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Brief Report

Put the Mission in Admission: Increasing In-State Matriculants Through a Mission-Aligned Admissions Framework

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ABSTRACT

Introduction. The University of Kansas School of Medicine (KUSOM) seeks to improve health care in Kansas by matriculating students likely to meet the state's needs. A decline in in-state applicants and increased national scrutiny of admissions policies prompted a review of the admissions process to ensure alignment with the school's mission. Authors of this study evaluated the implementation and outcomes of a mission-driven, holistic admissions approach.

Methods. For the 2024 admissions cycle, KUSOM adopted a structured process emphasizing mission alignment across four stages: (1) Pre-screening – prioritizing applicants with strong Kansas ties; (2) Screening – enhancing secondary application questions to assess mission-relevant attributes; (3) Interviewing – using structured evaluations to assess mission-driven qualities; and (4) Selection – integrating mission alignment into committee deliberations. Admissions data from 2021 to 2024 were analyzed to assess impact.

Results. The updated process resulted in notable improvements. Kansas resident matriculants increased from 74% in 2023 to 85% in 2024. Interview invitations and admission offers to Kansas residents also rose. Matriculants with Kansas ties increased from 11 in 2021 to 19 in 2024.

Conclusions. A mission-driven, holistic admissions process successfully increased the number of students likely to practice in Kansas. These findings support the effectiveness of structured admissions strategies in meeting state health care workforce needs. Further research is needed to evaluate the long-term impact on residency and practice locations.

INTRODUCTION

The mission of The University of Kansas School of Medicine (KUSOM) is to "improve lives and communities in Kansas and beyond through innovation in education, research, and health care."[1] As the state's medical school, KUSOM gives preference to in-state applicants. The Admissions Committee emphasizes recruiting students who are likely to practice medicine in Kansas, supported by longstanding research indicating that students often return to their home communities to practice after completing their medical education.[2,3]

Recent studies further highlight how the location of medical school influences both residency placement[4,5] and eventual practice location.[6] According to the Association of American Medical Colleges (AAMC), 58.6% of individuals who completed residency training between 2014 and 2023 now practice in the same state where they trained.[7] In Kansas, that figure is 55.2%.[8]

Following the 2021 application cycle, KUSOM observed a decline in applications and matriculations from Kansas students. In addition, the U.S. Supreme Court's 2023 decision on affirmative action in admissions[9] prompted increased scrutiny of admissions practices nationwide, prompting a review to ensure legal compliance and alignment with KUSOM's mission. After a thorough evaluation of current practices, and in response to concerns shared by KUSOM senior leadership regarding the steady decline in Kansas student enrollment, a decision was made to restructure admissions procedures.

The 2024 application cycle now features a mission-driven, holistic admissions process designed to better support the school's goals and values.

METHODS

In spring and summer 2023, the KUSOM Office of Admissions reviewed each stage of the admissions process to better identify mission-aligned candidates. Guided by the AAMC's definition of holistic admissions, which considers applicants' experiences, attributes, and academic metrics alongside their potential contributions to learning, practice, and teaching,[10] KUSOM moved from a weighted model to a more balanced and systematic approach.

Once restructuring goals were defined, KUSOM tailored its holistic model to meet the specific needs of Kansas communities. The admissions process was evaluated and updated in four key phases to improve alignment with the school's mission:

1. **Pre-screening Phase:** Establishing criteria to identify mission-aligned candidates.
2. **Screening Phase:** Conducting a mission-aligned review of out-of-state applicants.
3. **Interviewing Phase:** Evaluating candidates on mission-driven attributes.
4. **Selection Phase:** Centering committee discussions around mission alignment.

Pre-screening Phase. The admissions team refined the AAMC's pre-medical competencies to emphasize qualities aligned with KUSOM's mission. A key component was prioritizing applicants with a strong connection to Kansas, defined as a "Kansas tie." Non-resident applicants met this designation if they fulfilled at least one of the following:

- Currently reside and work in Kansas.
- Have a parent residing in Kansas.
- Graduated from a Kansas high school or four-year college.
- Have a parent who is a KUSOM graduate or faculty member.

The "Kansas tie" designation, long recognized by the Admissions Committee, was approved for automatic interview invitations to streamline screening and ensure mission-aligned applicants familiar with the state were prioritized. As shown in Table 1, Kansas tie applicants are more likely to matriculate than non-residents without such ties.

Screening Phase. In 2024, new questions were added to the secondary application to assess non-residents' alignment with the

KUSOM mission. Reviewers were trained to identify mission-relevant experiences and connections among all applicants. Prior to 2024, the secondary application only asked non-residents to explain their interest in KUSOM. The updated questions assess both motivation and mission alignment.

Interviewing Phase. Applicants who advanced to interviews were evaluated using structured assessments that included mission alignment alongside traditional attributes such as communication skills and professionalism. Interviewers received sample mission-focused questions and an updated evaluation form. Previously, interviews were broader in focus and lacked a dedicated section for assessing mission-driven experiences.

Selection Phase. The final selection process was redesigned to consistently prioritize mission alignment. Presentations by Admissions Committee members now begin with a summary of each applicant's mission-relevant attributes. Interview feedback and supplemental application responses are accessible to all members. Presenters also highlight the applicant's Kansas tie, time spent in the state, and the relevance of their experiences. While a structured presentation format existed before 2024, it did not explicitly include mission alignment, which often was addressed informally during general comments.

Table 1. The University of Kansas School of Medicine entering class profile, 2021-2024.

Entering Class	2021	2022	2023	2024
Total Applications	**3,275**	**2,554**	**3,250**	**4,742**
Kansas residents	588	472	444	427
Non-residents with "Kansas tie"	126	102	125	154
Non-residents, no "Kansas tie"	2,572	1,967	2,651	4,123
No state	19	13	30	38
Applicants Interviewed	**619**	**546**	**528**	**473**
Kansas residents	406	350	311	296
Non-residents with "Kansas tie"	84	69	85	73
Non-residents, no "Kansas tie"	128	127	131	103
No state	1	0	1	1
Total Admission Offers	**260**	**274**	**277**	**279**
Kansas residents	203	189	187	202
Non-residents with "Kansas tie"	20	34	27	20
Non-residents, no "Kansas tie"	37	51	63	57
No State	0	0	0	0
Matriculants	**211**	**211**	**211**	**211**
Kansas residents	182	164	157	180
Kansas percent	86%	78%	74%	85%
Non-residents with "Kansas tie"	11	24	22	19
Non-residents, no "Kansas tie"	18	23	32	12
No state	0	0	0	0
Kansas rural	43	38	32	37
Kansas metro	18	7	9	6
Kansas urban	121	119	116	134

RESULTS

Implementing a mission-driven admissions process led to measurable improvements in the composition of the entering class, particularly in increasing Kansas representation, as shown in Table 1. A descriptive time-series trend analysis of class profiles from 2021 to 2024 revealed the following:

- Interview offers to Kansas residents increased from 59% in 2023 to 63% in 2024.
- Admission offers to Kansas residents rose from 68% in 2023 to 72% in 2024.
- Kansas resident matriculation reached 85% in 2024, up from a two-year low of 74% in 2023 and 78% in 2022.
- Non-residents with significant Kansas ties increased from 11 in 2021 to 19 in 2024.
- Rural representation remained steady, with 37 rural Kansans in the 2024 class compared to 32 in 2023 and 43 in 2021.
- Non-residents without Kansas ties declined from 32 in 2023 to 12 in 2024, reflecting stronger alignment with the school's mission.

Although there is no formal requirement for a specific number of in-state matriculants, a class composed of at least 80% Kansas residents is preferred. These data trends underscore the effectiveness of aligning admissions practices with institutional priorities.

DISCUSSION

KUSOM successfully increased the number of mission-aligned matriculants and addressed short-term enrollment challenges. Implementing a mission-driven admissions framework demonstrates how structured, intentional changes can influence class composition, particularly by increasing in-state representation, and reinforce the school's commitment to serving Kansas communities.

The initial increase in Kansas residents and applicants with Kansas ties is encouraging; however, further research is needed to assess the long-term impact of these changes. Future studies should examine whether the rise in Kansas matriculants influences residency placement in the state, specialty selection, and eventual practice location. Evaluating whether mission-aligned admissions policies contribute to greater physician retention in Kansas, especially in rural and underserved areas, will be critical.

State medical schools can adopt similar mission-driven admissions strategies to strengthen in-state enrollment. KUSOM's experience illustrates how aligning admissions practices with institutional goals can help address broader health care workforce needs. These early successes should encourage medical school leaders, policymakers, and educators to consider and implement comparable strategies.

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Case Report

Steroid-Induced Mania in a 12-Year-Old Adolescent: A Case Report

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INTRODUCTION

Corticosteroids, known for their potent anti-inflammatory and immunosuppressive effects, are essential in treating a wide range of conditions, from autoimmune disorders to allergic reactions. Systemic corticosteroids, such as oral prednisolone and prednisone, commonly are used in pediatric respiratory illnesses.[1] Despite their therapeutic benefits, these medications have been associated with neuropsychiatric side effects in 2% to 60% of cases.[2] In a comprehensive review by Kenna and colleagues,[3] mania or hypomania was the most common presentation, observed in 54.5% (30/55) of adult cases. However, these complications remain underrecognized and often misdiagnosed in children.

Potential risk factors for steroid-induced neuropsychiatric symptoms, which are explored in this case, include recent viral infections (e.g., COVID-19), metabolic disturbances such as hypoalbuminemia, concurrent use of CYP3A4 inhibitors, and prior high-dose corticosteroid exposure, which may create a sensitization effect.

This case report describes steroid-induced mania in a 12-year-old male with a history of corticosteroid use and recent COVID-19 infection, illustrating the unique diagnostic and therapeutic challenges in pediatric populations. Alternative diagnoses, including pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections (PANDAS) and delirium, are considered. The case emphasizes the importance of maintaining a high index of suspicion for neuropsychiatric side effects in children undergoing corticosteroid therapy.

CASE REPORT

A 12-year-old male presented to the hospital with acute behavioral changes three days after initiating a treatment regimen of amoxicillin and a five-day course of prednisolone (50 mg daily) for group A streptococcal pharyngitis. His past medical history included expressive language disorder, speech delay, and reactive airway disease requiring intermittent corticosteroid inhalers. He also had experienced previous viral infections and a mild SARS-CoV-2 (COVID-19) infection five months prior to presentation.

The patient exhibited symptoms consistent with a severe manic episode with psychotic features, including hypersexuality, disinhibition, psychomotor agitation, decreased need for sleep, paranoia, and irritability, which escalated to aggression toward his dentist. Psychotic features included persecutory delusions. His affect was labile, alternating between inappropriate euphoria ("giddy and giggly") and irritability, along with pressured speech and motor restlessness (fidgeting). These symptoms marked a stark deviation from his baseline behavior.

Suspecting an allergic reaction, the pediatrician discontinued prednisolone and prescribed diphenhydramine 25 mg every four-six hours, which the patient took for two days prior to hospitalization. However, his mental status deteriorated, with increasing confusion, agitation, excessive laughter, unusual vocalizations, and socially inappropriate behavior. Due to concern for PANDAS, his pediatrician recommended hospital admission.

On the medical floor, the psychiatry consultation team noted disorganized, tangential, and illogical thought processes, as well as paranoid delusions directed at both family and hospital staff. He was distractible, intrusive, disinhibited, and exhibited self-stimulatory behaviors. The clinical picture strongly suggested an acute manic episode with psychotic features, likely triggered by corticosteroid use. However, steroid-induced psychosis and delirium also were considered in the differential.

A full medical workup to evaluate for PANDAS included a complete blood count, metabolic panel, thyroid studies, and a comprehensive autoimmune panel (antinuclear antibody, antinuclear antibody, anti-double-stranded DNA antibody, anti-extractable nuclear antigen, complement levels, anti-neutrophil cytoplasmic antibodies, anti-cardiolipin, lupus anticoagulant, anti-N-methyl-D-aspartate receptor, and anti-α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor antibodies). Inflammatory markers, including erythrocyte sedimentation rate (reference range: 0-15 mm/hr) and C-reactive protein (reference range: <0.3-1.0 mg/dL), also were obtained. All results were unremarkable.

Neuroimaging with non-contrast head computed tomography was normal. Lumbar puncture was not performed. A rapid strep test was positive, confirming recent group A streptococcal infection. Anti-streptolysin O (ASO) titer measured four days after a 1 g ceftriaxone dose, and approximately 10 days post-infection, was 186 (reference range: 0-199). Based on clinical findings and laboratory results, PANDAS was considered unlikely.

Collateral history from the patient's parents and grandmother confirmed a dramatic change from his typical behavior. Premorbidly, he was described as respectful, quiet, and academically successful, with no history of psychiatric disorders, behavioral issues, learning difficulties, or substance use. He alternated living with his divorced parents weekly and had a longstanding close relationship with his grandmother. Developmentally, he had experienced mild speech delays requiring therapy until fifth grade but otherwise met all milestones. Birth history was notable for severe maternal hemorrhage due to placenta accreta requiring a five-day hospitalization.

Following psychiatric assessment, risperidone 0.25 mg orally twice daily was initiated; however, due to refusal, he was switched to intramuscular olanzapine 2.5 mg at bedtime. He also received a single dose of intramuscular penicillin to complete treatment for streptococcal pharyngitis. Despite treatment, the patient continued to exhibit

hypersexuality, disorganized behavior (e.g., attempting to drink from a remote control), and hallucination-like experiences (e.g., calling for deceased relatives), necessitating transfer to inpatient psychiatry.

On the adolescent psychiatry unit, the patient showed minimal engagement, poor insight, perseveration, anxiety, and restlessness. Risperidone 0.25 mg twice daily was resumed but led to minimal improvement. By Hospital Day 4, disinhibition and inappropriate behaviors worsened, requiring another intramuscular olanzapine dose. Risperidone was titrated to 0.25 mg in the morning and 0.5 mg at bedtime, which resulted in gradual symptom improvement and discharge on Hospital Day 9.

The patient had 12 outpatient follow-up visits over the course of one year (Appendix A). At the first visit, one-week post-discharge, hypersexuality, agitation, and impulsivity had resolved with medication adherence, although the patient had poor recall of the events surrounding hospitalization. Approximately six weeks later, his family tapered the medication without medical supervision, leading to symptom recurrence and an urgent follow-up visit. He presented with disorientation, distractibility, expansive affect, confabulation, and impaired insight. While hypersexuality did not recur, collateral sources reported belligerence, impulsivity, and behavior inconsistent with the patient's self-report. Risperidone was reinstated and titrated to 0.5 mg twice daily, leading to symptom resolution within two weeks.

In subsequent follow-ups, the patient remained stable. A cautious taper was attempted during winter break, reducing risperidone to 0.25 mg in the morning and 0.5 mg at bedtime. Another taper during spring break three months later was well tolerated. Over the next several months, two additional 0.25 mg tapers were completed, and risperidone was fully discontinued at the one-year mark. At his most recent visit, the patient remained stable off medication for one month.

DISCUSSION

Previously documented cases of steroid-induced mania in pediatric populations reveal a spectrum of presentations, ranging from mild symptoms resolving after steroid discontinuation to severe episodes requiring intensive psychiatric intervention (Appendix B). Several key patterns emerge from these reports: steroid-induced mania can occur in individuals without a personal or family history of psychiatric illness, and symptoms typically develop rapidly, often within the first week of treatment, regardless of the route of administration (inhaled, oral, or intravenous).[4-6] Our patient's progression from behavioral changes to frank mania with psychosis within three days of starting prednisolone aligns with this characteristic rapid onset, offering an important diagnostic clue.

Our patient's presentation illustrates several diagnostic pitfalls. Initial consideration of PANDAS was deemed unlikely for several reasons: the patient's age was outside the typical four - nine year range,[7] he lacked hallmark features such as tics or obsessive-compulsive behaviors, and, most notably, his symptoms emerged acutely within days of streptococcal infection, rather than the gradual onset (3 to 12 months) described in a large case-control study of 75,000 children.[8] Additionally, his ASO titer of 186 was not significantly elevated. According to diagnostic criteria, ASO titers should ideally be obtained at symptom onset (within two weeks) and repeated four - eight weeks later, with a fourfold rise considered supportive of a streptococcal trigger.[9] As Prato et al.[10] note, PANDAS typically is associated with marked elevations in ASO and anti-DNase B titers, along with inflammatory markers such as erythrocyte sedimentation rate and C-reactive protein (CRP), none of which were present in our patient. While a limitation of this case was the absence of repeat ASO testing, the normal one-time titers, absence of obsessive-compulsive disorder or tics, and rapid symptom onset collectively argued against a PANDAS diagnosis.

The diagnostic picture was further complicated by signs of possible superimposed delirium, including confusion and disorientation, potentially worsened by diphenhydramine's deliriogenic effects. In alignment with Kenna et al.,[3] who reported a mean resolution time of 9.6 days for steroid-induced delirium, our patient's confusional symptoms improved relatively quickly, while manic features persisted, supporting the presence of comorbid delirium and mania rather than either condition alone. This underscores the complexity of steroid-induced neuropsychiatric presentations.

Several risk factors likely contributed to our patient's psychiatric episode. His prescribed prednisolone dose (50 mg daily) exceeded the 40 mg threshold associated with increased risk for neuropsychiatric side effects.[11] Individual variability in steroid metabolism and distribution further may influence susceptibility, particularly in the presence of CYP3A4 inhibitors or hypoalbuminemia, which increase free corticosteroid availability (odds ratio 2.2).[12] While our patient had previously tolerated both inhaled and oral corticosteroids for respiratory illnesses, cumulative exposure may have contributed to vulnerability. Sullivan et al.[13] reported a 1.29-fold increase in the risk of new adverse effects with more than four corticosteroid prescriptions in a year.

The timing of our patient's psychiatric manifestations also suggests the role of preceding inflammatory insults. While he had previously tolerated corticosteroids, mania emerged only after sequential infections, a mild COVID-19 infection five months prior, followed by streptococcal pharyngitis. Emerging research indicates that COVID-19 may predispose children to psychiatric sequelae through elevation of pro-inflammatory cytokines such as interleukin-6, tumor necrosis factor-α, and CRP.[14,15] This inflammatory cascade may be particularly disruptive during periods of neurodevelopmental vulnerability in brain regions such as the amygdala, hippocampus, and prefrontal cortex.[16,17]

The glucocorticoid vulnerability hypothesis provides a potential mechanistic framework, positing that high densities of glucocorticoid receptors in limbic and prefrontal regions[18,19] render these areas susceptible to corticosteroid-induced changes. Chronic corticosteroid exposure has been associated with dendritic retraction in the hippocampus,[20] possibly extending the window of neuronal vulnerability. This may explain how prior inflammatory insults (e.g., viral and bacterial infections) and corticosteroid exposure culminated in a "perfect storm" for steroid-induced mania in our patient.

Understanding this pathophysiology informs therapeutic decision-making. While tapering corticosteroids is a standard first-line approach,

effective in 94% of cases in Lewis and Smith's series and half of those in Appenzeller's study,[3] our experience demonstrates that tapering alone may be insufficient. For steroid-induced mania specifically, Kenna et al.'s[3] systematic review highlights successful outcomes using antipsychotics and/or mood stabilizers, including haloperidol (with or without lithium), risperidone, quetiapine, olanzapine (alone or with valproate), carbamazepine, lithium, and lamotrigine plus clonazepam.

This is consistent with previously reported cases. Khan et al.[4] reported resolution with steroid discontinuation alone, while Couturier et al.[5] and Cassidy et al.[6] described patients requiring antipsychotics, mood stabilizers, and benzodiazepines. Notably, the patient who received inhaled corticosteroids alone had the shortest duration of symptoms (48 hours) and recovered with steroid discontinuation alone, likely due to lower systemic and central nervous system exposure.[4-6]

Our patient required prolonged treatment with risperidone, which may reflect the complexity of his case, including inflammatory priming from prior infections. Following a year of stabilization, he was successfully tapered off risperidone and remained symptom-free for one month without medication.

CONCLUSIONS

This case offers several important clinical takeaways:

1. Education: Patients and families should be thoroughly counseled about potential neuropsychiatric side effects of corticosteroids. Education should occur at treatment initiation, during therapy, and before any dose adjustments. In our case, lack of counseling led to symptom recurrence following unsupervised medication tapering.
2. Monitoring: Structured psychiatric monitoring protocols are essential, particularly during high-dose steroid treatment and dose changes. Follow-up should extend beyond corticosteroid discontinuation, as recovery may be delayed.
3. Risk Assessment: This case illustrates the need for a comprehensive risk assessment strategy. Our patient's episode was likely precipitated by a convergence of risk factors: recent viral and bacterial infections, high-dose steroid exposure, and anticholinergic use. Current monitoring guidelines for long-term corticosteroid use emphasize physical parameters, weight, glucose, blood pressure, lipids, and bone density, but do not adequately address neuropsychiatric monitoring.[21,22]

We propose the development of a standardized neuropsychiatric risk assessment tool to complement existing physical monitoring protocols. This tool should account for both non-modifiable (e.g., age, psychiatric history) and modifiable factors, such as concurrent medications (e.g., CYP3A4 inhibitors), albumin levels, sleep hygiene, psychosocial stressors, and substance use. Such a multidimensional approach could help guide clinicians in identifying high-risk patients and implementing timely preventive interventions.

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Keywords: glucocorticoids, mania, Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal infections

Presentation: Case Report was presented at The University of Kansas School of Medicine-Wichita Research Symposium in April 2024.

APPENDIX A

Timeline of Risperidone Taper

APPENDIX B

Comparative Table of Three Published Cases of Steroid-induced Mania in Pediatric Patients.

Case	Demographics	Medical & Substance History	Corticosteroid Info	Psychiatric Symptoms	Treatment & Response	Duration of Treatment
Couturier et al.[5]	Age: 15, Gender: F	Asthma; Weekly alcohol and marijuana use; Significant recurrent major depression (father, maternal grandfather)	Salbutamol, fluticasone, and prednisone 25 mg daily + clarithromycin 250 mg BID; Prednisone advanced to 50 mg daily for five days; Previous exposure: Short-term oral prednisone (30–50 mg daily); IV methylprednisolone 160 mg daily × three days => oral prednisone up to 80 mg daily	Elated mood, hyper-religiosity, pressured speech, auditory/visual hallucinations	Olanzapine was titrated from 5 mg to 10 mg at bedtime over five days, Valproic acid to 750 mg daily, and Lithium to 600 mg daily. Symptoms resolved in 7, 9, and 20 days and remained stable despite prednisone re-exposure (45 mg daily for seven days with clarithromycin 250 mg BID).	20 days of acute treatment and six months of planned maintenance therapy
Khan et al.[4]	Age: 16, Gender: F	Asthma; No substance or family history	Beclomethasone inhaler 42 µg one – two times each nostril twice daily; Previous exposure unknown	Euphoric mood, religious grandiosity, flight of ideas, impulsivity (self-mutilating behavior), racing thoughts, pressured speech, decreased need for sleep, increased energy	Discontinuation of inhaler. Mania resolved 48 hours after cessation of corticosteroid inhalers; no antipsychotics or mood stabilizers.	48 hours
Cassidy et al.[6]	Age: 17, Gender: M	Acute Lymphocytic Leukemia (ALL); No substance or family history	Dexamethasone 10 mg daily for 28 days; Previous exposure unknown	Affective lability, decreased need for sleep, increased energy, talkativeness, grandiosity	Risperidone 1 mg BID and Lorazepam 1 mg BID were titrated to a total of 4 mg daily. The patient was discharged on Risperidone 1 mg qam and 2 mg qhs, tapered to 0.25 mg at bedtime, remaining stable despite intermittent dexamethasone (up to 20 mg daily)	Three weeks of acute inpatient treatment; outpatient tapering duration not specified

KANSAS JOURNAL of MEDICINE

Case Report

Massive Upper Gastrointestinal Bleeding Secondary to a Large Pedunculated Brunner Gland Hamartoma in the Duodenum

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INTRODUCTION

Brunner glands are deep mucosal and submucosal alkaline secreting glands mostly located in the proximal duodenum with their quantity and size diminishing in the distal regions.[1] These alkalotic secretions neutralize the acidic contents of the stomach, therefore maintaining the duodenal mucosal epithelium and alkalotic milieu necessary for absorption.[2]

Brunneroma, or Brunner gland hamartoma (BGH), is a rare benign tumor with prevalence of 0.008%, comprising 10% of all benign tumors in the duodenum.[3] Most cases occur in patients aged 50 to 70 years.[3] Visualization of BGH is done by esophagogastroduodenoscopy (EGD), although endoscopic ultrasonography has been increasingly utilized for the evaluation of the origin, extent, and vascularity of the lesion.[3] Computed tomography (CT) scans may serve as an initial tool to detect large BGHs. It also can be done to confirm the absence of extraluminal extension.[2] Confirmation of diagnosis is established with histopathology, which shows Brunner glands, ducts, smooth muscle, fibrous tissue, and lymphocytes with immunohistochemistry positive for Mucin 6 (MUC6).[4] We present a rare case of massive upper gastrointestinal (GI) bleed caused by a large pedunculated BGH in a 70-year-old patient.

CASE REPORT

A 70-year-old female with previous history of iron deficiency anemia presented to the emergency department for fatigue and lightheadedness accompanied by new onset black tarry stools of two days' duration. The patient had no previous history of non-steroidal anti-inflammatory drug (NSAID) or aspirin use. Upon presentation, she was hemodynamically stable. The abdominal examination revealed no significant findings, with the absence of tenderness or distension. Initial labs revealed a hemoglobin level of 5.4 g/dl, significantly lower than her baseline of 9 g/dl. Labs also indicated iron deficiency anemia. Intravenous (IV) fluids and IV pantoprazole were started. Two units of packed red blood cells were transfused. Although the patient's hemoglobin improved to 7.9 g/dl following the transfusion, it dropped back to 6.8 g/dl, while still having persistent black stools. EGD showed a polypoid mass with irregular mucosa and ulceration in the second portion of the duodenum away from the major papilla, so biopsies were taken (Figure 1). A subsequent CT of the abdomen and pelvis showed no extraluminal masses. With ongoing hemoglobin decline, dropping to 5.1 g/dl, CT angiography of the abdomen and pelvis revealed bleeding from the gastroduodenal artery, which was managed by embolization. Initially, pathology indicated inflamed and ulcerated benign mucosa with reactive epithelial changes. *Helicobacter pylori* testing was negative on biopsy. The patient underwent another EGD for complete endoscopic mucosal resection (EMR) and an endoscopic ultrasound (EUS). EUS demonstrated a hypoechoic pedunculated mass measuring up to 21 mm confined to the mucosal layers. The lesion was noted to be well away from the ampulla. Epinephrine was injected to decrease the bleeding risk, followed by successful placement of a Polyloop™ ligature (Figure 2). Hot snare mucosal resection was successful at the level of the polyp and the BGH was completely retrieved (Figure 3). A hemostatic clip was placed to prevent rebleeding afterwards. Pathology results after complete resection revealed prominent lamina propria capillaries, gastric mucin cell metaplasia, Brunner's gland hyperplasia, and cystic dilatation, indicative of BGH (Figure 4). After resection, the patient's tarry stools resolved, and her hemoglobin stabilized at around 9 g/dl.

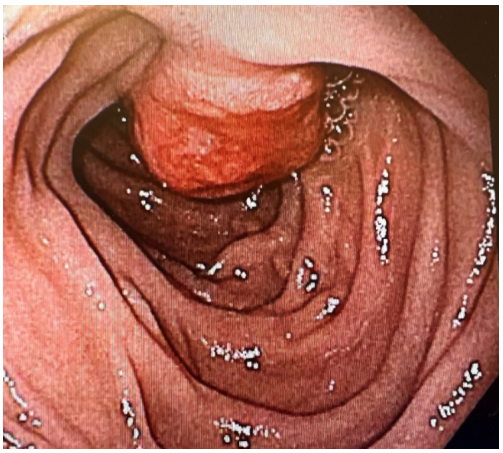

Figure 1. Brunner Gland Hamartoma (BGH) in the second portion of the duodenum away from the major papilla.

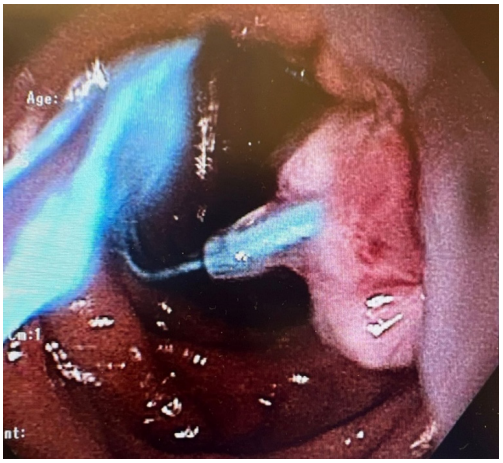

Figure 2. Polyloop™ ligature placed on Brunner Gland Hamartoma (BGH).

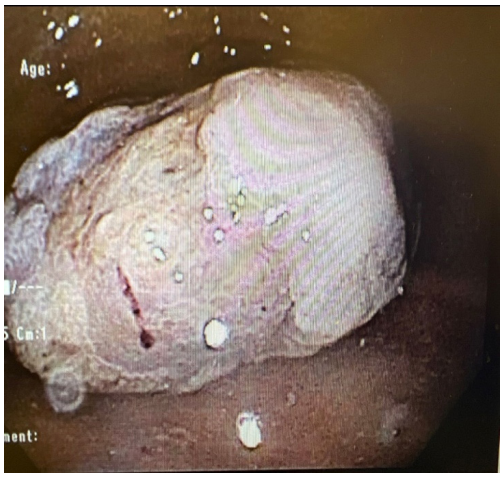

Figure 3. Brunner Gland Hamartoma (BGH) post endoscopic mucosal resection (EMR).

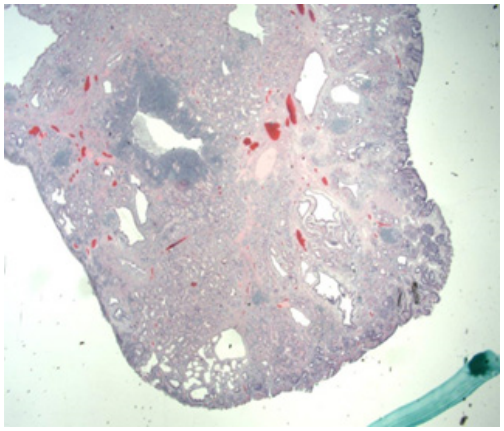

Figure 4. Brunner Gland Hamartoma (BGH) under 1.25x magnification hematoxylin and eosin (H&E) staining.

DISCUSSION

BGHs are rare benign duodenal tumors ranging from 1 to 3 cm in size.[5] Up to 70% of these tumors have been found to occur near the duodenal ampulla.[6] BGHs generally are solitary and become pedunculated and polypoidal as they grow.[7] While they often are asymptomatic and detected incidentally, they can occasionally present with symptoms such as intestinal obstruction or upper GI bleeding.[6] Symptomatic patients with upper GI bleeding often present with anemia from chronic blood loss. They also report melena four times more commonly than hematemesis.[8] In rare cases where a tumor extends into the pancreas, it can obstruct the ampulla of Vater or pancreatic duct, potentially leading to complications such as inflammation, pancreatitis, or obstructive jaundice.[8]

In this case, we found a large pedunculated BGH measuring 21 mm in the second portion of the duodenum away from the major papilla, which is an atypical location. The patient presented with lightheadedness, weakness, fatigue, and black tarry stools, indicative of anemia and melena secondary to BGH bleeding.

To diagnose BGH, EGD and CT scanning are done initially to visualize the lesion causing the bleed.[8] CT scans often may show a polypoid filling defect, indicating extraluminal extension of the tumor.[9] In this case, we first visualized the tumor on EGD, and a subsequent CT scan showed no filling defects or extension into the duodenal wall.

For a definitive diagnosis of BGH, histological examination of the tumor is needed.[10] Endoscopic biopsies usually are superficial and not sufficient to reach the BGH in the submucosa, which often leads to falsely negative results initially.[2] Hence, the final diagnosis is made based upon histological features after tumor resection.[10] Microscopically, BGHs show polypoidal growth of Brunner glands, with fibromuscular and adipose tissue components, lymphoid aggregates, and cystic dilatation of Brunner glands.[1]

Treatment usually involves endoscopic polypectomy for pedunculated tumors >2 cm in size.[3] It is generally safe, minimally invasive, and cost-effective.[5] However, surgical resection sometimes is necessary if endoscopic measures are not possible or have failed.[8] In the literature, there are no reported cases of BGH recurrence after complete resection, and the long-term outcomes are highly favorable.[5]

This case underscores the importance of complete resection for accurate diagnosis, as initial biopsies may not always reveal characteristic features due to its submucosal location.

CONCLUSIONS

BGHs typically are benign lesions diagnosed incidentally during EGD or imaging. However, in some cases they can present with significant complications, such as GI hemorrhage or obstruction. Given the diagnostic challenges associated with biopsy sampling, complete resection of the tumor is imperative for achieving a definitive diagnosis. Both diagnosis and treatment of symptomatic BGH involve complete resection of the tumor with endoscopic or surgical approaches.

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Keywords: Brunner Glands, Hamartoma, Gastrointestinal Hemorrhage, Duodenal Neoplasms, Upper Gastrointestinal Tract

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KANSAS JOURNAL of MEDICINE

Case Report

Metastatic Cardiac Angiosarcoma Presenting as Superior Vena Cava Syndrome and Cardiac Tamponade

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INTRODUCTION

Primary cardiac angiosarcomas are rare, aggressive vascular endothelial tumors associated with a poor prognosis among soft tissue sarcomas.[1] Their clinical presentation often is variable and nonspecific, commonly including chest pain, shortness of breath, and palpitations, symptoms that result from hemodynamic changes due to structural invasion of the heart.[2,3] The diagnostic workup involves a combination of noninvasive imaging, such as transthoracic echocardiogram (TTE), computed tomography (CT), and magnetic resonance imaging (MRI), along with invasive biopsy and immunohistochemical analysis for confirmation.

There is limited literature establishing a robust standard of care for cardiac angiosarcoma. Current treatment approaches include surgery, radiotherapy, and chemotherapy.[1,4] However, recent advances in care, particularly those guided by immunogenetics, show promise, with success often dependent on early diagnosis.[4]

We describe a challenging case of newly diagnosed cardiac angiosarcoma in a 50-year-old male who presented with superior vena cava (SVC) syndrome, complicated by cardiac tamponade.

CASE REPORT

A 50-year-old male with a history of hypertension initially presented with throat pain, right-sided sublingual swelling, episodic secretions, and voice changes lasting three weeks. CT imaging of the neck revealed retropharyngeal effusion and significant edema of the upper oropharyngeal airway, though the laryngeal airway remained intact. Given the patient's chills and new leukocytosis (16,430 WBCs/μL), he was started on broad-spectrum intravenous (IV) antibiotics and experienced symptomatic improvement. Flexible fiberoptic laryngoscopy showed esophageal collapse without edema, and cultures were negative. The patient was discharged on oral antibiotics with a decreased suspicion for Ludwig's angina.

Two days post-discharge, the patient returned with recurrent throat pain and sublingual swelling, now accompanied by new facial plethora. CT of the chest revealed a filling defect within the SVC and upper right atrium, prominent mediastinal venous collaterals, moderate circumferential pericardial effusion (up to 1.6 cm anteriorly), and a 1.3 cm right thyroid nodule. He was started on IV heparin due to concern for SVC syndrome from a possible thrombus or embolus. Positron emission tomography (PET)/CT identified a hypermetabolic right atrial mass (5 x 4.6 cm, Standardized Uptake Value [SUV] max 11.6) infiltrating the atrial appendage, and focal activity in the right thyroid nodule (SUV max 10.6). Given a newly elevated thyroid stimulating hormone (TSH) level (10.5 mIU/L) and a family history of head and neck cancer, the thyroid nodule was biopsied and found to be benign. The patient opted for discharge with close outpatient follow-up and was transitioned to oral apixaban after resolution of facial plethora and throat symptoms.

Two weeks later, he returned with facial swelling and new-onset dizziness. He was found to be in atrial flutter with a resting heart rate of 100 beats per minute. Repeat imaging showed an enlarging right atrial mass (5.6 x 5.3 x 4.9 cm) invading the right mediastinum and SVC, with thrombus extending to the level of the azygos vein. Mediastinal edema was seen extending into the SVC-cavoatrial junction (Figure 1). CT of the chest also revealed a new, large pericardial effusion. The patient exhibited clinical signs of cardiac tamponade (Beck's triad: hypotension, muffled heart sounds, and jugular venous distension), requiring urgent pericardiocentesis with drainage of 650 mL of dark blood and catheter placement. Limited TTE showed reduced systolic function, which improved following the procedure.

Biopsy of the right atrial mass and pericardial fluid analysis were positive for erythroblast transformation-specific related gene (ERG) and cluster of differentiation (CD31) on immunohistochemistry. The fluid was negative for MOC31, Ber-EP4, WT1, and Calretinin. These findings were concerning for metastatic angiosarcoma. The case was presented at a multidisciplinary sarcoma tumor board. Due to extensive tumor infiltration into the right atrial wall (Figure 2), the patient was not a surgical candidate. He was started on neoadjuvant chemotherapy with weekly paclitaxel.

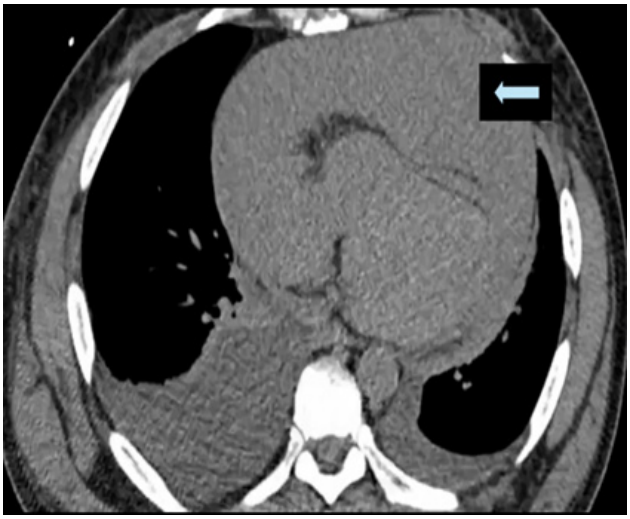

Figure 1. Axial CT demonstrating new large pericardial effusion with tamponade.

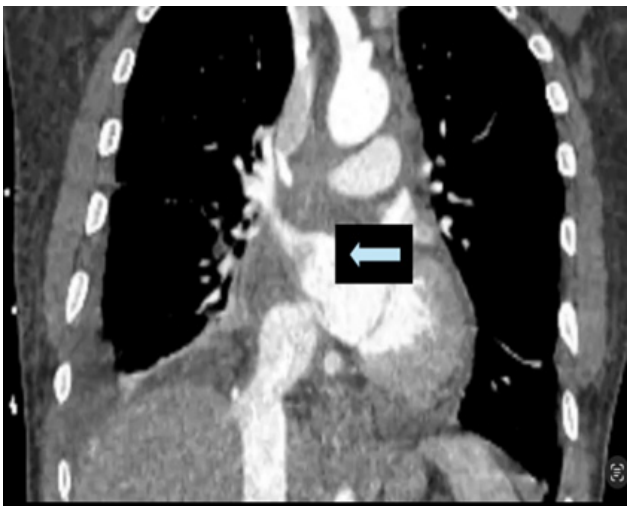

Figure 2. Coronal CT demonstrating right atrial mass measuring 5.6 x 5.3 x 4.9 cm, invading the superior vena cava with thrombosis and right atrial wall.

DISCUSSION

Cardiac angiosarcomas account for less than 2% of all sarcomas.[5] The case presented in this article highlights the extensive challenges involved in the timely and accurate diagnosis of cardiac angiosarcoma. Although these tumors are highly aggressive, their initial presentation often is variable and nonspecific, complicating diagnosis.

This patient initially presented with throat pain, voice changes, and swelling in the floor of the mouth, raising concern for Ludwig's angina, a condition that requires urgent evaluation and was ultimately ruled out.[6] The presentation of Ludwig's angina in the context of an underlying sarcoma is rare. Vijapur et al.[7] reported a case of a 21-year-old with submandibular swelling concerning for Ludwig's angina; subsequent imaging revealed mandibular osteomyelitis and led to the diagnosis of osteosarcoma. However, to date, there are no similar reports linking Ludwig's angina–like presentation with cardiac angiosarcoma. This underscores the complexity of clinical decision-making, especially when initial imaging suggests a potential airway emergency in the absence of thrombosis.

Concern for SVC syndrome arose following new CT imaging that revealed a filling defect within the SVC and right atrium, along with prominent venous collaterals.[8] While over 50% of SVC syndrome cases are associated with malignancy, they are most commonly due to non-small cell lung cancer, small cell lung cancer, or lymphoma.[9-14] Few cases in the literature describe SVC syndrome caused by angiosarcoma. Abratt et al.[15] described a 7 cm mediastinal mass from a primary angiosarcoma involving the SVC, and Salgueiro et al.[16] reported SVC thrombosis secondary to primary angiosarcoma involving the brachiocephalic veins. SVC syndrome also has been observed in other sarcomas, such as sarcomatoid renal cell carcinoma and osteosarcoma.[7,17,18]

The clinical suspicion for SVC syndrome in this case led to early CT angiography, which helped identify thrombosis. The presence of venous collaterals can be an important imaging clue. Subsequent PET/CT imaging aided in characterizing metabolic activity and helped distinguish benign from malignant lesions.[8,15,16]

Cardiac tamponade is another rare presentation of cardiac angiosarcoma.[20-22] In this case, the patient initially had a moderate (1.6 cm) circumferential pericardial effusion, which progressed to tamponade and required pericardiocentesis. The aggressive nature of cardiac angiosarcoma may contribute to rapid fluid accumulation. While there are case reports of direct oral anticoagulants causing hemorrhagic tamponade, no studies to date have explored this specifically in the setting of cardiac angiosarcoma, although it remains a plausible contributing factor.[23-25]

Due to its rarity, there are no standardized treatment protocols for cardiac angiosarcoma. Surgical resection or debulking often is considered, though the tumor's aggressive and infiltrative nature may necessitate complex reconstruction of intrathoracic structures to achieve negative margins.[26,27] In this case, atrial flutter further complicated clinical management. Patients without surgical resection typically have a median survival of only three to six months.[28,29]

Some cases have reported the use of neoadjuvant chemotherapy in unresectable cardiac angiosarcoma.[30,31] A multicenter phase II trial of paclitaxel in patients with metastatic or unresectable angiosarcoma demonstrated a non-progression rate of 62.5% and a median survival of 16 months.[31] However, additional research is needed to determine the most effective treatment strategies for this rare and challenging malignancy.

CONCLUSIONS

This case highlights the complex diagnostic challenges associated with cardiac angiosarcoma. The patient initially presented with symptoms suggestive of SVC syndrome, which were due to an underlying, undiagnosed cardiac angiosarcoma. The diagnosis was ultimately made during a subsequent presentation with cardiac tamponade. This underscores the importance of maintaining a high index of suspicion for cardiac angiosarcoma in patients presenting with SVC syndrome, as early recognition is crucial for initiating appropriate treatment.

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Keywords: superior vena cava syndrome, cardiac tamponade, sarcoma, clinical oncology

KANSAS JOURNAL *of* MEDICINE

Case Report: Hemoglobin of 2.0 g/dL- Secondary to Excessive Cow's Milk Intake in a 2-Year-Old Male

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INTRODUCTION

Iron deficiency anemia (IDA) is a common condition in young children, particularly those who consume large amounts of cow's milk, which is low in iron and can contribute to iron depletion over time. The incidence of IDA is as high as 20.1% in industrialized countries and 39% in developing countries among children aged 0-4 years.[1] Excessive cow's milk consumption can lead to occult gastrointestinal blood loss, colitis, and impaired iron absorption.[2] This report describes a 2-year-old male with severe IDA secondary to excessive cow's milk intake, requiring admission to the pediatric intensive care unit and multiple blood transfusions. This case highlights the importance of assessing nutrition in young children and recognizing the risk of anemia associated with overconsumption of cow's milk.

CASE REPORT

A 2-year-old male presented to the emergency department (ED) with his parents, who reported worsening weakness, fussiness, and poor appetite over the past six months. They noted that he could no longer climb a flight of stairs without becoming fatigued and fussy.

On presentation, his vital signs were within normal limits: temperature 36.7°C, heart rate 139 beats per minute, respiratory rate 22 breaths per minute, and oxygen saturation 97% on room air. His height was 82.55 cm, and his weight had declined from the 97th percentile a year prior to the 37th percentile. Physical examination revealed a pale but alert, active, and playful child. There were no signs of acute illness, rash, bruising, or hepatosplenomegaly.

Initial laboratory workup showed profound anemia (Table 1). Peripheral smear revealed microcytic anemia with anisopoikilocytosis, including ovalocytes, polychromasia, and elliptocytes. Given the severe anemia, additional history was obtained from the parents, who disclosed that the child primarily consumed cow's milk, approximately half a gallon per day, and was a highly selective eater, refusing most other foods. They denied any significant bleeding, epistaxis, or easy bruising but reported low-volume, hard stools without visible blood.

The child was admitted to the pediatric intensive care unit for further management. His anemia was attributed to iron deficiency from inadequate dietary intake and chronic gastrointestinal blood loss secondary to cow's milk protein-induced colitis. He received a total of 15 mL/kg of packed red blood cells, divided into three equal aliquots administered six hours apart. Hematology was consulted and recommended continued iron supplementation following transfusion. He received iron dextran (100 mg daily for five days) and was started on oral elemental iron (30 mg daily).

Table 1. Initial labs in the emergency department.

Lab	Result
Hemoglobin	2.0 g/dL
Hematocrit	9.9%
Mean Corpuscular Volume	50.8 fL
Mean Corpuscular Hemoglobin	10.3 pg
Mean Corpuscular Hemoglobin Concentration	20.2 g/dL
Red Cell Distribution Width	24.9%
White Blood Cell Count	8,300/µL
Platelet Count	279,000/µL
Reticulocyte Count	2.7%
Guaiac Test	Positive for occult blood
Iron	37 µg/dL
Total Iron Binding Capacity	682 µg/dL
Iron Saturation	5%
Transferrin	458 mg/dL
Ferritin	5 ng/dL
Lead Level	<1µg/dL

Throughout his hospital stay, the patient's energy and pallor improved without signs of transfusion-related complications. The presence of occult blood in the stool was attributed to cow's milk protein-induced colitis. Hematology confirmed the diagnosis of severe iron deficiency anemia (IDA) based on dietary history, clinical presentation, laboratory findings of microcytic, hypochromic anemia, and a positive guaiac test.

After five days of hospitalization, the patient was discharged in stable condition. His parents received extensive counseling on dietary diversification, including increasing iron-rich foods and limiting cow's milk intake. He also was referred for outpatient nutritional counseling.

At a one-month follow-up with his pediatrician, his hemoglobin had improved to 11.5 g/dL, and he had resumed eating solid foods.

DISCUSSION

IDA is a common condition in toddlers, particularly those who consume excessive amounts of cow's milk. Cow's milk is low in iron, inhibits iron absorption, and can cause occult gastrointestinal bleeding due to cow's milk protein-induced colitis.[3] This case highlights the severity of anemia that can develop in such circumstances and underscores the importance of dietary screening and counseling to prevent iron deficiency.

The American Academy of Pediatrics (AAP) recommends limiting cow's milk intake to 16-24 ounces per day for children aged 1-5 years to prevent nutritional deficiencies.[4] In cases of severe anemia, blood transfusion may be necessary, as seen in this patient. Following transfusion, iron supplementation is essential to replenish iron stores and prevent recurrence.

CONCLUSIONS

This case of a 2-year-old male with severe IDA illustrates the risks associated with excessive cow's milk consumption in young children. It underscores the importance of early recognition, dietary counseling, and appropriate management to prevent potentially life-threatening complications.

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Keywords: anemia, iron deficiencies

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Case Report

Traumatic Facial Artery Dissection from Chinstrap Injury: A Case Report

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INTRODUCTION

Traumatic dissection of the facial artery due to blunt trauma has not been previously reported. In contrast, blunt cerebrovascular injuries (BCVI) involving the carotid and vertebral arteries are well-documented and recognized as potentially devastating.[1,2] This case report presents a rare instance of facial artery dissection resulting from blunt trauma caused by a chinstrap injury. Clinicians should maintain a high index of suspicion for vascular injury in patients presenting after chinstrap-related trauma or other high-grade blunt injuries.

CASE REPORT

A 48-year-old male construction worker presented as a trauma activation after falling from scaffolding, sustaining blunt trauma to the head, face, and chest. His hard hat became caught on the scaffolding, and he was briefly suspended by his chinstrap before falling to the ground. He did not lose consciousness.

Upon arrival to the emergency department (ED), the patient reported pain in the right jaw and right chest. His primary survey was intact: airway was patent, bilateral breath sounds were present, central pulses were palpable, and his Glasgow Coma Scale (GCS) score was 15. The secondary survey revealed a 3 cm laceration over the angle of the right mandible. Vital signs remained stable.

Non-contrast computed tomography (CT) of the head, maxillofacial bones, and cervical spine revealed a small cortical chip fracture of the right mandible underlying the laceration (Figure 1). CT angiography (CTA) of the head and neck demonstrated segmental occlusion and/or high-grade stenosis of the right facial artery within the perimandibular laceration area, consistent with post-traumatic dissection (Figure 2). There was no evidence of hematoma, pseudoaneurysm, or active contrast extravasation. No additional injuries were identified on imaging.

The patient received cefazolin for the open fracture. Otolaryngology (ENT) and vascular surgery were consulted. ENT repaired the laceration and recommended nonoperative management of the mandibular chip fracture without need for follow-up. Vascular surgery advised against intervention for the isolated facial artery occlusion, as there was no involvement of the common, internal, or external carotid arteries. The patient was discharged home in stable condition with a prescription for amoxicillin-clavulanate.

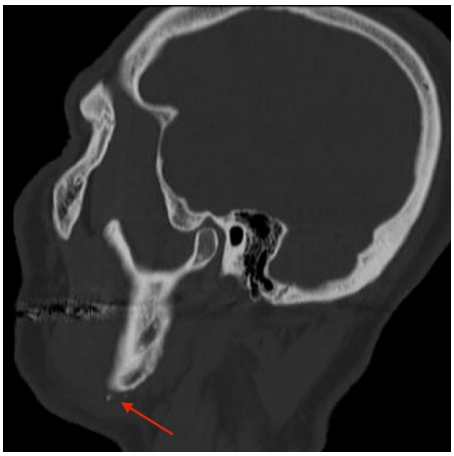

Figure 1. CT maxillofacial without contrast showing tiny right mandibular cortical chip fracture (arrow).

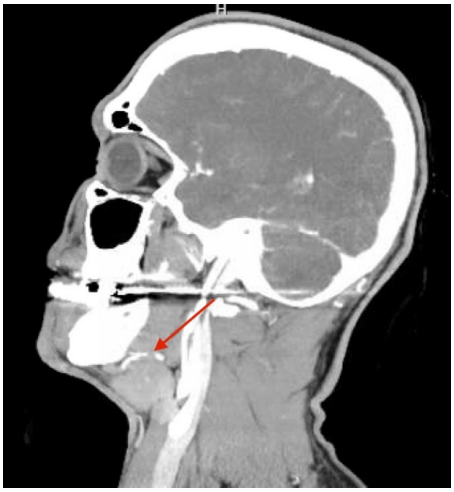

Figure 2. CTA head and neck showing traumatic occlusive dissection of the right facial artery (arrow).

DISCUSSION

The facial artery is a major branch of the external carotid artery (ECA). It originates from the anterior surface of the ECA within the carotid triangle of the neck and courses deep to the digastric and stylohyoid muscles before curving upward to cross over the body of the mandible. It follows a tortuous path toward the angle of the mouth and continues along the lateral aspect of the nose, terminating near the medial canthus of the eye.[3] The facial artery primarily supplies the superficial structures of the face.

There is limited literature describing blunt injury to the branch arteries of the internal and external carotids. To our knowledge, traumatic occlusive dissection of the facial artery due to blunt facial or neck trauma has not been previously reported. As a result, the appropriate treatment and surveillance of such injuries remain undefined and are largely guided by expert opinion. In such cases, it may be reasonable to extrapolate management strategies from existing guidelines for BCVIs.

BCVIs, non-penetrating injuries of the carotid and vertebral arteries, are well-described in the literature. They are identified in approximately 1-2% of blunt trauma patients and can result in severe complications

such as ischemic stroke and long-term neurologic impairment.[1,4] Current guidelines recommend early initiation of antithrombotic therapy in BCVI.[5]

In theory, blunt injury to a branch artery also may pose a risk of thrombotic or embolic complications, though with variable clinical significance. More likely sequelae may include acute bleeding or delayed pseudoaneurysm formation. In this case, management was guided by expert consultation. Vascular surgery reviewed the CTA and identified an isolated occlusion of the right facial artery, without injury to the common, internal, or external carotid arteries. Unlike carotid or vertebral artery injuries, which carry high risks for ischemic events, the extensive collateral circulation of the face significantly reduces the likelihood of serious ischemic outcomes from facial artery occlusion.

As with BCVI, the decision to initiate antithrombotic therapy for facial artery dissection should be based on patient-specific factors such as injury severity and bleeding risk. Our patient was deemed low risk for complications and did not require antithrombotic therapy or vascular follow-up. He was discharged from the ED in stable condition.

CONCLUSIONS

Traumatic dissection of the facial artery following blunt trauma is extremely rare and, to our knowledge, has not been previously described in the literature. More data are needed to better understand its incidence, potential complications, and appropriate management. In such rare presentations, emergency providers should consider consulting vascular surgery or other relevant specialists to guide care decisions.

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Keywords: dissection, blunt injury, vascular system injuries, mandibular fracture, mandibular injury

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Case Report

An Alteration to Standardized Treatments: Defunctioning Colostomy in Ultra-Low Stage IIIC Rectal Adenocarcinoma

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INTRODUCTION

In the United States, approximately 46,000 cases of rectal adenocarcinoma are diagnosed each year.[1] The most common presenting symptoms include rectal bleeding, the sensation of a rectal mass, iron deficiency anemia, and abdominal pain.[2] A change in bowel habits also is frequently reported, though this typically refers to changes in frequency or stool consistency rather than incontinence.[2]

Approximately 27% of patients with colorectal cancer present with large bowel obstruction.[3] For patients who present prior to obstruction, clinicians must weigh the risks of disease progression or radiotherapy-associated edema leading to obstruction, which could interrupt neoadjuvant therapy, against the risks of prophylactic stoma creation (ileostomy or colostomy) before beginning treatment.[3,4] According to Mesri et al.,[3] protocols for non-emergent stoma creation, particularly in cases of potentially obstructing rectal cancer during neoadjuvant chemoradiation therapy (CRT), or when other quality-of-life complications exist, are poorly defined.[3] As a result, patients may face difficult decisions about undergoing a preventive surgery that could ultimately prove unnecessary.[5]

To contribute to the growing body of case-based evidence on factors influencing this decision, we present the case of a patient with stage IIIC rectal adenocarcinoma who experienced significant fecal incontinence symptoms at presentation.

CASE REPORT

A man in his 70s was referred to general surgery as part of a multidisciplinary plan to determine the best course of treatment for recently diagnosed stage IIIC rectal cancer. He had presented to the emergency department three weeks earlier with a chief complaint of unexplained weight loss (60 pounds over four months; body mass index 22.2 kg/m^2). During review of systems, he also reported weakness, night sweats, intermittent hematochezia, hair loss, dizziness, and cold intolerance. His medical history included hypertension and type 2 diabetes mellitus, managed with insulin glargine. His only prior abdominal surgery was resection of a left lower quadrant liposarcoma five years ago, which did not involve adjacent structures.

On rectal examination, low sphincter tone and a palpable intraluminal mass were noted in the distal rectum. Computed tomography (CT) imaging of the abdomen and pelvis revealed an irregular cystic/solid lesion in the mesorectal space, likely arising from the rectum (Figure 1). Colonoscopy identified a large, partially obstructing distal mass occupying approximately 90% of the lumen, extending from the rectum to the sigmoid colon. Biopsies confirmed well to moderately differentiated adenocarcinoma with no evidence of mismatch repair deficiency.

Staging chest CT showed no evidence of distant metastases. Pelvic magnetic resonance imaging (MRI) demonstrated a 61.5 mm lobulated, heterogeneously enhancing mass located 1 cm from the anal verge (Figure 2). The tumor was staged as T4b, N2a, cM0. Pretreatment serum carcinoembryonic antigen (CEA) was 235.22 ng/mL, indicating a poor prognosis but serving as a valuable baseline for assessing future treatment response and recurrence.[6]

During his oncology consultation, the patient reported worsening fecal incontinence since his weight loss began, requiring the use of disposable undergarments. He had not previously disclosed these symptoms, stating he was never directly asked and felt uncomfortable volunteering the information. The Wexner fecal incontinence score, a validated tool evaluating the severity and impact of incontinence on quality of life, was administered and returned a score of 20/20, consistent with complete incontinence.[7,8]

Given the patient's severe incontinence, there was concern for worsening dermatitis or infection during chemoradiation therapy (CRT),[9] as well as the risk of acute bowel obstruction from tumor-related edema. These factors made the risks associated with prophylactic stoma creation prior to CRT acceptable. After discussing the options, including proceeding directly to CRT or undergoing surgery first, the patient opted for surgery.

A defunctioning stoma procedure was planned; however, during surgical mobilization of the sigmoid colon, scarring and mesenteric shortening from the prior liposarcoma resection were identified. Due to devascularization of the colon, a Hartmann's procedure was performed: the devascularized segment was resected, the rectum was stapled closed (just proximal to the tumor), and a colostomy was created from the remaining colon. The patient recovered without significant complications.

Post-operatively, he began long-course chemoradiation with capecitabine, followed by 16 weeks of FOLFOX chemotherapy per National Comprehensive Cancer Network (NCCN) guidelines.[6] Restaging with serum CEA, MRI and CT were planned 20 weeks after the start of CRT. Based on tumor response, additional CRT, surgical resection, or a watch-and-wait strategy will be considered.

The patient acknowledged challenges adjusting to the colostomy, including dietary changes, supply access, and social anxiety, but reported that it provided improved hygiene, comfort, and control compared to his prior incontinence. If total tumor regression is achieved or sufficient sphincter function remains after resection, reversal of the colostomy will be considered through shared decision-making and clinical judgment.

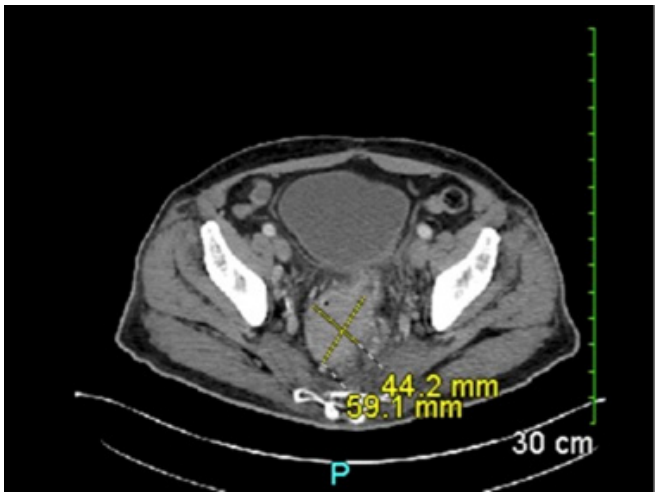

Figure 1. Abdomen/pelvis CT showing the rectal mass measuring 44.2 x 59.1 mm.

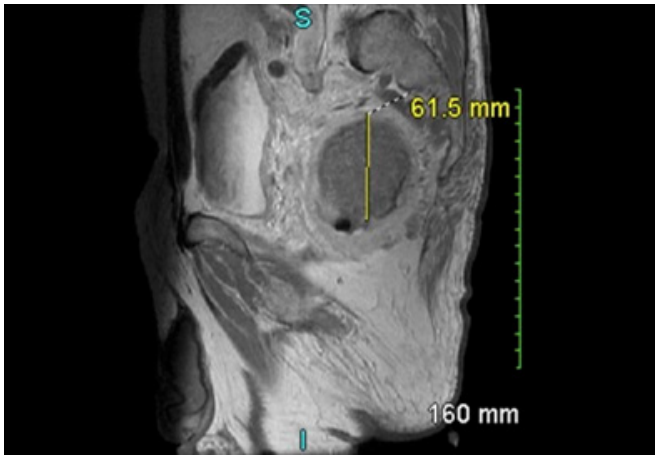

Figure 2. Abdomen/pelvis MRI showing the rectal mass with a height of 61.5 mm.

DISCUSSION

Ultra-low rectal cancer is associated with a risk of bowel obstruction requiring emergency surgery at presentation,[3] as well as significant changes in bowel function.[10] In non-emergent cases, the NCCN guidelines recommend CRT for stage T3 or T4 rectal cancers, followed by restaging to determine candidacy for surgical resection with anastomosis or colostomy if needed.[6]

The role of defunctioning stomas prior to neoadjuvant therapy remains controversial, as reliable prognostic indicators for bowel obstruction during CRT are not well established.[3] Quality-of-life considerations, such as severe fecal incontinence, may influence the decision to pursue pre-treatment stoma creation as part of the treatment plan.

It is important to counsel patients on the possibility that a temporary stoma may become permanent. In one study of 134 patients, 17% ultimately did not undergo reversal.[11] Because many patients may be reluctant to disclose sensitive symptoms such as incontinence, direct and empathetic conversations are essential to ensure comprehensive symptom management during treatment.

More research is needed to define optimal timing and selection criteria for defunctioning stoma creation in rectal cancer. At present, decisions should be made collaboratively by the patient and a multidisciplinary care team, with individualized consideration of both clinical and quality-of-life factors.

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Keywords: rectal cancer, ostomy, fecal incontinence

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