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CASE REPORT

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Avoidant/restrictive food intake disorder-like syndrome and cerebellar tumor in an adolescent: a case report

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Abstract

Background Avoidant/Restrictive Food Intake Disorder (ARFID) is a recently recognized eating disorder category in the DSM-5 and ICD-11classifications. Recent functional neuroimaging studies have suggested alterations in the cerebellar intrinsic connectivity networks in patients suffering from eating disorders. Associations between eating disorders and central nervous system tumors have been documented, but to date no studies have linked eating disorders to cerebellar lesions. This case report presents a 13-year-old boy with ARFID symptoms and a cerebellar tumor, exploring the potential connection between the two.

Case presentation A 13-year-old adolescent with a history of dental agenesis, escalating food restriction, severe abdominal pain, impaired weight gain and statural growth was initially diagnosed with ARFID. A brain magnetic resonance imaging revealed a large and threatening cerebellar tumor, leading to urgent neurosurgery. After tumor removal, the patient's eating behaviors, weight, and growth but also puberty improved dramatically. One year later, a tumor remnant was found, necessitating targeted therapy.

Conclusions This case underscores the possibility that cerebellar tumors can mimic ARFID-like syndrome, suggesting a neurological origin for the observed disordered eating behaviors. The marked improvement in eating patterns and growth after tumor removal strongly suggests a direct link between the cerebellar tumor and the patient's symptoms. Given the rarity and complexity of such cases, neurological evaluations, including brain magnetic resonance imaging, should be considered in any children and adolescents with eating disorder or severe disordered eating especially when growth is affected, or unusual symptoms are present.

Level of evidence level V, case report.

Keywords Avoidant/restrictive food intake disorder, Cerebellar tumor, Disordered eating behaviors, Neurological manifestations, Case report

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Background

Avoidant/Restrictive Food Intake Disorder (ARFID) was introduced as a specific diagnosis in the DSM-5 classification of feeding and eating disorder (ED) and more recently in the ICD-11 [1, 2]. ARFID, which replaces and enhances the "feeding disorder of infancy and early childhood" category, is a heterogeneous disorder that does not only affect children. On neuroimaging, patients with ARFID would have lower subcortical surface areas and volumes than their peers without ED [3]. Associations between EDs and central nervous system (CNS) tumors involving several cerebral areas have been documented [4]. Clinical presentation of ED seems to differ depending on the histological type and/or the position of the cerebral tumor. Anorexia Nervosa (AN) appears to be more common in low-grade gliomas and in pilocytic astrocytomas, whereas hyperphagia is more frequently observed in craniopharyngiomas. It is currently established that hypothalamic lesions can influence eating behaviors, with a loss or increase of appetite [5]. Lesions of the brainstem seem to be associated with a symptomatology of food restriction and with the presence of vomiting by direct stimulation of the vomiting center located in the brainstem [4]. To date, there are no studies reporting association between EDs and cerebellar lesions despite the involvement of the cerebellum on body perception and self-image, as well as its regulation of autonomic functions that impact eating behaviors [6]. We reported the case of a 13-year-old boy with a clinical course suggestive of ARFID and a massive cerebellum tumor.

Case presentation

A 13-year-old boy was referred to our specialized consultation for EDs. He was born full-term with a birth weight of 3410 g and a length of 50 cm. He had no eating difficulties in early childhood. An agenesis of the two upper lateral incisors was observed. Around the age of 10, the patient began to complain of abdominal pain, which was described as intense, beginning around the umbilical region, and spreading rapidly throughout the abdominal area. The pain was characterized by its severity, with a desire to "cut open his stomach with a knife" due to its distressing intensity. Concomitantly, the patient displayed an escalating food restriction, particularly exacerbated during school time. These dietary symptoms gradually worsened in severity over time. He limited his food intake, out of anxious anticipation and for fear of abdominal pain. Food quantities became increasingly reduced. The patient showed food selectivity, preferring fatty and sweet foods and excluding fruits and vegetables. He was not purging, was not taking medication to control his weight, and was not engaging in physical hyperactivity. The patient did not show any lack of interest in eating or sensory problems. Significant family history among first degree relatives included: agenesis of the upper lateral incisors in the oldest brother, and pulmonary teratoma and desmoid tumor of the rectus muscle in the mother in adulthood. A high-grade glial tumor in the maternal grandfather, pleural cancer in the paternal grandfather, and ovarian cancer in the paternal grandmother were also reported. There were no previous familial cases of EDs nor psychiatric disorders. At the age of 13, the adolescent was referred because of his eating difficulties. During the initial ED specialized consultation, the adolescent's weight stood at 30 kg for 1.51 m, with a body mass index of 13.0 kg/m² (-3 SD). We observed a loss of 1.5 SD in weight and 1 SD in height compared with the last biometric data (Fig. 1).

The patient did not express a desire for voluntary weight loss, exhibited no preoccupation with body image, and had no fear of gaining weight. The clinical examination was strictly normal with a puberty stage at Tanner 2. The patient did not report symptoms of intracranial hypertension, such as headaches, vomiting, or balance disorders. The patient was alert and oriented. He had no sensory-motor neurological deficits at the time of consultation. Examination of the cranial pairs was unremarkable. Osteotendinous reflexes were normal. There were no walking or balance disorders, no coordination problems and no tightness of the neck. The family only reported a marked change in the adolescent's handwriting over the past year. The initial blood biology test revealed no abnormalities (Table 1).

Combined with his eating difficulties, the adolescent presented with increasingly severe anxiety, which became more pronounced during the school year. He also had episodes of intense anger whenever he was frustrated. The patient and his family underwent regular psychological assessments. No relevant psychosocial difficulties were identified that might contribute to EDs. He had no other psychiatric symptoms and did not require psychotropic or anxiolytic medication. Given the impact on growth, writing difficulties and intense abdominal pain, a brain MRI was requested. The cerebral MRI revealed a median mixed cystic mass in the posterior fossa, measuring 6 × 5 × 6 cm, causing an upward displacement of the cerebellum and anterior displacement of the medulla oblongata and pons. This mass presented a T1 hyposignal and a T2 hypersignal with several mural nodules in intermediate T2 hypersignal, enhancing after injection. In addition, the pituitary gland was abnormally small, with a radiological image of an empty sella turcica, associated with dilation of the lateral ventricles and the fourth ventricle (Fig. 2).

The patient was immediately referred to the pediatric neurosurgical department for removal of the tumor as an extreme emergency. The operation was performed without intraoperative complications. Histopathological

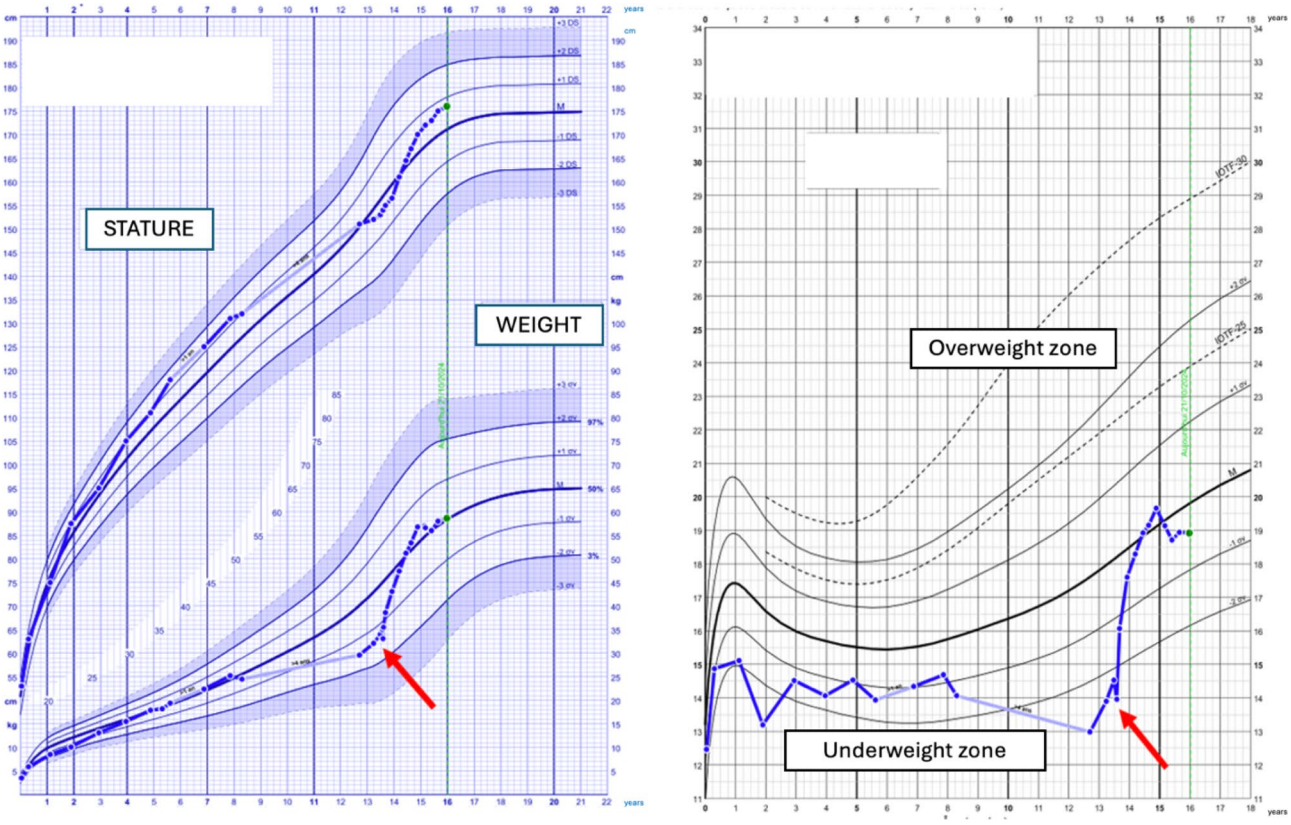

Fig. 1 Patient growth curves. The arrow represents the time of surgical removal of the tumor, followed by a significant ponderal gain, a resumption of growth and an increasing BMI

Table 1 Patient's pre-operative blood test results

Hemoglobin (12.1–16.6 g/dL)	14.0 g/dL
Leukocytes (3.7–13.0 G/L)	5.6 G/L
Platelets (166–395 G/L)	261 G/L
C-reactive protein (< 5.0 mg/l)	< 1.0 mg/l
Sodium (130–145 mmol/L)	140 mmol/L
Potassium (3.4–4.7 mmol/L)	4.0 mmol/L
Pre-albumin (0.18–0.45 g/L)	0.21 g/L
IGF-1 (64–508 nmol/L)	183 nmol/L
TSH (0.5–4.3 mIU/L)	1.6 mIU/L
FT3 (3.9–7.7 pmol/L)	4.7 pmol/L
FT4 (12.6–21.0 pmol/L).	14.4 pmol/L
Prolactin (*N* < 450 mIU/L)	60 mIU/L
Testosterone (0.2–3.0 ng/mL)	0.4 ng/mL
FSH (0.4–5.0 IU/L)	1.1 IU/L
LH (0.5–3.0 IU/L)	< 0.1 IU/L
Cortisol at 8h00 (102–535 nmol/L)	384 nmol/L

assessment of the surgical specimen revealed features consistent with a grade 1 ganglioglioma, expressing the BRAF V600E mutation. A post-operative MRI performed the day after the surgery demonstrated residual tumor tissue, partially hemorrhagic (Fig. 3). A few days after neurosurgery, the patient's eating habits improved impressively. He resumed eating a diet adapted to his age and was no longer afraid of having a stomachache after eating. We continued to follow this patient in our specialized consultation for EDs after the surgical treatment.

One year following neurosurgery, the patient successfully adhered to a diet aligning with age-appropriate nutritional standards, progressively integrating a diverse array of foods without any selectivity. A significant improvement in growth and weight was observed, characterized by a gain of 20.3 kg and 13 cm one year after surgery (Fig. 1). Puberty also progressed to Tanner stage 4. The adolescent no longer reported intense and disabling abdominal pain. His handwriting also improved a few months after surgery. A follow-up brain MRI, conducted one year later, revealed progressive growth of the tumor remnant located in the left cerebellar hemisphere. This remnant measured 41 × 24 mm transversely. The proximity of the tumor to the dentate nucleus ruled out the possibility of further surgery. A decision was made to initiate targeted anti-BRAF therapy, involving a combination of BRAF and MEK inhibitors, with a partial response to this treatment. The improvement in eating habits was constant throughout this period. There were no relapses in dietary difficulties or concerning the patient's weight growth. The 2-year follow-up showed continued increase in weight, growth and puberty, with a weight of 59 kg for

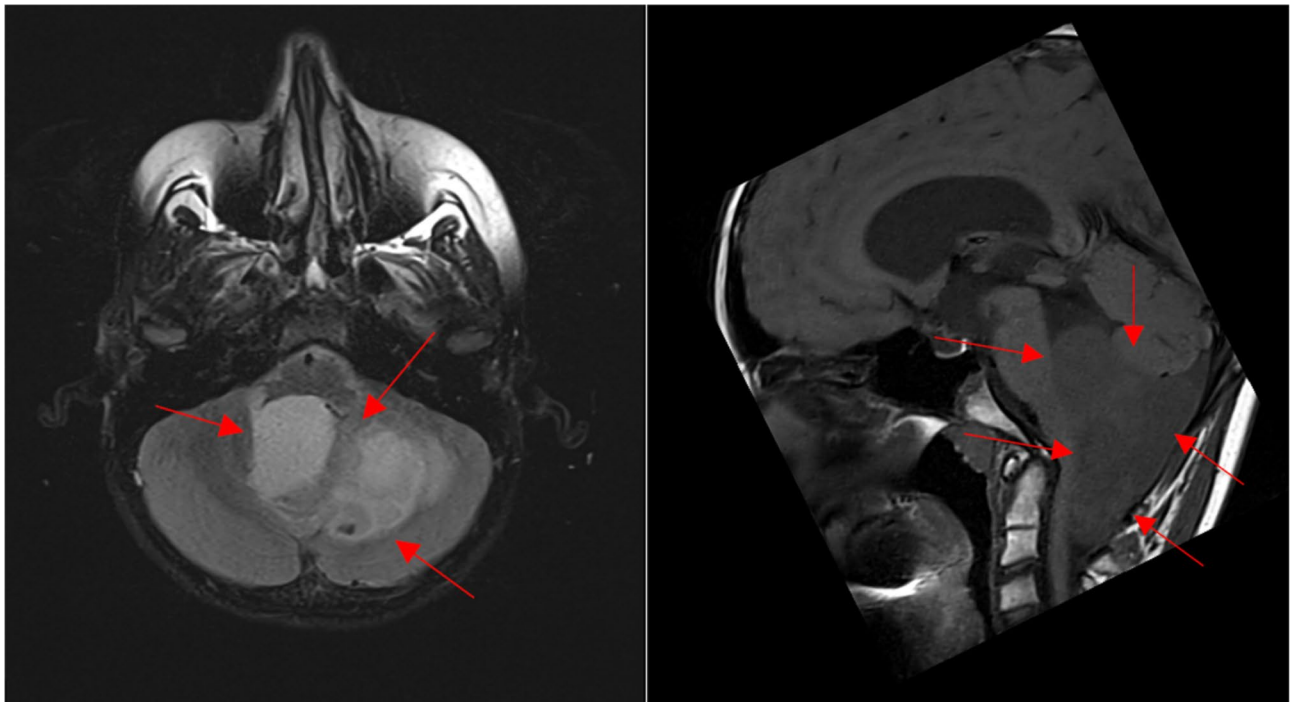

Fig. 2 Pre-operative non-contrast MRI axial Flair and sagittal T1. Arrows represent the cerebellar tumor

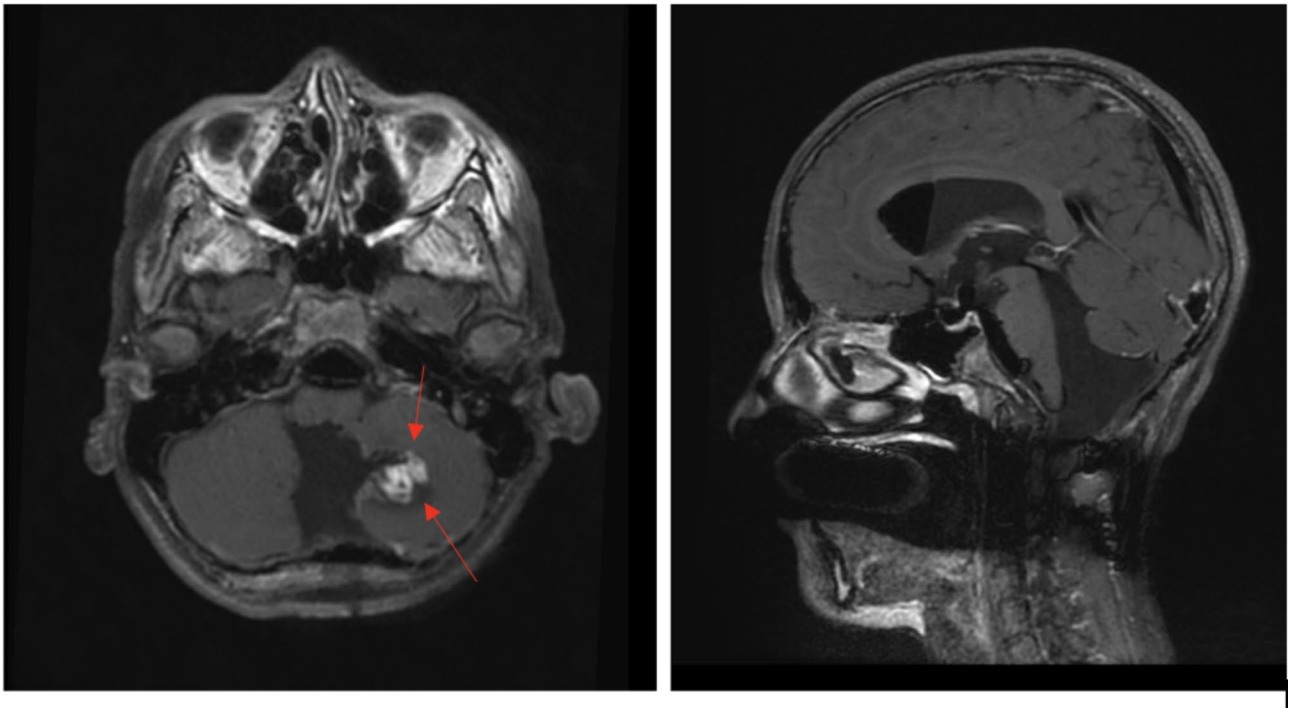

Fig. 3 Post-operative MRI, axial and sagittal with gadolinium injection. Arrows represent the post-operative tumor residue

1m76 (BMI 19). Biological assessment at 2 years revealed a possible incipient thyrotropic deficiency with low TSH level of 0.49 mIU/L (0.5–4.3 mIU/L), slight decrease FT4 level of 12.2 pmol/L (12.6–21 pmol/L) and normal FT3 level of 5,5 pmol/L (3,9 – 7,7 pmol/L), with no disturbance of the other hypothalamic-pituitary axes. The patient and his family have been satisfied and reassured by the collaborative work between our adolescent medicine team and the neurosurgery and oncology departments. Patient has shown excellent adherence and tolerance to anti-BRAF

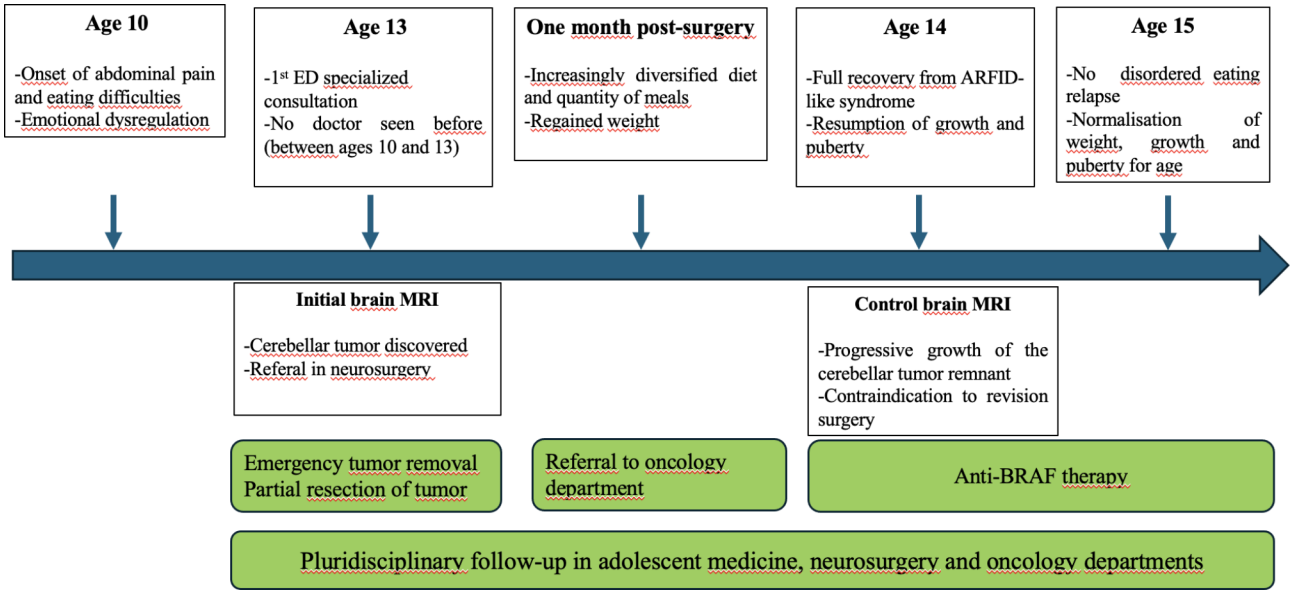

Fig. 4 Clinical events and therapeutic interventions timeline

therapy, and regularly visits our specialized adolescent medical center for somatic and endocrinologic follow-up and psychological support. The sequence of medical events is reported in a timeline (Fig. 4).

Discussion

The diagnosis of ARFID was initially made because the patient had been suffering from marked food restriction and selectivity for several years, with an inability to maintain his daily requirements for age, and without specific diagnostic criteria for AN [1]. There were possibly three years between the onset of symptoms and our first medical assessment. Although this may seem a long time, patients with ARFID have a longer duration of illness before diagnosis than other EDs (12 to 33 months in ARFID versus 8 to 23 months in other EDs) [7]. Chronic eating difficulties had a significant impact on the patient's growth, with a drop-off in height and weight chart patterns. Patients with ARFID may present with gastrointestinal symptoms primarily involving abdominal complaints, attributed to slowed transit or a sensation of fullness and early satiety resulting from delayed gastric emptying as a consequence of malnutrition [8]. The severity and overall description of the abdominal pain experienced by our patient were not typical of the gastrointestinal symptoms usually seen in this ED. ARFID patients may limit their food intake due to fear of aversive consequences, a lack of interest in eating, or sensory preoccupations [9]. Adolescents with the ARFID class characterized by fear of aversive consequences often have normal eating habits before the onset of the disease. Their growth before the eating difficulties was regular, then deteriorates with the ED [10]. Before the brain MRI results, we were confronted with a clinical presentation compatible with a diagnosis of ARFID, more specially fear of aversive reaction subtype [9]. However, we were intrigued by the intense abdominal pain and altered handwriting mentioned by the parents. We therefore decided to perform brain MRI because of these unusual features in ARFID and the severe growth retardation. The scientific literature has already noted cases of ED leading to the discovery of neurological tumors [11]. ED type seems to differ depending on the histological type and/or the position of cerebral tumor. Each histologic type of tumor preferentially affects a specific area of the CNS (hypothalamic-pituitary system, brain, brainstem, or cerebellum). It is not known whether it is the histological type or the location of the tumor that gives rise to the variety of ED presentation. AN seems more common in low-grade gliomas and in pilocytic astrocytoma. Hyperphagia seems to be more frequent in craniopharyngiomas [5]. It is currently established that hypothalamic lesions can influence eating behavior, with a loss or increase in appetite [5]. Lesions of the brainstem seem to be associated with a symptomatology of food restriction and with the presence of vomiting by direct stimulation of the vomiting center located in the brain stem [4]. As for hemispheric lesions, studies show that tumors of the right frontal lobe are more often associated with typical AN symptomatology, although no causal link has yet been established [4, 11]. The presence of new elements in our case with the discovery of the cerebellar tumor, strongly suggests a neurological origin for the altered eating behaviors, mimicking an ARFID presentation. However, the dramatic post-operative improvement raises questions about whether this change is primarily due to

the relief of intracranial hypertension (HTIC) or to the restoration of disrupted connectivity between the cerebellum and other brain regions. In either case, neurosurgery appears to have played a crucial role in reestablishing certain functional cerebellar connections, which likely contributed to the resolution of both the eating behaviors and other associated symptoms [6, 12]. Grade 1 gangliogliomas account for 1–5% of pediatric CNS tumors. Most of these gangliogliomas are found in the cortex, mainly in the temporal lobe (> 70%), although they can also appear in the posterior fossa as was the case in our patient [13]. The BRAF V600E mutation present in our patient is common in this type of tumor, accounting for around 50% of cases [13]. There are no cases in the literature describing EDs in the context of a BRAF V600E mutation and any association with dental agenesis of the incisors [14]. The control of food intake and eating behaviors is based on a complex network of neuro-anatomical and neuro-endocrine structures located mainly in the nuclei of the lateral and ventromedial hypothalamus [15]. Although the cerebellar area does not appear to be classically involved in regulating food intake, this highly sophisticated behavioral process is not yet fully understood. Recent studies have reported altered connectivity network encompassing cerebellum in patients with EDs compared to healthy subjects [10, 11]. The cerebellum plays a crucial role in regulating emotional responses and is interconnected with various brain regions involved in emotional processing, visceral sensations and in the autonomic system. Hyperconnectivity between the cerebellum and the anterior cingulate cortex in people with EDs has been associated with poor emotional regulation and impulsive behavior [6]. This could have an impact on the ability to manage responses related to visceral sensations and emotional processing. Connectivity deficits into the flocculonodular lobe, which is primarily connected to the sensorimotor cortices, have been reported. These data highlight the complex involvement of the cerebellum, not only in motor functions, but also in emotional and cognitive processes, which could explain some of the psychopathological manifestations associated with EDs [16]. Our patient presented with a combination of disordered eating behaviors and difficulty in managing emotional self-regulation. Patients suffering from EDs seem to display greater emotional dysregulation than healthy individuals and, more specifically, patients with ARFID present extremely high rates of anxiety disorders [7]. The dramatic improvement in our patient's condition after neurosurgery supports the hypothesis of an underlying neurological cause justifying both the disturbed eating habits, psychological distress and intracranial hypertension with secondary empty sella. In children, tumors in the hypothalamic-optic chiasma or near the fourth ventricle can manifest as severe failure to thrive [17]. This clinical presentation was first described by Russell in 1951 and is known as diencephalic syndrome characterized by a severe growth retardation or emaciation associated with alteration of hypothalamic structures. Abnormalities in hormonal regulation have also been suggested, such as central hypothyroidism [18]. As seen in our patient, posterior fossa tumors can alter cerebral fluid circulation causing intracranial hypertension with dilatation of the ventricles and a continuous increased intracranial pressure leading to an intrasellar arachnoidocele and a flattened pituitary gland, resulting in an empty sella turcica on MRI [19]. This compression can alter antero-pituitary functions, impacting one or multiple hypothalamo-pituitary axes concurrently. Central hypothyroidism occurs in up to 50% of cases, with some being isolated [20]. Due to the significant risk of developing central hypothyroidism, and the recent fluctuation in our patient's TSH and FT4 levels, vigilant hormonal monitoring was advised. The postoperative improvement in our patient's eating behaviors could also be interpreted as the result of a persuasive implicit suggestion, based on various induction levers. Furthermore, the announcement of the tumor diagnosis could potentially have had a favorable indirect impact on the evolution of the ED's course by mobilizing family interaction and support. Although this hypothesis cannot be definitively ruled out, the full reversibility of eating behavior and the improvement in growth, abdominal pain and handwriting significantly support the explanation of a causal link between the cerebellar ganglioglioma and our patient's clinical presentation. However, the long-term prognosis regarding the impact of the residual tumor on the patient's general health and eating habits remains uncertain.

Conclusion

To our knowledge, this is the first case described in the literature of an adolescent with a cerebellar tumor suffering from a disordered eating mimicking an ARFID-like syndrome. We hypothesize that the altered eating patterns may be neurologically driven, linked to disruptions in cerebellar connectivity networks involving other brain regions. Recent scientific data support the role of cerebellar dysfunction in emotional regulation and eating behaviors, suggesting that alterations in these neural networks—potentially implicating the hypothalamus and cerebellum—may contribute to both the eating disturbances and mood dysregulation. Our case argues for greater clinical awareness of the possible neurological contributions in ARFID. This study strongly supports the role of brain imagery in adolescents with recent disordered eating or atypical ED, particularly in cases of growth retardation, to exclude the possibility of underlying central nervous system tumors.

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Author contributions
M.B., K.B. J.D. and C.B have made substantial contributions to the conception, M.B. and C.B wrote the main manuscript text, M.B., K.B. and C.B prepared Figs. 1, 2 and 3 and M.B., K.B., J.D. , MR.M and C.B revised the work.All the authors have approved the submitted version (and any substantially modified version that involves the author's contribution to the study).All the authors have agreed both to be personally accountable for the author's own contributions and to ensure that questions related to the accuracy or integrity of any part of the work, even ones in which the author was not personally involved, are appropriately investigated, resolved, and the resolution documented in the literature.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Competing interests
The authors declare no competing interests.

Consent to participate and to publish
Informed consent was obtained from all individual participants included in the study. Written informed consent was obtained from the parents. "The patient and the parents provided informed consent for publication of the images in Figure(s) 1, 2 and 3.

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References

1. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Washington, DC: American Psychiatric Publishing; 2013. Available on:. https://library.nic.bc.ca/c.php?g=713252&p=5084517.
2. World Health Organization. International Classification of Diseases (ICD). Available on: https://www.who.int/standards/classifications/classification-of-diseases
3. Moreau CA, Ayrolles A, Ching CRK, Bonicel R, Mathieu A, Stordeur C et al. Neuroimaging insights into brain mechanisms of early-onset Restrictive Eating disorders. Cold Spring Harbor Laboratory; 2024. Available on: https://doi.org/10.1101/2024.11.12.24317128
4. Ghandour F, Squassina A, Karaky R, Diab-Assaf M, Fadda P, Pisanu C. Presenting Psychiatric and neurological symptoms and signs of brain tumors before diagnosis: a systematic review. Brain Sci. 2021;11(3):301.
5. Sherman SJ, Tanaka R, Qaddoumi I. Psychiatric symptoms in children with low-grade glioma and craniopharyngioma: a systematic review. J Psychiatr Res. 2022;148:240–9.
6. Amianto F, D'Agata F, Lavagnino L, Caroppo P, Abbate-Daga G, Righi D, et al. Intrinsic connectivity networks within cerebellum and beyond in eating disorders. Cerebellum Lond Engl. 2013;12(5):623–31.
7. Sanchez-Cerezo J, Nagularaj L, Gledhill J, Nicholls D. What do we know about the epidemiology of avoidant/restrictive food intake disorder in children and adolescents? A systematic review of the literature. Eur Eat Disord Rev. 2023;31(2):226–46.
8. Gibson D, Watters A, Mehler PS. The intersect of gastrointestinal symptoms and malnutrition associated with anorexia nervosa and avoidant/restrictive food intake disorder: functional or pathophysiologic?—A systematic review. Int J Eat Disord. 2021;54(6):1019–54.
9. Katzman DK, Guimond T, Spettigue W, Agostino H, Couturier J, Norris ML. Classification of children and adolescents with Avoidant/Restrictive food intake disorder. Pediatrics. 2022;150:e2022057494.
10. Tanner AB, Richmond TK. Assessing growth in children and adolescents with Avoidant/Restrictive food intake disorder. J Eat Disord. 2024;12:82.
11. Goddard E, Ashkan K, Farrimond S, Bunnage M, Treasure J. Right frontal lobe glioma presenting as anorexia nervosa: further evidence implicating dorsal anterior cingulate as an area of dysfunction. Int J Eat Disord. 2013;46(2):189–92.
12. Strick PL, Dum RP, Fiez JA. Cerebellum and nonmotor function. Annu Rev Neurosci. 2009;32:413–34.
13. Pagès M, Beccaria K, Boddaert N, Saffroy R, Besnard A, Castel D, et al. Co-occurrence of histone H3 K27M and BRAF V600E mutations in paediatric midline grade I ganglioglioma. Brain Pathol. 2018;28(1):103–11.
14. Goodwin AF, Oberoi S, Landan M, Charles C, Groth J, Martinez A, et al. Craniofacial and dental development in cardio-facio-cutaneous syndrome: the importance of Ras signaling homeostasis. Clin Genet. 2013;83(6):539–44.
15. Iovino M, Messana T, Lisco G, Mariano F, Giagulli VA, Guastamacchia E, et al. Neuroendocrine modulation of Food Intake and Eating Behavior. Endocr Metab Immune Disord Drug Targets. 2022;22(13):1252–62.
16. Gaudio S, Olivo G, Beomonte Zobel B, Schiöth HB. Altered cerebellar–insular–parietal–cingular subnetwork in adolescents in the earliest stages of anorexia nervosa: a network–based statistic analysis. Transl Psychiatry. 2018;8:127.
17. La Spina M, Caruso M, Gulizia C, Comella M, Soma R, Meli M, et al. Diencephalic Syndrome: Misleading Clinical Onset of Low-Grade Glioma. Curr Oncol. 2023;30(9):8401–10.
18. Dejkhamron P, Likasitwattankul S, Unachak K. Diencephalic Syndrome: A Rare and Easily Overlooked Cause of Failure to Thrive. 2004;87.
19. Miljic D, Pekic S, Popovic V et al. Empty sella. In: Feingold KR, Anawalt B, Blackman MR, Boyce A, Chrousos G, Corpas E,., éditeurs. Endotext. South Dartmouth (MA): MDText.com, Inc.; 2000. Available on: http://www.ncbi.nlm.nih.gov/books/NBK532084
20. Rani Pr, Maheshwari R, Reddy TSK. Prasad Nr, Reddy Pa. Study of prevalence of endocrine abnormalities in primary empty sella. Indian J Endocrinol Metab. 2013;17(7):125.

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