Radiology Case Reports 20 (2025) 1317–1322

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/radcr

Case Report

Pineal cyst in bipolar patient with normolithiaemia and positive fibromyalgic tender points ☆

Salvatore Marrone, MD[a], *Antonio Alessandro Biancardino, MD*[b,*], *Evier Andrea Giovannini, MD*[a,b], *Federica Paolini, MD*[a,b], *Benedetta Maria Campisi, MD*[b], *Jaime Mandelli, MD*[a], *Domenico Santangelo, MD*[c], *Salvatore Fanara, MD*[c], *Giuseppe Vaccaro, MD*[d], *Michele Vecchio, MD*[c], *Domenico Gerardo Iacopino, MD, PhD*[b], *Luigi Basile, MD*[a]

[a] *Unit of Neurosurgery, Sant'Elia Hospital, via Luigi Russo n° 6, Caltanissetta, Italy*
[b] *Neurosurgical Clinic, AOUP "Paolo Giaccone", Post Graduate Residency Program in Neurologic Surgery, Department of Biomedicine Neurosciences and Advanced Diagnostics, School of Medicine, University of Palermo, Palermo, Italy*
[c] *Unit of Neurology, Sant'Elia Hospital, via Luigi Russo n° 6, Caltanissetta, Italy*
[d] *Unit of Neuroradiology, Sant'Elia Hospital, via Luigi Russo n° 6, Caltanissetta, Italy*

ARTICLE INFO

Article history:
Received 27 September 2024
Revised 27 October 2024
Accepted 11 November 2024

Keywords:
Pineal cyst
Bipolar disorder
Fibromyalgia
Systemic inflammation
Lithium therapy

ABSTRACT

Pineal cysts are benign, nonneoplastic lesions of the pineal gland, often identified incidentally on MRI scans. Although these cysts are usually asymptomatic, they can occasionally enlarge and compress adjacent structures, leading to neurological complications such as obstructive hydrocephalus and Parinaud's syndrome. The underlying mechanisms of pineal cyst development remain largely unclear, although inflammation - common in rheumatological conditions such as fibromyalgia - and mechanical stress have been suggested as contributing factors. In addition, the incomplete blood-brain barrier of the pineal gland raises the possibility that chronic lithium therapy, commonly used for psychiatric disorders and also known for its hyperplastic effects, could facilitate cysts formation through lithium accumulation and epithelial stimulation.

We report the case of a 49-year-old woman with bipolar disorder on long-term lithium treatment who presented with a pineal cyst and clinical symptoms consistent with fibromyalgia. A review of the literature highlights possible links between pineal cyst formation, systemic inflammation associated with rheumatological disorders and prolonged lithium exposure.

Although the hyperplastic properties of lithium in glandular tissue are well documented, there is no conclusive evidence directly linking lithium use to the development of pineal cysts in humans. The possibility of cystic growth driven by the pro-inflammatory environment of fibromyalgia remains plausible and warrants further investigation of the complex

☆ Competing Interests: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

* Corresponding author.
E-mail address: antonio.alessandro.biancardino@gmail.com (A. Alessandro Biancardino).

https://doi.org/10.1016/j.radcr.2024.11.028

interactions between lithium therapy, systemic inflammation and pineal cystogenesis, particularly in patients with coexisting rheumatological and psychiatric disorders.

Introduction

Pineal cysts are benign, nonneoplastic lesions of the pineal gland that are often identified incidentally during magnetic resonance imaging (MRI) in adults [1]. MRI studies estimate their prevalence to be around 10% in the general population, with a higher incidence in women.

However, postmortem examinations report a significantly higher prevalence, with pineal cysts found in up to 40% of individuals, suggesting that many cases remain asymptomatic and undetected throughout life [1,2]. Although typically asymptomatic, some cysts may enlarge and compress adjacent structures such as the tectum, cerebral aqueduct and venous pathways, potentially leading to complications such as obstructive hydrocephalus and Parinaud's syndrome in which case surgical intervention is required [3–5]. Although factors like inflammation and post-traumatic mechanical stress have been implicated in the development of acquired pineal cysts, the natural history and pathophysiology of these lesions remain poorly understood [6,7]. Neuronal cysts and neuroanatomical changes have been reported in rheumatological conditions such as fibromyalgia, supporting a broader connection between systemic inflammation and neuroanatomical alterations [8,9]. Additionally, emerging evidence indicates that chronic lithium accumulation may contribute to cyst formation in various organs, including the kidneys, uterus, and even in arachnoid. In particular, given the incomplete blood-brain barrier of the pineal gland, it is hypothesized that lithium deposition may similarly promote pineal cyst formation in patients on chronic lithium therapy, highlighting the need for further investigation into the role of lithium in pineal cystogenesis [10–12].

In this context, we report the case of a patient with bipolar disorder on lithium therapy who presented with a pineal cyst and clinical features consistent with fibromyalgia. A literature review was conducted to explore potential associations between pineal cysts formation, chronic lithium accumulation and the systemic pro-inflammatory environment characteristic of rheumatological disorders.

Case description

A 49-year-old female with a history of bipolar disorder, managed with long-term lithium therapy, presented to the emergency department with progressively worsening bilateral headaches, primarily localized to the occipital-cervical and parietal regions. The headache intensity was rated as 7/10 on the Visual Analogue Scale (VAS). In addition to the headache, the patient reported significant difficulty sleeping and widespread muscle stiffness affecting both the scapular and pelvic girdles. Upon admission, her Glasgow Coma Scale (GCS) was 15/15, indicating full consciousness.

Physical examination revealed severe pain elicited by percussion of the trapezius and latissimus dorsi muscles, consistent with fibromyalgia tender points (Fig. 1), despite no prior diagnosis of fibromyalgia in her medical history. Digital pressure applied to these regions also triggered a pronounced painful response. Laboratory results were within normal ranges, with lithium levels measured at 0.71 mEq/L (reference range: 0.5-1.2 mEq/L). Serum β-HCG was 2.10 mIU/mL, and α-fetoprotein was 0.97 ng/mL, both within normal limits. A comprehensive neurological examination was otherwise unremarkable. The patient had no motor deficits, her cranial nerves were intact, and there were no signs of myelopathy.

A brain MRI with gadolinium contrast revealed a cystic lesion in the pineal region without evidence of third-ventricle obstruction or compression of surrounding parenchymal or cerebrospinal fluid structures (Fig. 2).

In the absence of compression signs over the parenchymal-ventricular structures and without midline shift, the patient was instructed to undergo clinical and neuroradiological follow-up. She was treated with corticosteroid medication (Dexamethasone), with scaled dosage, which allowed moderate benefit on the symptomatology.

Discussion

The development of acquired pineal cysts is influenced by several factors, with inflammatory and post-traumatic mechanical stimulation being key contributors [13]. In particular, inflammation is a critical component in the pathophysiology of fibromyalgia, although the precise mechanisms remain elusive. Studies primarily focus on the pro-inflammatory state in fibromyalgia, where generalized pain is triggered by pressing specific muscle-tendon junctions known as tender points [12,14]. Research by Bäckryd et al. [15] demonstrated elevated levels of cytokines IL-6 and IL-8 in the CSF of fibromyalgia patients, reinforcing the inflammatory hypothesis. Diagnostic sensitivity for fibromyalgia increases with the number of positive tender points, with 11 out of 18 being desirable for diagnosis; our patient presented with 10 positive points, with pain predominantly localized in the posterior regions.

Lithium, known for its anti-inflammatory properties [16–18], has been investigated for its potential to alter collagen structures in the pachymeninges and leptomeninges [19]. However, in this case, the patient's lithium levels were within normal limits, reducing the likelihood of lithium having a significant anti-inflammatory effect on the widespread inflammation observed in fibromyalgia [20,21]. Furthermore, patients with rheumatological diseases or psychiatric disorders often exhibit elevated pro-inflammatory profiles, which

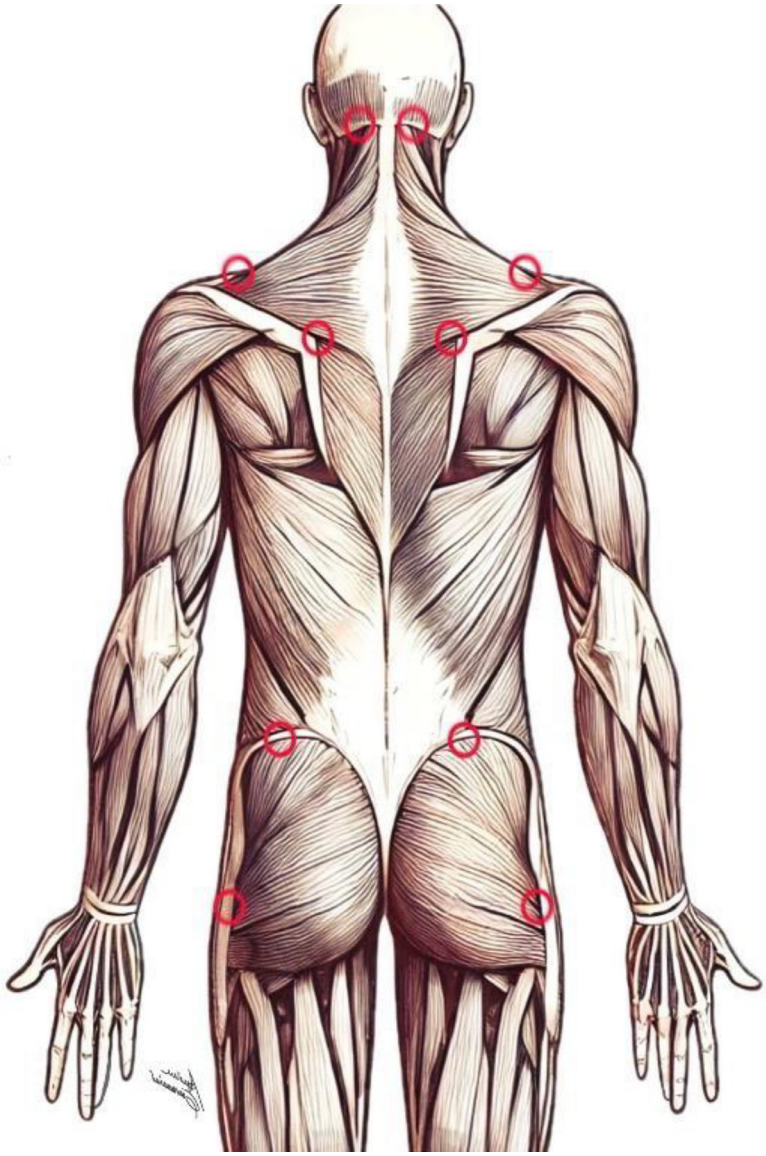

Fig. 1 – This image highlights 10 of the 18 classic fibromyalgia tender points, specifically located at the occipital base, trapezius, supraspinatus, upper gluteal regions, and greater trochanters. These marked areas represent key sites of localized pain and tenderness, which are characteristic of the widespread musculoskeletal pain commonly observed in fibromyalgia.

may influence the permeability of the blood-brain barrier and potentially stimulate cellular proliferation in areas such as the pineal gland [22–25]. This suggests that inflammatory molecules circulating systemically could contribute to cyst formation in the pineal region [26].

The dynamic model of CSF flow, proposed by Hulens and colleagues [27], offers further insight, linking the formation of perineural cysts in fibromyalgia to mechanical forces such as traction and stretching on nerve roots during episodes of idiopathic CSF hypertension. Leon-Llamas' study found a 29.6% prevalence of pineal cysts among 50 fibromyalgia patients, suggesting a potential association between fibromyalgia and cyst formation in the pineal gland [28].

Additionally, Idris et al. [29] reported the coexistence of adhesive arachnoiditis and pelvic cysts in a fibromyalgia patient, underscoring the role of inflammatory stimuli, such as those following subarachnoid hemorrhage, in promoting arachnoidocyte hyperplasia, layer splitting, and cystic formation [30].

While the imaging characteristics of our patient's lesion suggested a pineal cyst, laboratory tests were conducted to rule out germ cell tumors, which can present with cystic necrosis and high mitotic activity [31,32]. Morphological and volumetric changes in the pineal gland have been documented in various psychiatric conditions [33–37], with gland enlargement sometimes leading to cystic transformation due to differential flow in superficial versus deep microcirculation [38]. Warsza's research indicates that pineal cysts exceeding 1 mm in diameter are more commonly associated with anatomical variations such as pineal recess crowding and a high

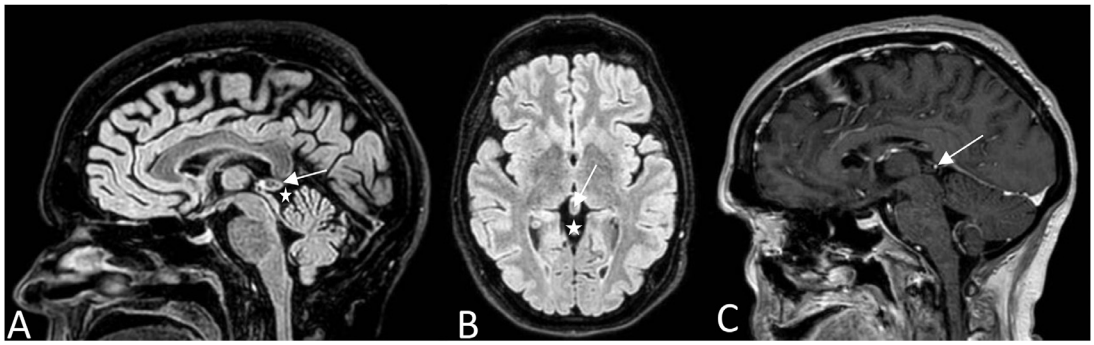

Fig. 2 – MRI study showing the pineal cyst. (A, B) Hyperintense lesion in FLAIR sequences. (C) T1 Contrast-enhancement sagittal sequence of the lesion. (A) The white arrow points to the cyst wall appearing slightly hyperintense. (B-C) White arrow indicates a cystic lesion in the pineal region. The white star highlights the quadrigeminal cistern (Bichat's canal) that surrounds the pineal gland anteriorly, the splenium of the corpus callosum superiorly, the quadrigeminal tubercles antero-inferiorly and the superior surface of the cerebellar vermis inferiorly (A-B).

cyst-tectum-splenium (CST) ratio, highlighting the importance of neuroradiological parameters in assessing cyst development [39].

The potential interaction between exogenous growth hormone (GH) and brain cyst size has been described in pediatric populations [40], with studies suggesting that molecules like lithium could influence endocrine function, including the stimulation of GH [41]. While lithium's effect on parathyroid morphology and secretion has been well-documented, few studies have explored its impact on the pineal gland [42–46]. Takahashi et al. [47,48] observed increased pineal gland volume in melancholic patients treated for major depressive disorder, although no correlation was found between other psychiatric comorbidities and pineal cyst development. In cases of hyperlithiemia, renal cyst formation has been linked to dilated renal tubules, suggesting that lithium accumulation may have similar effects on cystic development in other tissues [49].

While lithium is known to reduce melatonin secretion in some mammals by inhibiting the enzyme arylalkylamine-N-acetyltransferase, this was not a factor in our patient as her pineal cyst morphology and normal lithium levels did not suggest a direct correlation [42]. However, the patient's systemic pro-inflammatory state likely played a critical role in the development of the pineal cyst, further supporting the hypothesis that inflammation, rather than lithium, may be the primary driver of cystogenesis in this setting.

Conclusions

Lithium is recognized for its hyperplastic effects on glandular epithelia and various organs. Although no direct evidence currently links lithium use to the formation of pineal cysts in humans, the potential for cystic growth induced by chronic inflammation, particularly in the context of systemic rheumatologic conditions such as fibromyalgia, cannot be entirely dismissed. Pro-inflammatory stimuli characteristic of these disorders may contribute to epithelial proliferation, raising the possibility of late-onset pineal cystogenesis. Further research is essential to elucidate the complex interplay between inflammatory pathways and prolonged lithium therapy in the development of acquired pineal cysts, particularly in patients with overlapping psychiatric and rheumatological conditions.

Patient consent

Written informed consent for the publication of this report was obtained from the patient.

REFERENCES

[1] Jouvet A, Vasiljevic A, Champier J, Fèvre Montange M. Pineal parenchymal tumours and pineal cysts. Neurochirurgie 2015;61(2-3):123–9. doi:10.1016/j.neuchi.2013.04.003.
[2] Taveras JM, Wood EH. Diagnostic neuroradiology. Acad Med 1964;39(12):1135.
[3] Kabuto M, Hayashi M, Kawano H, Kobayashi H, Ishii H, Shirasaki N, et al. [A case of non-neoplastic pineal cyst presenting Parinaud's syndrome]. No Shinkei Geka 1987;15(3):335–8.
[4] Fleege MA, Miller GM, Fletcher GP, Fain JS, Scheithauer BW. Benign glial cysts of the pineal gland: unusual imaging characteristics with histologic correlation. AJNR Am J Neuroradiol 1994;15(1):161–6.
[5] Jenkinson MD, Mills S, Mallucci CL, Santarius T. Management of pineal and colloid cysts. Pract Neurol 2021;21(4):292–9. doi:10.1136/practneurol-2020-002838.
[6] Epelman M, Daneman A, Blaser SI, Ortiz-Neira C, Konen O, Jarrín J, et al. Differential diagnosis of intracranial cystic lesions at head US: correlation with CT and MR imaging. Radiographics 2006;26(1):173–96. doi:10.1148/rg.261055033.
[7] Biancardino AA, Marrone S, Paolini F, Giovannini EA, Cinquemani G, Lipani R, et al. Coexistence of mastoid, frontal and vertebral hemangiomas in a patient with diabetic neuropathy: possible correlation between diabetic angiopathy and intraosseous neoangiogenesis. Radiol Case Rep. 2024;19(8):2937–42. doi:10.1016/j.radcr.2024.03.087.

[8] Fallon N, Alghamdi J, Chiu Y, Sluming V, Nurmikko T, Stancak A. Structural alterations in brainstem of fibromyalgia syndrome patients correlate with sensitivity to mechanical pressure. Neuroimage Clin 2013;3:163–70. doi:10.1016/j.nicl.2013.07.011.
[9] Hulens M, Bruyninckx F, Thal DR, Rasschaert R, Bervoets C, Dankaerts W. Large- and small-Fiber neuropathy in patients with Tarlov cysts. J Pain Res 2022;15:193–202. doi:10.2147/JPR.S342759.
[10] Gunin AG, Emelianov VU, Mironkin IU, Morozov MP, Tolmachev AS. Lithium treatment enhances estradiol-induced proliferation and hyperplasia formation in theuterus of mice. Eur J Obstet Gynecol Reprod Biol 2004;114(1):83–91. doi:10.1016/j.ejogrb.2003.09.023.
[11] Tuazon J, Casalino D, Syed E, Batlle D. Lithium-associated kidney microcysts. ScientificWorldJournal 2008;8:828–9. doi:10.1100/tsw.2008.112.
[12] Sarzi-Puttini P, Giorgi V, Marotto D, Atzeni F. Fibromyalgia: an update on clinical characteristics, aetiopathogenesis and treatment. Nat Rev Rheumatol 2020;16(11):645–60. doi:10.1038/s41584-020-00506-w.
[13] Costanzo R, Balasubramanian K, Marrone S, Porzio M, Iacopino DG, Nicoletti GF, et al. Letter to the editor regarding "natural history of intracranial arachnoid cysts". World Neurosurg 2023;173:286–8. doi:10.1016/j.wneu.2023.01.115.
[14] Tastekin N, Birtane M, Uzunca K. Which of the three different tender points assessment methods is more useful for predicting the severity of fibromyalgia syndrome? Rheumatol Int 2007;27(5):447–51. doi:10.1007/s00296-006-0232-2.
[15] Bäckryd E, Tanum L, Lind AL, Larsson A, Gordh T. Evidence of both systemic inflammation and neuroinflammation in fibromyalgia patients, as assessed by a multiplex protein panel applied to the cerebrospinal fluid and to plasma. J Pain Res 2017;10:515–25. doi:10.2147/JPR.S128508.
[16] Matur E, Akyol S, Toplan S, Ozdemir S, Akyazı I, Darıyerli N. Impact of lithium on the immune system: an investigation of T-cell subpopulations and cytokine responses in rats. Biol Trace Elem Res 2024;1:1–9. doi:10.1007/s12011-024-04202-8.
[17] Queissner R, Lenger M, Birner A, Dalkner N, Fellendorf F, Bengesser S, et al. The association between anti-inflammatory effects of long-term lithium treatment and illness course in Bipolar Disorder. J Affect Disord 2021;281:228–34. doi:10.1016/j.jad.2020.11.063.
[18] Sakrajda K, Szczepankiewicz A. Inflammation-related changes in mood disorders and the immunomodulatory role of lithium. Int J Mol Sci 2021;22(4):1532. doi:10.3390/ijms22041532.
[19] Tzaphlidou M, Kounadi E. The effect of lithium treatment on collagenous tissues: an electron microscope study. Micron 1998;29(2-3):235–48. doi:10.1016/s0968-4328(97)00060-7.
[20] O'Mahony LF, Srivastava A, Mehta P, Ciurtin C. Is fibromyalgia associated with a unique cytokine profile? A systematic review and meta-analysis. Rheumatology (Oxford) 2021;60(6):2602–14. doi:10.1093/rheumatology/keab146.
[21] Ernberg M, Christidis N, Ghafouri B, Bileviciute-Ljungar I, Löfgren M, Bjersing J, et al. Plasma cytokine levels in fibromyalgia and their response to 15 weeks of progressive resistance exercise orRelaxation therapy. Mediators Inflamm 2018;2018:3985154. doi:10.1155/2018/3985154.
[22] Ranzolin A, Duarte AL, Bredemeier M, da Costa Neto CA, Ascoli BM, et al. Evaluation of cytokines, oxidative stress markers and brain-derived neurotrophic factor in patients with fibromyalgia - A controlled cross- sectional study. Cytokine 2016;84:25–8. doi:10.1016/j.cyto.2016.05.011.
[23] Tanaka Y, Luo Y, O'Shea JJ, Nakayamada S. Janus kinase-targeting therapies in rheumatology: a mechanisms-based approach. Nat Rev Rheumatol 2022;18(3):133–45. doi:10.1038/s41584-021-00726-8.
[24] Müller N, Ackenheil M. Psychoneuroimmunology and the cytokine action in the CNS: implications for psychiatric disorders. Prog Neuropsychopharmacol Biol Psychiatry 1998;22(1):1–33. doi:10.1016/s0278-5846(97)00179-6.
[25] Williams JA, Burgess S, Suckling J, Lalousis PA, Batool F, Griffiths SL, et al. PIMS Collaboration. Inflammation and brain structure in schizophrenia and other neuropsychiatric disorders: a mendelian randomization study. JAMA Psychiatry 2022;79(5):498–507. doi:10.1001/jamapsychiatry.2022.0407.
[26] Dong BT, Tu GJ, Han YX, Chen Y. Lithium enhanced cell proliferation and differentiation of mesenchymal stem cells to neural cells in rat spinal cord. Int J Clin Exp Pathol 2015;8(3):2473–83.
[27] Hulens M, Bruyninckx F, Dankaerts W, Rasschaert R, De Mulder P, Stalmans I, et al. High prevalence of perineural cysts in patients with fibromyalgia and chronic fatigue syndrome. Pain Med 2021;22(4):883–90. doi:10.1093/pm/pnaa410.
[28] Leon-Llamas JL, Villafaina S, Murillo-Garcia A, Rohlfs Domínguez P, Gusi N. Relationship between pineal gland, sleep and melatonin in fibromyalgia women: a magnetic resonance imaging study. Acta Neuropsychiatr 2021;34(2):1–9. doi:10.1017/neu.2021.35.
[29] Idris Z, Ghazali FH, Abdullah JM. Fibromyalgia and arachnoiditis presented as an acute spinal disorder. Surg Neurol Int 2014;5:151. doi:10.4103/2152-7806.143364.
[30] Tumialán LM, Cawley CM, Barrow DL. Arachnoid cyst with associated arachnoiditis developing after subarachnoid hemorrhage. Case report. J Neurosurg 2005;103(6):1088–91. doi:10.3171/jns.2005.103.6.1088.
[31] Hayashida Y, Hirai T, Korogi Y, Kochi M, Maruyama N, Yamura M, et al. Pineal cystic germinoma with syncytiotrophoblastic giant cells mimicking MR imaging findings of a pineal cyst. AJNR Am J Neuroradiol 2004;25(9):1538–40.
[32] Upadhyayula PS, Neira JA, Miller ML, Bruce JN. Benign and malignant tumors of the pineal region. Adv Exp Med Biol 2023;1405:153–73.
[33] Sarrazin S, Etain B, Vederine FE, d'Albis MA, Hamdani N, Daban C, et al. MRI exploration of pineal volume in bipolar disorder. J Affect Disord 2011;135(1-3):377–9. doi:10.1016/j.jad.2011.06.001.
[34] Wetterberg L. The relationship between the pineal gland and the pituitary–adrenal axis in health, endocrine and psychiatric conditions. Psychoneuroendocrinology 1983;8(1):75–80. doi:10.1016/0306-4530(83)90042-2.
[35] Fındıklı E, Inci MF, Gökçe M, Fındıklı HA, Altun H, Karaaslan MF. Pineal gland volume in schizophrenia and mood disorders. Psychiatr Danub 2015;27(2):153–8.
[36] Chauhan S, Barbanta A, Ettinger U, Kumari V. Pineal Abnormalities in psychosis and mood disorders: A systematic review. Brain Sci 2023;13(5):827. doi:10.3390/brainsci13050827.
[37] Zhao W, Zhu DM, Zhang Y, Zhang C, Wang Y, Yang Y, et al. Pineal gland abnormality in major depressive disorder. Psychiatry Res Neuroimaging 2019;289:13–17. doi:10.1016/j.pscychresns.2019.05.004.
[38] Higashi K, Katayama S, Orita T. Pineal apoplexy. J Neurol Neurosurg Psychiatry 1979;42(11):1050–3. doi:10.1136/jnnp.42.11.1050.
[39] Warsza B, Due-Tønnessen P, Due-Tønnessen P, Pripp A, Ringstad G, Eide PK. Prevalence of pineal cysts in healthy individuals: emphasis on size, morphology and pineal recess crowding. J Neurol Sci 2023;453:120801. doi:10.1016/j.jns.2023.120801.

[40] Baird K, McCroskey J, Arynchyna A, Abdullatif H, Ashraf AP, Simpson L, et al. Incidental pituitary cysts in children: does growth hormone treatment affect cyst size? Endocr Pract 2021;27(11):1128–32. doi:10.1016/j.eprac.2021.04.887.
[41] Hirschowitz J, Zemlan FP, Garver DL. Growth hormone levels and lithium ratios as predictors of success of lithium therapy in schizophrenia. Am J Psychiatry 1982;139(5):646–9. doi:10.1176/ajp.139.5.646.
[42] Fuentes LB, Calderón CP, García Aseff SB, Muñoz EM, Moller M, Pelzer LE. Effect of lithium on the melatonin production in the pineal gland of viscacha. Biological Rhythm Research 2007;39(1):43–55. doi:10.1080/09291010701292086.
[43] Seggie J, Werstiuk ES, Grota L. Lithium and circadian patterns of melatonin in the retina, hypothalamus, pineal and serum. Prog Neuropsychopharmacol Biol Psychiatry 1987;11(2-3):325–34. doi:10.1016/0278-5846(87)90077-7.
[44] Pablos MI, Santaolaya MJ, Agapito MT, Recio JM. Influence of lithium salts on chick pineal gland melatonin secretion. Neurosci Lett 1994;174(1):55–7. doi:10.1016/0304-3940(94)90117-1.
[45] Hallam Karen T, Olver James S, Horgan Jennifer E, McGrath Caroline, Norman Trevor R. Low doses of lithium carbonate reduce melatonin light sensitivity in healthy volunteers. Int J Neuropsychopharmacol 2005;8(Issue 2):255–9 Pages. doi:10.1017/S1461145704004894.
[46] Pattan V, Singh B, Abdelmoneim SS, Gopinath C, Sundaresh V. Lithium-induced hyperparathyroidism: an ill-defined territory. Psychopharmacol Bull 2021;51(3):65–71.
[47] Takahashi T, Sasabayashi D, Yücel M, Whittle S, Lorenzetti V, Walterfang M, et al. Pineal gland volume in major depressive and bipolar disorders. Front Psychiatry 2020;11:450. doi:10.3389/fpsyt.2020.00450.
[48] Takahashi T, Wood SJ, Yung AR, Nelson B, Lin A, Yuen HP, et al. Pineal morphology of the clinical high-risk state for psychosis and different psychotic disorders. Schizophr Res 2022;244:1–7. doi:10.1016/j.schres.2022.04.005.
[49] Vanacker A, Van Dorpe J, Maes B. Cystic kidney disease in a patient with long-term lithium therapy. NDT Plus 2009;2(2):179–80. doi:10.1093/ndtplus/sfn206.