leads to significant limitations in spinal movement and functional losses in daily living activities (Rudwaleit *et al*., 2009; Li *et al*., 2020). The prevalence of AS ranges from 0.1 % to 1 % in the general population and is particularly common in young adult males (Dean *et al*., 2014).

Melatonin, primarily secreted by the pineal gland, is a neuroendocrine mediator with important roles such as reducing oxidative stress and supporting neuroprotective mechanisms (Hardeland, 2019). It has been reported that changes in the volume of the pineal gland may affect melatonin production and secretion rhythm, and therefore may be associated with neurodegenerative and inflammatory diseases (Matsuoka *et al*., 2018). In recent years, the effects of changes in melatonin levels on inflammatory response and bone metabolism have become a notable area of research in AS pathophysiology. It has been reported that serum melatonin levels in AS patients are significantly higher than in healthy individuals; furthermore, a positive correlation has been found between melatonin levels and the modified Stoke AS Spine Score (Li *et al*., 2020; Cho *et al*., 2021). These findings suggest that melatonin may play a potential regulatory role in the pathological osteogenesis and ossification processes observed in AS (Li *et al*., 2020).

No original study has been found in the literature that quantitatively examines the volume of the pineal gland in AS patients. This study aims to determine the possible structural changes in the brain that may cause the clinical symptoms observed in individuals with ankylosing spondylitis. Our study aims to provide a comprehensive view of the systemic nature of the disease by revealing the possible effects of AS on the neuroendocrine system, beyond being an inflammatory disease that only affects the axial skeleton.

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MATERIAL AND METHOD

Study Design and Ethical Approval

This study was designed as a retrospective, cross-sectional, observational study. All procedures were conducted in accordance with the ethical principles of the Declaration of Helsinki. Approval for the research was obtained from the Clinical Research Ethics Committee of Tokat Gaziosmanpasa University (Ethics Committee Approval Number: 15235480-050.04-546940). Brain imaging data were obtained retrospectively from the university hospital's PACS (Picture Archiving and Communication System). All MRI data were anonymized prior to analysis, and participants' personal information was protected in accordance with privacy principles.

Participants and Sample Size

The study included images from AS patients and healthy individuals with similar characteristics in terms of age and sex. The required sample size was determined based on the study by Wu *et al*. (2013) using G*Power 3.1.9.4 software with parameters α=0.05, β=0.10, and effect size=0.95, and it was calculated that at least 13 individuals would be sufficient for each group. To increase statistical power, data from a total of 135 individuals diagnosed with AS in the archive records of Tokat Gaziosmanpasa University Faculty of Medicine Hospital between 2019 and 2024 were screened. Images from 25 adult patients with appropriate brain MRI scans and 25 healthy adults who presented to our hospital with symptoms such as headache or dizziness, underwent brain MRI, and had no diagnosis were included in the study. Cases were excluded from the study if there was a history of cranial or spinal surgery, head trauma, persistent headache, neurological deficit, serious systemic disease, vertebral deformity, diagnosed endocrine disease, or related genetic syndrome. Cases were excluded from the study if there was significant calcification in the pineal gland, if measurements could not be made due to imaging artifacts, or if there was missing demographic or clinical data.

Imaging Protocol and Volume Measurement

Brain imaging data for all participants were obtained at Tokat Gaziosmanpasa University Faculty of Medicine Hospital using a Philips Medical Systems 3.0 T Gyroscan NT device. T1-weighted, three-dimensional, high-resolution sagittal plane images were acquired with a 1 mm^3 isovoxel size, TR=8.1 ms, TE=3.7 ms, TFE=230 ms, flip angle=8°, 224×224 pixel matrix size, and 224×224 mm FOV. All images were recorded in DICOM format and anonymized prior to evaluation.

The pineal gland volume was calculated using manual segmentation with the open-source 3D Slicer software (version 4.10.2). The boundaries of the gland in each plane were marked using the "Paint" tool in the "Segment Editor" module. Then, the total volume was automatically obtained in millimeters cubed (mm^3) using the "Segment Statistics" and "Quantification" tabs. All segmentation procedures were performed in a double-blind manner by two independent observers experienced in the field of anatomy.

The VolBrain automatic segmentation method was used for cerebrum and intracranial volume measurements. Intra-observer and inter-observer reliability were assessed using the intraclass correlation coefficient (ICC). According to the literature, ICC < 0.50 is considered low, 0.50–0.75 is moderate, 0.75–0.90 is good, and > 0.90 is excellent reliability. In this study, intra-observer reliability was found to be excellent (ICC: 0.902–0.991), while inter-observer reliability was found to be good (ICC: 0.780–0.956).

Statistical analysis

Data were analyzed using IBM SPSS Statistics for Windows, Version 26.0 (IBM Corp., Armonk, NY, USA) software. The distribution characteristics of the data were assessed using the Shapiro–Wilk test. For variables showing a normal distribution, the Independent Samples T-Test was applied to compare the differences between two independent groups. Results were presented as mean ± standard deviation, and the level of statistical significance was set at $p < 0.05$.

RESULTS

The mean age of the evaluated individuals was calculated as 45.92 ± 10.54 years. The pineal gland volume was found to be lower in AS (105.94 ± 17.74 mm^3) than in the control group (111.99 ± 26.30 mm^3) ($p = 0.345$). There was no significant difference between the healthy control group and the group of patients diagnosed with ankylosing spondylitis in terms of cerebrum and intracranial volumes and the ratios of the pineal gland to cerebrum and intracranial volumes (Table I; Figs. 1 and 2).

Table I. Comparison of pineal gland and cerebrum volumes in ankylosing spondylitis and healthy control groups.

Parameters	AS (n=25) Mean ± SD	HC (n=25) Mean ± SD	p-value
Gl. pinealis volume (mm^3)	105.94 ± 17.74	111.99 ± 26.30	0.345
Cerebrum volume (mm^3)	959.32 ± 136.80	997.60 ± 117.92	0.295
Intracranial volume (mm^3)	1285.60 ± 178.05	1315.96 ± 135.20	0.501
Gl. pinealis/cerebrum ratio	0.1140 ± 0.0308	0.1132 ± 0.0286	0.928
Gl. pinealis/intracranial ratio	0.0848 ± 0.0229	0.0855 ± 0.0209	0.913

AS: Ankylosing spondylitis, HC: Healthy control group, Data are presented as mean ± standard deviation (Mean ± SD). Differences between groups were evaluated using an independent two-sample t-test. The significance level was set at $p < 0.05$. Gl.: Glandula

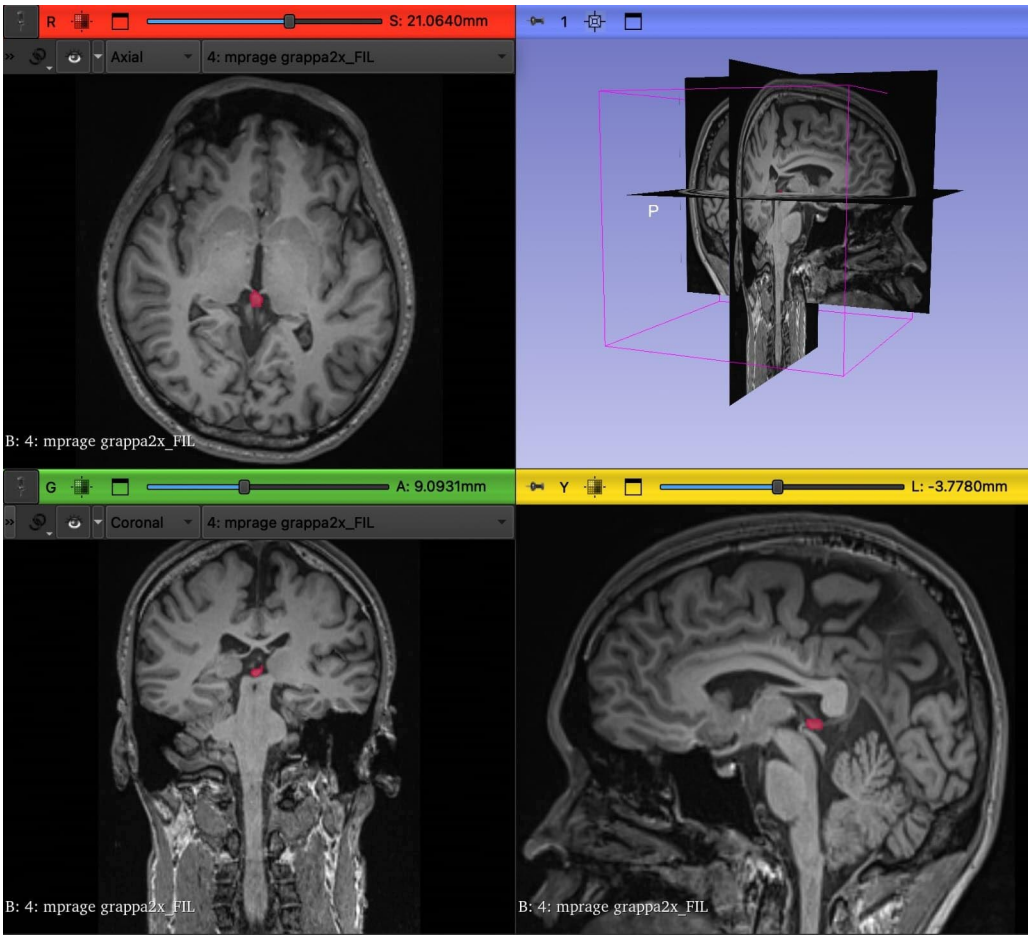

Fig. 1. Manual segmentation of the pineal gland using 3D Slicer software in a 45-year-old AS patient (3D Slicer software (version 4.10.2)).

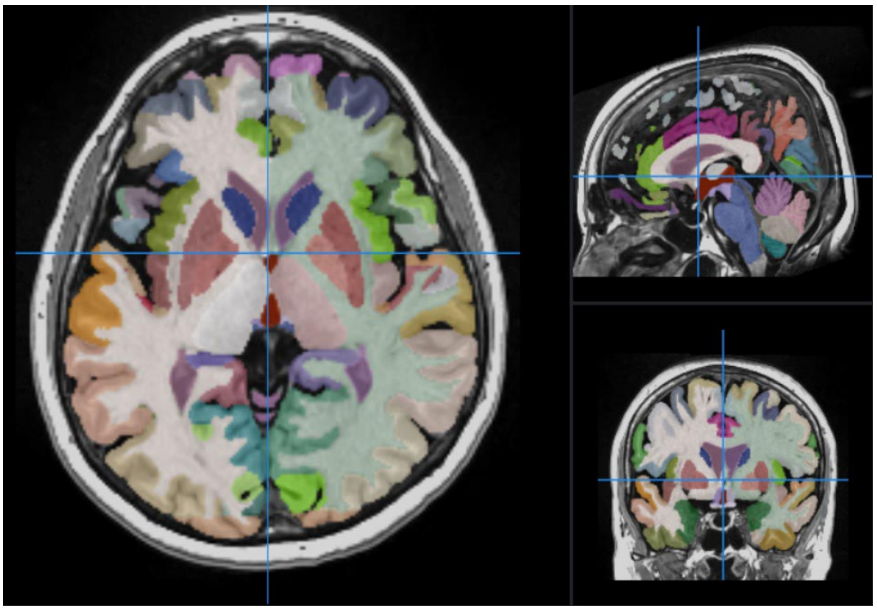

Fig. 2. Cerebrum image obtained using VolBrain automatic segmentation in a 45-year-old AS patient (https://www.volbrain.net/jobs/1916575/preview).

DISCUSSION

This study was conducted to examine the effect of AS on pineal gland volume. In this context, pineal gland volume, cerebrum volume, intracranial volume, pineal gland/cerebrum volume ratio, and pineal gland/intracranial volume ratio were examined in the AS and control groups.

When reviewing the literature, we found various neuroimaging studies examining the potential effects of AS on different brain structures; however, we did not encounter any studies investigating the pineal gland, cerebrum, and intracranial volumes in individuals with AS. Li *et al*.

(2017) detected a decrease in gray matter in regions such as the anterior cingulate cortex, insula, hippocampus, and thalamus in AS patients in relation to pain and fatigue severity, and found a positive correlation between pain scores and insula and thalamus activity. In addition, they reported that fatigue levels were associated with weakened prefrontal–limbic connections (Li *et al*., 2017). These findings indicate that neuroplastic adaptations develop at the central nervous system level beyond peripheral inflammation in AS. Liu *et al*. (2020) found decreased spontaneous brain activity in regions such as the anterior cingulate cortex, insula, prefrontal cortex, and thalamus in AS patients at rest; conversely, they observed increased compensatory activity in somatosensory areas. The study reported that connectivity impairments between the limbic system and the default mode network correlated with pain intensity and disease activity (Liu *et al*., 2020). Hua *et al*. (2020) reported that the volume of the left putamen was significantly increased in AS patients and that this increase showed a positive correlation with pain intensity. The putamen is one of the basal ganglia structures involved in the motor and affective components of pain perception. This finding supports the notion that chronic pain in AS arises not only from peripheral inflammation but also from neuroplastic reorganizations occurring at the central nervous system level (Hua *et al*., 2020). Similarly, Li and Liu's fMRI studies also reported structural and functional changes in pain processing centers such as the anterior cingulate cortex, insula, and thalamus. When these neuroanatomical adaptations are considered alongside changes in pineal gland volume and melatonin balance, they suggest that pain, sleep, and neuroendocrine regulation in AS are influenced by a common central mechanism.

The relationship between pineal gland volume and various neurological and psychiatric disorders such as Alzheimer's disease, schizophrenia, obsessive-compulsive disorder, personality disorders, multiple sclerosis, fibromyalgia, adolescent idiopathic scoliosis, and REM sleep behavior disorder has been investigated (Fındıklı *et al*., 2015; Matsuoka *et al*., 2018; Atmaca *et al*., 2019; Park *et al*., 2020; Batın *et al*., 2023; Vukovic´ *et al*., 2024; Çiçek *et al*., 2025). In all of these studies, it has been reported that the pineal gland volume in individuals with these diseases is statistically significantly lower compared to the healthy control group. The literature indicates that there are studies investigating the possible relationship between spinal deformities and the pineal gland, particularly in the pathophysiology of postural curvatures such as scoliosis, both in humans and in animal models (Turgut *et al*., 2003; Fjelldal *et al*., 2004; Batın *et al*., 2023). Following pinealectomy in experimental chicken and fish models, it has been reported that significant structural abnormalities such as scoliosis and kyphosis developed in the spine. These structural changes were accompanied by significant decreases in the mineral density and mechanical strength of the spinal bones. The findings suggest that melatonin deficiency may be a significant etiological factor in the development of these deformities (Turgut *et al*., 2003; Fjelldal *et al*., 2004). In a study examining the pineal gland in children with adolescent idiopathic scoliosis (AIS), the gland volume was found to be 38 % lower than in healthy controls. It has been reported that this reduction in volume may play a role in the development of scoliosis (Batın *et al*., 2023).

A review of the literature reveals that, while there are no studies directly evaluating the effect of AS on pineal gland volume, there are numerous studies on melatonin levels in individuals with AS (Senna *et al*., 2012; Hizmetli *et al*., 2015; Li *et al*., 2020). Li *et al*. (2020) reported that melatonin levels in individuals with AS were significantly higher than in healthy individuals and suggested that this increase may be related to spinal ossification processes. In addition to melatonin's known antioxidant and anti-inflammatory effects, it was emphasized that it supports new bone formation by affecting bone metabolism and increasing osteoblast activity. The same study stated that, in addition to chronic inflammation in AS, increased melatonin levels may play a role in the development of ankylosis and spinal fusion (Li *et al*., 2020). Senna *et al*. (2012) reported that serum melatonin levels were significantly elevated in AS patients, with this increase being more pronounced in the active disease group. The study found a positive correlation between melatonin levels and both inflammatory markers (CRP, ESR) and radiographic progression. These findings suggest that melatonin may be associated not only with the inflammatory response in AS pathophysiology, but also with ossification processes (Senna *et al*., 2012). According to these studies, more multifaceted studies have also been conducted examining the relationship between melatonin levels and pineal gland volume, and it has been found that pineal gland parenchymal volume shows a positive correlation with melatonin levels (Sigurdardottir *et al*., 2016; Bazzi *et al*., 2021; León-Llamas *et al*., 2022).

Previous studies have reported that melatonin levels in individuals with AS are significantly higher than in healthy individuals (Senna *et al*., 2012; Hizmetli *et al*., 2015; Li *et al*., 2020). Since our study was retrospective, melatonin measurements were not performed, and no data could be obtained regarding the relationship between possible changes in the levels of this hormone and the volume of the pineal gland.

In our study, there was no statistically significant difference between the AS group and the control group in terms of pineal gland volume, cerebrum volume, intracranial

volume, pineal gland/cerebrum volume ratio, and pineal gland/intracranial volume ratio. This suggests that the increase in melatonin levels cannot be explained solely by changes in pineal gland volume; various factors such as neurohormonal regulation, physiological adaptations, and interindividual functional differences are also quite important. Furthermore, the effects of chronic inflammation in AS on the central nervous system have not yet been fully elucidated. Considering all these reasons, it can be argued that the relationship between melatonin production and pineal gland volume is complex and that volumetric assessments alone may not be conclusive unless supported by biochemical parameters.

Our study is a pioneering investigation comparing the pineal gland, cerebrum, and total intracranial volumes in AS patients. However, there are some methodological limitations. First, the retrospective design of the study did not allow for the standardization of variables such as disease duration, medication use, physical activity level, and lifestyle. Second, the relatively limited sample size may have made it difficult to statistically detect small volumetric differences.

CONCLUSIONS

Our study revealed no significant differences between individuals with AS and healthy individuals in terms of pineal gland, cerebrum, and total intracranial volumes. This result suggests that chronic inflammation associated with AS may not cause a gross volume change detectable at the structural level with current imaging resolution and methods, and that neuroanatomical effects at the microstructural or functional level may not yet be apparent at the volumetric level. We believe that our study will make important contributions to the literature in terms of pioneering future studies.

URHAN, A. T.; DOGAN, B.; IRMAK SAPMAZ, H.; ASLAN BARAN, S.; KOKCU, F. & OZTURK, M. F. ¿Afecta la espondilitis anquilosante el volumen de la glándula pineal? *Int. J. Morphol., 44(3)*:1008-1013, 2026.

RESUMEN: Se cree que la espondilitis anquilosante (EA), que afecta principalmente al esqueleto axial, puede causar efectos estructurales y funcionales en el cerebro a través de la inflamación crónica y mecanismos neuroendocrinos. El objetivo de este estudio fue investigar los posibles efectos de la EA en la glándula pineal (Gl), el cerebro y el volumen intracraneal mediante resonancia magnética (RM). En este estudio, se evaluaron retrospectivamente imágenes de 25 pacientes con EA y 25 individuos sanos de edad y sexo similares. El volumen de la glándula pineal se examinó mediante segmentación manual con el software 3D Slicer (versión 4.10.2) en imágenes de resonancia magnética ponderadas en T1 de alta resolución, mientras que los volúmenes cerebrales e intracraneales se examinaron mediante el método de segmentación automática VolBrain. Se utilizó la prueba t de Student para el análisis estadístico. No se encontraron diferencias estadísticamente significativas entre el grupo con espondilitis anquilosante (EA) y el grupo control en cuanto al volumen de la glándula pineal, el cerebro y el volumen intracraneal ($p > 0,05$). Del mismo modo, no se encontraron diferencias significativas en las proporciones de volumen glándula pineal/cerebro y glándula pineal/volumen intracraneal ($p > 0,05$). Si bien se cree que los mecanismos neuroinflamatorios y neuroendocrinos desempeñan un papel en la fisiopatología de la EA, nuestros hallazgos indican que estos procesos no dan lugar a diferencias estructurales significativas en el volumen cerebral. Sin embargo, estudios más avanzados con grupos de muestra más grandes que también analicen bioquímicamente los niveles de melatonina contribuirán a una comprensión más completa de esta relación.

PALABRAS CLAVE: Espondilitis anquilosante; Glándula pineal; Cerebro; VolBrain.

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