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Chronic Affective Blunting and Acute Exacerbation After Repeat Transsphenoidal Resection of a Pituitary Epidermoid Cyst

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Abstract

Intracranial epidermoid cysts are uncommon, benign, slow-growing tumors that typically present with neurologic symptoms secondary to mass effect. Surgical resection remains the primary treatment modality, but it may contribute to dysregulation of the hypothalamic-pituitary-adrenal axis, with downstream effects on cognition, mood, and behavior. These effects remain poorly understood despite well-described acute postoperative complications. We present a 52-year-old man with no prior psychiatric history who underwent two transsphenoidal resections for a recurrent sellar/suprasellar epidermoid cyst that, following his first resection, subsequently led to emotional blunting, apathy, and diminished emotional reactivity, progressively worsening and later requiring psychiatric consultation during hospitalization for an unrelated critical illness, where evaluation demonstrated a persistent flattened affect without evidence of major depressive disorder, psychosis, or suicidality. His presentation was more consistent with structural and neuroendocrine effects related to prior neurosurgical intervention than a primary psychiatric disorder. Tumors involving the infundibulotuberal region have been associated with impaired emotional regulation and personality change independent of hormonal status, and persistent cortisol deficiency has been linked to impaired amygdala-prefrontal connectivity central to emotional processing. No standardized screening protocols currently exist for pituitary-related neuropsychiatric effects. This case highlights the underrecognized long-term neuropsychiatric burden of pituitary epidermoid cyst resection. It supports the value of standardized postoperative neuropsychiatric screening to improve multidisciplinary management and outcomes in this population.

Categories: Psychiatry, Internal Medicine, Neurosurgery
Keywords: affective blunting, apathy, hypothalamic-pituitary-adrenal axis, neuropsychiatric complications, pituitary epidermoid cyst, postoperative screening, psychiatric neurosurgery, rare brain tumors, secondary adrenal insufficiency, transsphenoidal resection

Introduction

Intracranial epidermoid cysts are uncommon tumors that account for roughly 1% of all intracranial tumors [1]. They are benign, slow-growing masses that present with neurologic symptoms due to mass effect [1]. Affective blunting refers to a diminished range and intensity of emotional expression, while apathy reflects a broader reduction in motivation and goal-directed behavior. Both can resemble depressive symptoms such as loss of interest and psychomotor slowing but are distinguished from major depressive disorder by the absence of pervasive low mood, hopelessness, or anhedonia, and are thought to arise from disruption of hypothalamic-limbic circuitry rather than primary mood pathology [2,3]. The mainstay of management for intracranial epidermoid cysts is surgical resection, with the goal of gross total resection whenever safely feasible [4,5]. Lesions in the sellar/suprasellar region are typically approached endoscopically via an endonasal transsphenoidal route. Transsphenoidal surgery has been associated with dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis with downstream effects on cognition, mood, and behavior [6]. Although acute postoperative complications are well described in the literature, the chronic and long-term sequelae, particularly psychiatric outcomes, remain poorly understood and inadequately addressed in current literature. Prior reports describe persistent affective and cognitive disturbances following resection of other sellar/suprasellar lesions, including pituitary adenomas and craniopharyngiomas [2,7], but data specific to epidermoid cysts and the effects of repeat transsphenoidal surgery remain limited.

We present the case of a patient who underwent two transsphenoidal resections for a sellar/suprasellar epidermoid cyst and, following his first resection, subsequently developed flat affect that progressively worsened, with an acute exacerbation occurring three years after the initial intervention.

Case Presentation

A 52-year-old male with no prior psychiatric history presented with a history of well-known neurosurgical intervention, including two pituitary resections (Table *1*). Per outside records, the patient initially presented

to an outside neurosurgical facility with a complaint of headache. Magnetic resonance imaging (MRI) of the brain at the time showed a sellar/suprasellar mass with suspected mass effect on the optic chiasm. Further findings showed a ruptured epidermoid cyst, a diagnosis confirmed on histopathology. The patient underwent endoscopic resection of the epidermoid cyst in October 2022, followed by ventriculoperitoneal (VP) shunt placement later that month. After the surgery, the patient was put on hormone replacement therapy consisting of desmopressin, hydrocortisone, and levothyroxine, along with levetiracetam.

Date	Event
Oct 2022	Headache presentation at outside facility; MRI shows sellar/suprasellar mass with optic chiasm involvement; endoscopic resection of ruptured epidermoid cyst (histopathology-confirmed)
Oct 2022 (later that month)	Ventriculoperitoneal (VP) shunt placed at outside facility; catheter tract later confirmed on 2026 head CT, with no ventricular enlargement to suggest malfunction
Shortly after first surgery	Wife notes patient appears “cold,” unmotivated; no psychiatric evaluation sought
~2023	1-year follow-up; MRI shows sellar/suprasellar lesion (differential included Rathke cleft cyst, pituitary adenoma, craniopharyngioma, or other mass); index admission labs obtained; no intervention needed; the patient was safely discharged
2024	Repeat pituitary resection by outside neurosurgeon; morning cortisol 1.2 mcg/dL (below the reference range)
2025	Morning cortisol 0.5 mcg/dL (below the reference range)
April 2026	Admitted for aortic occlusion/critical limb ischemia; embolectomy, fasciotomy; course complicated by bilateral PE, AKI, rhabdomyolysis; above-knee amputation; morning cortisol 0.5 mcg/dL (below the reference range); psychiatry consulted for acute-on-chronic affective blunting
Discharge/follow-up	Hydrocortisone tapered to baseline; psychiatrically stable; clinically stable at outpatient follow-up

TABLE 1: Clinical timeline

VP: ventriculoperitoneal; PE: pulmonary embolism; AKI: acute kidney injury; CT: computed tomography.

A year later, the patient had a follow-up visit to our facility. MRI demonstrated a lobulated, nonenhancing T1- and T2-hyperintense sellar/suprasellar lesion with mild mass effect (Figure *1*). The differential included a Rathke cleft cyst, complex cystic pituitary adenoma, craniopharyngioma, or other mass, though the confirmed diagnosis, based on prior histopathology and clinical correlation, remained a recurrent epidermoid cyst.

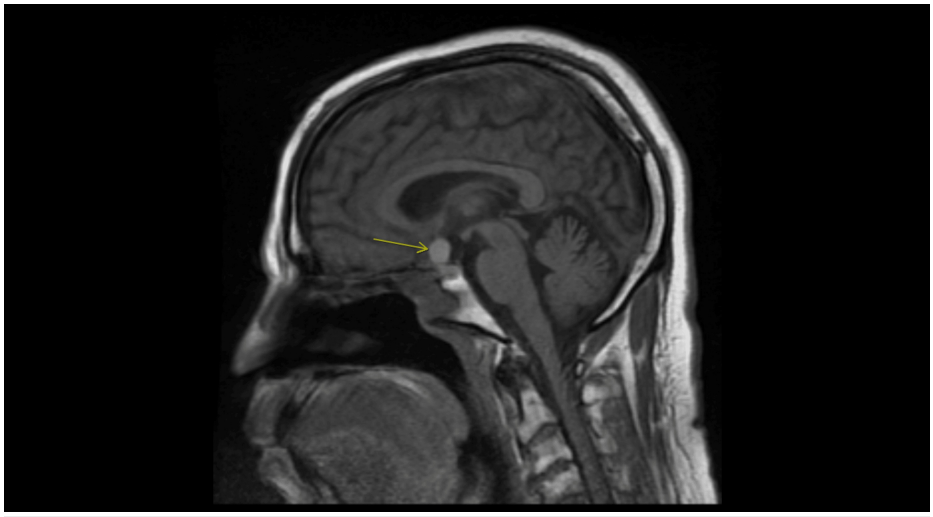


FIGURE 1: Sellar/suprasellar lesion on MRI

Sagittal T1-weighted MRI of the brain obtained November 2023, without and with intravenous gadolinium, demonstrating a lobulated, nonenhancing T1- and T2-hyperintense lesion in the sella and suprasellar region, measuring approximately 1.5 × 1.2 × 2 cm, with mild surrounding mass effect (arrow). No restricted diffusion was identified. The differential diagnosis included Rathke cleft cyst, complex cystic pituitary adenoma, craniopharyngioma, or other mass; the confirmed diagnosis, based on prior histopathology and clinical correlation, remained a recurrent epidermoid cyst.

Labs on index admission (Tables *2*, *3*) were notable for low adrenocorticotropic hormone (ACTH) (2.0 pg/mL), with normal prolactin (7.4 ng/mL) and thyroid-stimulating hormone (TSH) (3.501 mcIU/mL), consistent with pituitary dysfunction and secondary adrenal insufficiency. The basic metabolic panel was unremarkable. No neurosurgical intervention was needed at the time, and he was safely discharged. He later underwent repeat pituitary resection in 2024 by his previous outside neurosurgeon. After the operation, the patient had persistently low morning cortisol levels (1.2 mcg/dL in 2024, 0.5 mcg/dL in 2025, and 0.5 mcg/dL in 2026), consistent with ongoing secondary adrenal insufficiency (Table *4*).

	Value	Reference Range
Sodium	137 mEq/L	135 - 145 mEq/L
Potassium	4.1 mEq/L	3.5 - 5.0 mEq/L
Chloride	106 mEq/L	98 - 107 mEq/L
CO2	27 mEq/L	21–31 mEq/L
BUN	18 mg/dL	7 - 23 mg/dL
Creatinine	1.04 mg/dL	0.60 - 1.30 mg/dL
Glucose	104 mg/dL	70 - 110 mg/dL

TABLE 2: Index admission serum chemistry

mEq/L: milliequivalents per liter; mg/dL: milligrams per deciliter; CO2: carbon dioxide; BUN: blood urea nitrogen.

	Value	Reference Range
TSH	3.501 mcIU/mL	0.450 - 5.330 mcIU/mL
ACTH	2.0 pg/mL*	7.2 - 63.3 pg/mL
Prolactin	7.4 ng/mL	Male: <18 ng/mL

TABLE 3: Index admission endocrine labs

TSH: thyroid-stimulating hormone; ACTH: adrenocorticotropic hormone; mcIU/mL: micro-international units per milliliter; pg/mL: picograms per milliliter; ng/mL: nanograms per milliliter. *Below the reference range. ACTH suppression is expected in the setting of exogenous hydrocortisone therapy and should be interpreted in this context.

Date	Value	Reference Range
2024	1.2 mcg/dL*	AM: 4.3 - 22.4 mcg/dL
2025	0.5 mcg/dL*	AM: 4.3 - 22.4 mcg/dL
2026	0.5 mcg/dL*	AM: 4.3 - 22.4 mcg/dL

TABLE 4: Serial cortisol levels

mcg/dL: micrograms per deciliter. *Below the reference range. All values were drawn in the morning.

In April 2026, he was admitted with an abdominal aortic occlusion and critical limb ischemia requiring embolectomy and fasciotomy, with his course complicated by bilateral pulmonary embolism, acute kidney injury, and rhabdomyolysis. The patient ultimately underwent above-knee amputation (AKA). Head CT during this admission showed no evidence of acute hemorrhage, midline shift, or large-vessel infarct. The sellar/suprasellar mass was visualized compressing the optic chiasm and hypothalamic region, with the VP shunt catheter tract intact and no evidence of ventricular enlargement. Shortly after, the psychiatry team was consulted for acute worsening of his chronic blunting of affect and poor emotional reactivity, symptoms that had existed prior to the amputation and became more pronounced afterward. The patient stated that after his first surgery of resection of the epidermoid cyst, his wife said he was “cold” and unmotivated. His wife told him to seek medical attention, but he never sought any psychiatric evaluation. That affective state persisted for several months without resolution, continuing at a baseline level in the years that followed, placing significant strain on his marriage before its acute exacerbation in 2026.

On the current evaluation, he was alert and oriented to person, with good insight into the reason for hospitalization. He was noted to have flattened affect, monotone speech, and reduced output, frequently drifting toward sleep and endorsing feeling “numb.” He denied depression, hopelessness, helplessness, and any history or current ideation of suicidality or homicidality, ruling out major depressive disorder on clinical interview. He also denied perceptual disturbances, paranoia, internal preoccupation, and auditory or visual hallucinations. A formal delirium assessment (DSM-IV-based criteria) was negative, with no change from his baseline mental status, intact attention, and no disorganized thought process, making delirium an unlikely contributor to his presentation. Urine toxicology was negative for amphetamines, barbiturates, benzodiazepines, cocaine, opiates, phencyclidine, and tetrahydrocannabinol. During the hospitalization, he received intermittent morphine for severe pain; no other sedating or psychoactive medications were administered beyond his home regimen.

Further endocrine evaluation during this admission revealed multi-axis pituitary dysfunction (Table *5*). Free thyroxine (T4) was low at 0.45 ng/dL with an inappropriately normal TSH (2.285 mcIU/mL), consistent with central hypothyroidism. Gonadal axis evaluation showed severe hypogonadism, with a low total testosterone (<10 ng/dL by immunoassay; 1 ng/dL by LC/MS) and low free testosterone (0.1 pg/mL). Complete blood count and chemistry showed postoperative anemia and low creatinine, with electrolytes and renal function otherwise unremarkable (Tables *6*, *7*). His home hydrocortisone regimen totaled 15 mg/day (10 mg each morning, 5 mg each evening), corresponding to approximately 0.18 mg/kg/day based on his dosing weight of 85 kg.

	Value	Reference Range
Morning cortisol	0.5 mcg/dL*	AM: 4.3-22.4 mcg/dL
Free T4	0.45 ng/dL*	0.61-1.12 ng/dL
TSH	2.285 mcIU/mL	0.450-5.330 mcIU/mL
Testosterone, Total (immunoassay)	<10 ng/dL*	250-827 ng/dL
Testosterone, Total (LC/MS)	1 ng/dL*	250-1100 ng/dL
Testosterone, Free	0.1 pg/mL*	35.0-155.0 pg/mL

TABLE 5: April 2026 admission endocrine labs

T4: thyroxine; TSH: thyroid-stimulating hormone; LC/MS: liquid chromatography-mass spectrometry. *Below the reference range.

	Value	Reference Range
WBC	10.4 x10³/mm³	4.5 - 11.0 x10³/mm³
RBC	2.84 x10⁶/mm³*	4.33 - 5.83 x10⁶/mm³
Hemoglobin	8.2 g/dL*	13.5 - 17.5 g/dL
Hematocrit	27.3%*	41.0 - 53.0%
MCV	96.1 fL	80.0 - 100.0 fL
MCH	28.9 pg	26.0 - 32.0 pg
MCHC	30.0 g/dL*	31.0 - 37.0 g/dL
RDW	18.3%†	0.5 - 16.5%
RDW-SD	61.4%	40% - 70%
Platelets	425 x10³/mm³	140 - 440 x10³/mm³
MPV	9.3 fL	7.4 - 10.4 fL

TABLE 6: April 2026 admission complete blood count

WBC: white blood cell count; RBC: red blood cell count; MCV: mean corpuscular volume; MCH: mean corpuscular hemoglobin; MCHC: mean corpuscular hemoglobin concentration; RDW: red cell distribution width; RDW-SD: red cell distribution width standard deviation; MPV: mean platelet volume; x10³/mm³: thousand cells per cubic millimeter; x10⁶/mm³: million cells per cubic millimeter; g/dL: grams per deciliter; fL: femtoliters; pg: picograms. *Below the reference range. † Above the reference range.

	Value	Reference Range
Sodium	143 mEq/L	135 - 145 mEq/L
Potassium	4.6 mEq/L	3.5 - 5.0 mEq/L
Chloride	107 mEq/L	98 - 107 mEq/L
CO2	30 mEq/L	21 - 31 mEq/L
Glucose	90 mg/dL	70 - 110 mg/dL
BUN	13 mg/dL	7 - 23 mg/dL
Creatinine	0.51 mg/dL*	0.60 - 1.30 mg/dL

TABLE 7: April 2026 admission serum chemistry

mEq/L: milliequivalents per liter; mg/dL: milligrams per deciliter; CO2: carbon dioxide; BUN: blood urea nitrogen. *Below the reference range.

Given his extensive neurosurgical history, his presentation is more consistent with chronic brain-related changes than with a primarily psychiatric etiology, though causality cannot be established from a single case report. The physiologic burden of critical illness and major surgery further taxed his already compromised HPA axis, likely contributing to acute worsening of his affective symptoms. His management required a multidisciplinary approach with collaboration from psychiatry, neurology, surgery, and endocrinology.

On discharge, the patient was continued on his home hormone replacement regimen with hydrocortisone tapered back to his baseline dose, with plans for outpatient follow-up. From a psychiatric standpoint, he was deemed stable for discharge, without acute safety concerns, and did not require ongoing inpatient psychiatric intervention. At the outpatient follow-up, the patient was reported to be clinically stable, without acute psychiatric concerns.

Discussion

Epidermoid cysts are rare, benign, slow-growing tumors that cause insidious symptom progression as they expand, and in this patient, they presented initially with a headache. Despite their benign nature, intracranial epidermoid cysts pose a significant neurosurgical challenge due to adherence to critical neurovascular structures and cranial nerves [5]. Surgical resection is the standard of care, and thus, immediate and long-term management is critical given HPA axis dysregulation. Disruption of the HPA axis, whether hyperactive or hypoactive, has been shown to affect an individual's psychological state. This association has been recognized since early studies in the mid-20th century, in which dysregulation of cortisol was observed in patients with depressive disorders [7]. With limited research into the effects of repeated pituitary/brain surgery independent of hormonal dysregulation and its correlation to affective blunting, the mechanism by which repetitive trauma to the brain contributes to affective blunting remains undefined. However, an important factor to consider when evaluating the flat affect is the mass's anatomical location. Craniopharyngioma data provide the strongest model: tumors involving the infundibulotuberal region cause impaired emotional expression/control in 42% of cases and behavioral/personality changes in 48.5% of cases; specifically, these deficits map to the hypothalamic structure rather than hormonal status [2]. Emotional expression and personality change are distinct but related processes mediated by shared hypothalamic-limbic circuitry. The hypothalamus receives bidirectional limbic input through the stria terminalis, ventral amygdalofugal pathway, and medial forebrain bundle. Disruption of top-down regulation may lead to emotional dysregulation, manifesting as mood lability, emotional outbursts, or reduced frustration tolerance. In contrast, impaired bottom-up signaling may contribute to persistent personality changes characterized by apathy and diminished goal-directed behavior [8]. A lesion involving the infundibulotuberal region may disrupt both pathways simultaneously, resulting in overlapping emotional and personality disturbances. Craniopharyngiomas, however, more frequently involve the hypothalamus directly and behave differently from epidermoid cysts, which more typically arise within the sellar/suprasellar space without primary hypothalamic origin. This patient's imaging did not show direct hypothalamic invasion, so this comparison is offered as a mechanistic analogy rather than a direct equivalence.

Apathy syndrome is a consequence of hypothalamic-pituitary axis disruption that must be differentiated from depression. The hypothalamic-pituitary axis may affect behavioral control exerted by the prefrontal cortex through its deep connections with limbic structures, causing mood and personality alterations; in more severe cases, a distinct apathy syndrome develops that is neuroanatomically mediated rather than hormonally driven [3]. Maintaining adequate cortisol levels is crucial and a well-established modifiable contributor to psychiatric symptoms post-resection. In a randomized crossover study with 47 patients with

secondary adrenal insufficiency, there was a direct dose-dependent psychiatric effect on patients not reaching appropriate cortisol levels [9]. Patients with higher hydrocortisone doses (0.4-0.6 mg/kg/day) exhibited fewer depressive symptoms, less mental fatigue, and greater motivation than those receiving a lower dose (0.2-0.3 mg/kg/day) [9]. Our patient's home hydrocortisone dose of 15 mg/day (approximately 0.18 mg/kg/day at a body weight of 85 kg) fell below even the lower-dose arm of this trial, consistent with relative under-replacement as a contributor to his affective symptoms. However, during this same trial, there was found to be no difference in cognitive function/performance when comparing the two doses [10]. Assessing affective blunting in patients with known structural brain changes requires a multi-level approach.

Overlapping features such as apathy, deficits in emotional recognition, and reduced emotional reactivity complicate the assessment of affective blunting in brain-injured patients. Several tools exist to help measure and distinguish these features. Tests such as the Scale for Emotional Blunting (SEB) can be administered by a caregiver or clinician, and it is typically used to capture pre-morbid to current changes in emotional blunting [11]. Furthermore, another tool to utilize would be the Ekman faces/eyes test, which would assist in mapping specific lesion sites and perceptual level deficits [12]. An important distinction to keep in mind would be to differentiate between apathy and depression, as they both share overlap in symptoms such as loss of interest and psychomotor dysfunction. The relationship between chronic secondary adrenal insufficiency and emotional dysregulation/blunting occurs through multiple converging neurobiological mechanisms, with the relationship being bidirectional. One example is a disruption in the amygdala-prefrontal cortex coupling, with cortisol playing a direct role in modulating the connections between the two brain structures. Higher endogenous cortisol levels are associated with improved inhibitory function connectivity, which is shown to be an important circuit for emotional regulation [13]. In cortisol-deficient states, this inhibitory coupling weakens, leaving amygdala reactivity poorly regulated and contributing to emotional blunting [13]. Contrarily, excess glucocorticoids have been associated with psychiatric disturbances ranging from mood symptoms to frank psychosis, further supporting cortisol's central role in modulating this circuitry bidirectionally [14]. Cortisol plays an important function in the cortisol awakening response, in which a deficiency can cause impaired accuracy on emotional face matching tasks and alter the amygdala-prefrontal cortex connectivity [15]. Furthermore, it's described that the morning cortisol surge sets a neuroendocrine tone to prepare for emotional processing for the upcoming day [15].

Currently, no standardized, universally adopted protocol exists for screening for pituitary-related neuropsychiatric symptoms, but the evidence strongly supports integrating systematic neuropsychiatric screening into routine pituitary care. Furthermore, several evidence-based frameworks can guide protocol development. Some proposed screening protocols to implement in this patient population could include the following rapid screening tests: NCCN (National Comprehensive Cancer Network) Distress Thermometer, PHQ-2 (Patient Health Questionnaire-2), and single-item fatigue test [16,17]. The NCCN Distress Thermometer, though originally developed for oncology populations, has been applied more broadly as a rapid distress screen. Apathy-specific instruments such as the Apathy Evaluation Scale may better capture the blunted motivation and reduced emotional reactivity described in our patient than depression-focused tools like the PHQ-2. Indications of a positive test should lead to a targeted assessment of the underlying neuropsychiatric symptoms.

Given the complexity of this presentation, several alternative diagnoses were systematically evaluated. Hypoactive delirium was evaluated and excluded using formal DSM-based criteria, with the patient alert and appropriately interactive on exam. Medication effects were reviewed: levetiracetam, a known contributor to behavioral and mood changes, and as-needed morphine were the only psychoactive agents in use, with no other sedating medications on board. Ventriculoperitoneal shunt dysfunction was considered unlikely, as a 2026 head CT confirmed an intact catheter tract without ventricular enlargement. Cerebral ischemia was excluded by the same head CT, which showed no acute infarct or hemorrhage. Further evaluation of additional pituitary axes revealed central hypothyroidism (low free T4) and hypogonadism (low total and free testosterone); rather than competing with the proposed mechanism, these findings support a picture of panhypopituitarism converging on the same hypothalamic-pituitary structural injury (Table *5*). Growth hormone status (IGF-1) was not obtained. Adjustment and grief following the patient's recent AKA likely contributed to the acute-on-chronic worsening of his affective symptoms. However, his flattened affect and reduced emotional reactivity were present for months before this event. Major depressive disorder was formally ruled out on clinical interview, with the patient denying depressive cognitions, hopelessness, and anhedonia throughout.

Several limitations exist within this report. It reflects a single case, limiting generalizability. No standardized instruments (e.g., an apathy-specific scale) were administered at the time of evaluation, and no premorbid psychiatric baseline was available beyond collateral history from the patient's wife. Multiple confounders, such as critical illness, major amputation, and polypharmacy, occurred concurrently with the psychiatric evaluation and cannot be fully separated from the proposed neuroendocrine mechanism.

Conclusions

Pituitary epidermoid cyst resection with repeat transsphenoidal surgery may carry significant long-term

neuropsychiatric consequences that remain underrecognized in current literature. This case raises the possibility that converging mechanisms, including structural disruption of hypothalamic-limbic circuitry and chronic secondary adrenal insufficiency, contribute to persistent affective blunting following repeated neurosurgical interventions. The integration of brief screening tools into routine postoperative care is supported by existing evidence, and this case supports considering routine neuropsychiatric screening for patients undergoing repeat transsphenoidal resection of pituitary lesions.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Deng Aguto, Shaban Gheith

Acquisition, analysis, or interpretation of data: Deng Aguto, Shaban Gheith, Angelo Sica

Drafting of the manuscript: Deng Aguto, Shaban Gheith

Critical review of the manuscript for important intellectual content: Deng Aguto, Shaban Gheith, Angelo Sica

Supervision: Angelo Sica

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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