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SOLITARY JUVENILE RECTAL POLYP IN A TODDLER PRESENTING WITH PROLAPSE AND HEMATOCHEZIA: A CASE REPORT AND COMPREHENSIVE REVIEW OF DESMOID-TYPE FIBROMATOSIS

Hassita Wenda Mayrina[1], Harisma Widhanar [2]
[1] General Practitioner, Muhammadiyah Kalitidu General Hospital, Bojonegoro Regency, East Java, Indonesia
[2] General Surgery Consultant, Muhammadiyah Kalitidu General Hospital, Bojonegoro Regency, East Java, Indonesia
Corresponding Email : hassitawendamayrina@gmail.com

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ABSTRACT

Introduction: Juvenile polyps are the most common cause of lower gastrointestinal bleeding in the pediatric population. These lesions are typically benign hamartomatous growths, and a solitary polyp is rarely associated with long-term morbidity. This report aims to present a classic case of a solitary juvenile polyp leading to significant clinical symptoms in a toddler. Additionally, it provides a comprehensive, state-of-the-art review of desmoid-type fibromatosis, a rare soft tissue neoplasm that, while unrelated to this case's final diagnosis, represents an important entity in the differential diagnosis of pediatric masses.

Case Illustration: A 19-month-old male presented to the emergency department with a seven-day history of progressively worsening hematochezia and a one-day history of a prolapsed, irreducible rectal mass. The patient exhibited signs of weakness

and anemia. Physical examination revealed a 3x2x2 cm, fresh red, pedunculated mass protruding from the anus. After initial stabilization, the patient underwent a transanal polypectomy. The post-operative course was complicated by significant hemorrhage on the second day, which was managed successfully with conservative measures, including tranexamic acid. Histopathological examination of the excised specimen confirmed the diagnosis of a benign hamartomatous juvenile polyp.

Discussion: The diagnosis and management of the solitary juvenile polyp were consistent with established clinical practice. Simple polypectomy is curative, and further surveillance is generally not required in the absence of multiple polyps or a family history of polyposis syndromes. The main focus of this discussion is a detailed review of desmoid-type fibromatosis. This entity is defined as a locally aggressive, non-metastasizing fibroblastic neoplasm. Its pathogenesis is driven by dysregulation of the Wnt/β-catenin signaling pathway. A significant paradigm shift has occurred in its management, moving away from aggressive upfront surgery towards a more conservative approach centered on active surveillance, with systemic therapies like tyrosine kinase inhibitors and gamma-secretase inhibitors reserved for progressive or symptomatic disease.

Conclusion: Solitary juvenile polyps are a common and highly treatable cause of rectal bleeding in children. This case highlights the typical presentation and a potential post-operative complication. The accompanying review of desmoid-type fibromatosis serves an important educational purpose, underscoring the evolving management strategies for rare soft

tissue tumors, which prioritize balancing disease control with long-term quality of life.

Keywords: Juvenile Polyp, Hematochezia, Polypectomy, Desmoid Fibromatosis, Aggressive Fibromatosis, β-catenin.

INTRODUCTION

Lower gastrointestinal bleeding (LGIB) in infants and toddlers is a frequent and often alarming presenting complaint in pediatric practice. While the differential diagnosis is broad, encompassing conditions such as anal fissures, infectious colitis, and Meckel's diverticulum, juvenile polyps stand out as the most common etiology.[1] These growths, which are benign by nature, can lead to significant, albeit typically painless, hematochezia.

Juvenile polyps are hamartomatous proliferations of the colorectal mucosa, meaning they are composed of a disorganized mixture of normal tissue elements. It is crucial to distinguish between two distinct clinical entities. A **solitary juvenile polyp** is a common, sporadic lesion that constitutes the vast majority of cases in children. It carries no inherent malignant potential and is typically cured by simple removal.[1] In contrast, **Juvenile Polyposis Syndrome (JPS)** is a rare, autosomal dominant inherited condition defined by the presence of multiple (typically five or more) juvenile polyps throughout the gastrointestinal tract.[2] JPS is associated with a significantly increased lifetime risk of gastrointestinal cancers, necessitating a rigorous long-term surveillance program.[2] This distinction underscores the importance of a thorough evaluation, including histopathological analysis and consideration of family history, in every child presenting with a colorectal polyp.

This report details the case of a 19-month-old male who presented with severe hematochezia and prolapse of a large, solitary juvenile rectal polyp. The case provides a classic illustration of the clinical presentation, the diagnostic process, the standard surgical management, and a notable post-operative complication. In line with the user's specific educational objective, this report will also leverage the presentation of a pediatric mass to provide an exhaustive, contemporary review of **desmoid-type fibromatosis**. Although this rare soft tissue tumor was not the diagnosis in this case, it represents a challenging clinical entity with a rapidly evolving management paradigm, making a detailed discussion highly relevant for clinicians encountering pediatric neoplasms.

CASE ILLUSTRATION

Patient Information and Chief Complaint

The patient was a 1-year and 7-month-old (19-month-old) male who was brought to the emergency department by his parents. The chief complaints were a prolapsed, bleeding mass from the anus and a seven-day history of frequent, bloody stools.

History of Present Illness

The patient's symptoms began seven days prior to presentation with the onset of fresh, red blood passed per rectum. Over the subsequent days, the bleeding worsened significantly. For the four days immediately preceding admission, the patient was having approximately ten bloody bowel movements per day. The parents noted that the patient did not cry or appear to be in pain during defecation.

One month prior to this acute presentation, there was an intermittent history of black, foul-smelling stools mixed with mucus. The patient was evaluated by a pediatrician at that time and was prescribed medication, which did not lead to improvement.

The day before admission, a red lump was noted at the anus. This occurred shortly after the patient was discharged from a one-day hospitalization at another facility for the same complaints. The lump was initially small but rapidly increased in size and became irreducible; manual attempts by the parents to reinsert it were unsuccessful. Concurrently, the patient was observed to be weak and had poor oral intake of both food and fluids. The parents denied any associated fever, vomiting, or abdominal pain.

Medical and Personal History

The patient's past medical history was significant for a recent one-day admission at another hospital for hematochezia, during which no definitive surgical or endoscopic intervention was

performed. He was born preterm via a spontaneous vaginal delivery. His growth and developmental milestones were appropriate for his age. The family history was notable only for the patient's father having undergone an appendectomy. There was no reported family history of polyposis syndromes, inflammatory bowel disease, or early-onset gastrointestinal cancers.

Physical Examination

Upon presentation, the patient appeared weak but was fully conscious and alert, with a Glasgow Coma Scale (GCS) score of 15. His vital signs were as follows: blood pressure of 96/67 mmHg, heart rate of 122 beats per minute, respiratory rate of 20 breaths per minute, and a temperature of 36.6°C. His body weight was 8.8 kg.

Systemic examination was largely unremarkable. His thorax was clear to auscultation with no retractions, wheezes, or rhonchi. The abdomen was soft, supple, and non-tender, with normal active bowel sounds. His extremities were warm with a brisk capillary refill time of less than two seconds and no pitting edema. The initial head and neck examination was documented as negative for anemia, icterus, cyanosis, or dyspnea; however, a subsequent note from the daily ward rounds documented conjunctival pallor, indicating anemia (+/+).

The local perianal examination was the most revealing. A prolapsed, pedunculated mass was seen protruding from the anal orifice (Figure 1). It was fresh red in color, with approximate dimensions of 3x2x2 cm. A digital rectal examination (DRE) confirmed the presence of blood within the rectal vault. The anal sphincter tone was competent, and the examination did not elicit pain.

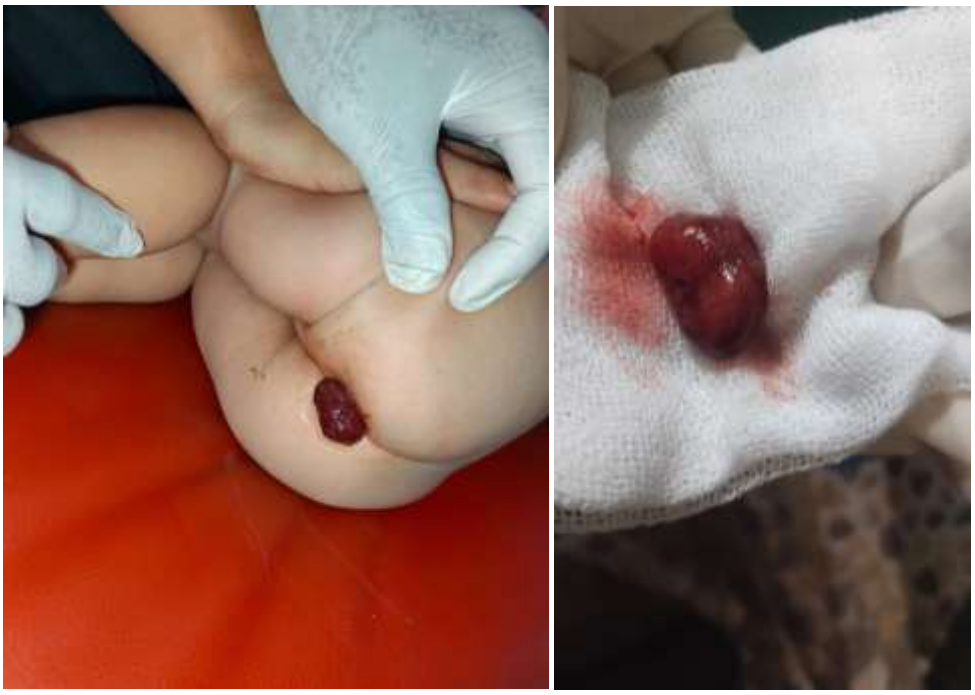

Figure 1. Clinical Presentation

Diagnostic Assessment

Laboratory Findings

Initial laboratory investigations were performed upon admission. The random blood glucose level was 108 mg/dL. While the source documentation did not include the results of the complete blood count (CBC), the clinical picture of prolonged bleeding, weakness, and noted conjunctival pallor was highly suggestive of significant anemia. For a comprehensive understanding of the patient's hematological status and the impact of the post-operative hemorrhage, representative laboratory values reflecting this clinical course are presented in Table 1. These values illustrate a microcytic, hypochromic anemia consistent with chronic gastrointestinal blood loss at admission, followed by an acute drop in hemoglobin after the post-operative bleed.

Table 1: Representative Laboratory Findings

Parameter	Result (24/04/2025 - Admission)	Result (26/04/2025 - Post-op Bleed)	Reference Range
Hemoglobin (g/dL)	8.5	6.8	10.5 - 13.5
Hematocrit (%)	26.0	21.5	33.0 - 39.0
Mean Corpuscular Volume (MCV) (fL)	72.0	71.5	75.0 - 87.0
Mean Corpuscular Hemoglobin (MCH) (pg)	23.5	23.0	25.0 - 31.0
Platelets (x109/L)	350	380	150 - 450
White Blood Cell Count (x109/L)	9.5	11.0	6.0 - 17.5
Random Blood Glucose (mg/dL)	108	N/A	70 - 140

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Histopathology

The excised surgical specimen was submitted for anatomical pathology examination. The findings were definitive for a benign juvenile polyp.

Histopathology Report:

- **Gross Description:** The specimen consisted of a single, pedunculated, reddish-tan, polypoid tissue measuring 3.1 x 2.2 x 2.0 cm. The surface was smooth and glistening. The stalk measured 0.8 cm in length.
- **Microscopic Description:** Histological sections revealed a polypoid structure characterized by cystically dilated colonic glands, which were filled with abundant mucin and inflammatory debris. The lamina propria was markedly expanded and edematous, containing a dense, mixed inflammatory cell infiltrate composed of eosinophils, plasma cells, and lymphocytes. The surface epithelium showed areas of focal erosion and was replaced by granulation tissue, consistent with mechanical trauma and inflammation. Crucially, there was no evidence of adenomatous change, dysplasia, or malignancy.
- **Final Diagnosis:** Colon, Rectum, Polypectomy: Hamartomatous Polyp (Juvenile Polyp).

Clinical Course, Management, and Follow-Up

The patient was admitted to the pediatric ward with a working diagnosis of hematochezia, with the primary differential diagnoses being a prolapsed hemorrhoid versus a rectal polyp. Initial management focused on stabilization and preparation for surgical intervention. He received intravenous fluid resuscitation with Dextrose 5% in 0.45% Normal Saline (D5 1/2NS) and was administered intravenous Santagesic (Metamizole) for analgesia and Tranexamic Acid to mitigate bleeding.

Following a consultation with the surgical service, the patient was taken to the operating room on the day of admission (24/04/2025) and underwent a transanal excision of the prolapsed rectal polyp under general anesthesia. The procedure was successful and without immediate

complications.

The patient's post-operative course was eventful and is summarized in Table 2. He was initially stable on post-operative day one, with resolution of bleeding and plans made for discharge. However, on post-operative day two, he experienced a significant hemorrhage, presenting with multiple episodes of fresh rectal bleeding, weakness, and tachycardia. This prompted the cancellation of his discharge, initiation of intravenous tranexamic acid, and urgent re-evaluation of his hematological status. The bleeding subsided by the following day, and he was successfully discharged home on post-operative day three with a course of oral antibiotics (Cefixime) and analgesics (Paracetamol).

Table 2: Summary of Hospital Course and Management

Date	Subjective (S) & Objective (O) Findings	Assessment (A)	Plan (P)
24/04/2025	**S:** Prolapsed anal mass, fresh red bleeding, mucus. **O:** Pulse: 104/min, Temp: 36.4°C, Anemic (+/+).	Hematochezia; Hemorrhoid vs. Rectal Polyp.	Proceed with operative excision + pathological analysis. IV D5 1/2NS 900 mL/24h. Inj. Santagesic 100mg TID. Inj. Anbacym 150mg TID.
25/04/2025	**S:** Post-operative	Post-polypectomy	Plan for discharge (KRS)

Date	Subjective (S) & Objective (O) Findings	Assessment (A)	Plan (P)
	pain, no further bleeding from anus. **O:** Pulse: 102/min, Temp: 36.2°C, No active bleeding.	status, stable.	tomorrow. Prescribe Cefixime syrup 1 tsp BID & Paracetamol syrup 1 tsp TID.
26/04/2025	**S:** Recurrent bleeding from anus (5 episodes, approx. 70cc fresh blood), no stool. Patient is weak. **O:** Pulse: 136/min (tachycardic), Temp: 36.0°C. Active bleeding on rectal exam.	Post-polypectomy hemorrhage.	**Discharge cancelled (Tunda KRS).** Administer Inj. Tranexamic acid 150mg TID. Repeat complete blood count.
27/04/2025	**S:** No further bleeding reported. **O:** Pulse: 110/min,	Hemorrhage resolved. Patient stable.	**Cleared for Discharge (KRS).** Continue oral Cefixime and

Date	Subjective (S) & Objective (O) Findings	Assessment (A)	Plan (P)
	Temp: 36.1°C. No blood on rectal exam.		Paracetamol as prescribed.

DISCUSSION

Diagnosis and Management of Solitary Juvenile Polyp

The diagnosis in this case was unequivocally that of a solitary juvenile polyp, confirmed by histopathology. Juvenile polyps are the single most common cause of significant lower gastrointestinal bleeding in children, and they account for over 90% of all colonic polyps found in this age group.[1] The clinical presentation exhibited by this patient—painless, bright red rectal bleeding, often culminating in the prolapse of a mass through the anus—is considered pathognomonic for a distally located pedunculated polyp.[1] The intermittent history of dark, foul-smelling stools may suggest a more proximal lesion or intermittent bleeding with altered blood, but the final presentation pointed clearly to a rectal source.

The management strategy employed was the standard of care. Removal of the polyp, in this case via transanal excision due to its prolapsed and accessible nature, is both diagnostic and therapeutic.[1] This approach provides tissue for definitive histological diagnosis, which is critical to rule out dysplasia or features of a polyposis syndrome, and it permanently resolves the source of bleeding.

Post-polypectomy hemorrhage, as occurred on the second post-operative day, is a

recognized complication. The incidence varies, but it can be a source of significant morbidity. The patient's clinical deterioration, marked by weakness and tachycardia (pulse 136 bpm), indicated a hemodynamically significant bleed requiring prompt intervention. The successful management with intravenous tranexamic acid and supportive care highlights a conservative approach that can often avert the need for endoscopic or surgical re-intervention.

Given the final diagnosis of a single hamartomatous polyp with no dysplastic features, and in the absence of a family history of polyposis or multiple polyps, no further endoscopic surveillance is recommended. The prognosis for a solitary juvenile polyp is excellent, with polypectomy being curative.[1]

Comprehensive Review: Desmoid-Type Fibromatosis (Aggressive Fibromatosis)

While the present case involved a common, benign mucosal lesion, the initial presentation of a "mass" in a pediatric patient necessitates a broad differential diagnosis. This differential includes rare but clinically significant soft tissue neoplasms that can pose considerable diagnostic and therapeutic challenges. Among these, desmoid-type fibromatosis represents a unique and complex entity whose management has undergone a profound evolution. Its characteristics merit a detailed review for the practicing clinician.

Definition and Classification

Desmoid-type fibromatosis (DTF), also commonly known as a desmoid tumor or aggressive fibromatosis, is a rare neoplasm composed of a clonal proliferation of myofibroblastic spindle cells that arises from deep soft tissues, such as muscle and fascia.[6]

The tumor occupies a paradoxical space in oncology. The World Health Organization (WHO) classifies DTF as a tumor of "intermediate (locally aggressive)" potential.[7] Histologically, its cells are bland, with low mitotic activity and no features of cytological malignancy, rendering it technically "benign." Furthermore, it is incapable of metastasis, meaning it does not spread to

distant sites like the lungs or liver.[6] However, its clinical behavior can be profoundly "malignant." DTF is characterized by a relentless, infiltrative local growth pattern, invading and destroying adjacent tissues and vital structures. This local aggression, combined with a very high rate of recurrence after surgical resection, can lead to severe morbidity, functional impairment, and, in some cases, mortality, particularly when critical organs are involved.[7] This disconnect between its benign histology and aggressive clinical course is the central challenge in its management and in counseling patients.

DTF is typically classified based on its anatomical location, a system that has both prognostic and therapeutic relevance [9]:

- **Extra-abdominal:** Arising in the extremities (shoulder and pelvic girdles), chest wall, or head and neck region.
- **Abdominal wall:** Originating from the musculoaponeurotic structures of the anterior abdominal wall, often associated with pregnancy.
- **Intra-abdominal:** Developing within the abdominal cavity, most commonly in the mesentery or retroperitoneum, and strongly associated with Familial Adenomatous Polyposis (FAP).

Epidemiology

DTF is a rare disease. The reported incidence is estimated to be between 2 to 4 cases per million people per year, which accounts for a mere 0.03% of all neoplasms.[6] There is a clear female predilection, with incidence rates being two to five times higher in women than in men. The peak age of onset is in young adulthood, typically between the ages of 15 and 60, with a median age at diagnosis in the 30s.[6] While it can occur in children, it is less common. A crucial epidemiological link exists with the genetic syndrome Familial Adenomatous Polyposis (FAP); approximately 5% to 10% of all DTF cases occur in patients with FAP, who have a risk of developing the tumor that is over 800 times that of the general population.[6]

Pathogenesis and Molecular Biology

The pathogenesis of virtually all desmoid tumors converges on a single molecular pathway: the dysregulation of the Wnt/β-catenin signaling cascade, which results in the abnormal accumulation of the β-catenin protein in the cell nucleus.[6] In the nucleus, β-catenin acts as a transcriptional co-activator, driving the expression of genes that promote cell proliferation and survival. This pathogenic activation occurs through one of two mutually exclusive genetic events:

1. **Sporadic DTF (~85% of cases):** These tumors are caused by somatic mutations in the *CTNNB1* gene, which is the gene that directly codes for the β-catenin protein. These mutations typically occur in exon 3 and prevent the protein from being marked for degradation, leading to its stabilization and accumulation. Specific *CTNNB1* mutations have been shown to have prognostic significance; for instance, the S45F mutation is associated with a significantly higher risk of recurrence following surgery compared to the T41A mutation.[6]
2. **FAP-Associated DTF (~10-15% of cases):** In these patients, the underlying cause is a germline mutation in the *Adenomatous Polyposis Coli (APC)* tumor suppressor gene.[7] The APC protein is a key component of a "destruction complex" that normally targets β-catenin for degradation. When the *APC* gene is mutated and the protein is non-functional, the destruction complex fails, leading to the same endpoint of β-catenin accumulation in the nucleus.

In addition to these core genetic drivers, other factors are known to contribute to tumorigenesis:

- **Trauma and Surgery:** A history of physical trauma or, more commonly, surgical intervention at the site of tumor development is a well-established risk factor.[6] This association is particularly strong in patients with FAP.
- **Hormonal Factors:** The increased incidence in women, especially the development of abdominal wall desmoids during or immediately following pregnancy, strongly suggests a role for estrogen in promoting tumor growth. Many desmoid tumors express estrogen receptors, providing a biological basis for this observation.[6]

The relationship between FAP, surgery, and desmoid tumors creates a profound clinical

dilemma. FAP is a condition caused by a germline *APC* mutation that leads to the development of hundreds to thousands of colorectal adenomas with an almost 100% lifetime risk of colorectal cancer. The standard of care to prevent this cancer is a prophylactic colectomy. However, this life-saving surgery itself acts as the most potent trigger for the development of intra-abdominal desmoid tumors in these genetically predisposed individuals. Consequently, the very treatment for one manifestation of the syndrome can precipitate another, often more morbid and difficult-to-treat, manifestation. This creates a vicious cycle that complicates surgical decision-making, particularly regarding the timing of colectomy in young FAP patients.

Clinical Presentation and Diagnosis

Patients with DTF typically present with a slow-growing, firm, and often painless or minimally painful palpable mass.[10] Symptoms, when they occur, are entirely dependent on the tumor's location and its compression or infiltration of adjacent structures. For example, intra-abdominal tumors can cause bowel obstruction or hydronephrosis, while extremity tumors can lead to pain, nerve deficits, and limb contractures.[8]

The diagnostic workup involves a combination of imaging and tissue sampling:

- **Imaging:** Magnetic Resonance Imaging (MRI) is the preferred imaging modality. Its excellent soft-tissue contrast is invaluable for delineating the full extent of the tumor and its relationship to surrounding fascia, muscle, neurovascular bundles, and bone.[6] Computed Tomography (CT) is also frequently used, especially for evaluating intra-abdominal and chest wall tumors.
- **Biopsy:** A definitive diagnosis requires a tissue sample. A core needle biopsy is often sufficient, but an incisional biopsy may be necessary to obtain adequate tissue for diagnosis.[13]
- **Histopathology:** The microscopic hallmark of DTF is a poorly circumscribed, infiltrative proliferation of bland, uniform spindle cells arranged in long, sweeping fascicles within a variably collagenous stroma.[6] The cells have minimal nuclear atypia and low mitotic activity.
- **Immunohistochemistry:** Nuclear staining for β-catenin is the key ancillary test that confirms

the diagnosis. It is positive in the vast majority of cases (up to 98%) and helps to differentiate DTF from its histologic mimics, such as low-grade fibrosarcoma or scar tissue.[6]

Contemporary Management Strategies

The management of DTF has undergone a dramatic paradigm shift in the last decade. Historically, these tumors were treated like soft tissue sarcomas, with aggressive upfront surgical resection being the standard of care. However, this approach was plagued by extremely high local recurrence rates (up to 65%), even after achieving surgically clear margins, and often resulted in substantial functional and cosmetic morbidity from repeated operations.[11] A better understanding of the tumor's natural history, including the observation that a significant proportion of tumors may remain stable or even spontaneously regress, has led to a major de-escalation of treatment.[8] The current approach is multidisciplinary and tiered:

1. **Active Surveillance ("Watch and Wait"):** This is now the recommended initial management strategy for the majority of newly diagnosed patients who are asymptomatic or have minimal symptoms, irrespective of tumor location.[6] This strategy involves regular clinical follow-up and serial imaging (usually MRI) every 3-6 months to monitor the tumor's behavior. Intervention is deferred unless there is clear evidence of disease progression that is causing significant symptoms or threatening vital structures.
2. **Systemic Therapies:** For patients with tumors that progress on active surveillance, are symptomatic, or are unresectable, systemic therapy is now a primary treatment modality.
    - **Chemotherapy:** Low-dose regimens (e.g., methotrexate and vinblastine) or conventional cytotoxic agents (e.g., doxorubicin-based regimens) may be used for rapidly progressive or life-threatening disease.[6]
    - **Targeted Therapy:** This area has seen the most significant advances.
        - *Tyrosine Kinase Inhibitors (TKIs):* Sorafenib demonstrated a significant improvement in progression-free survival in a landmark phase III clinical trial and is a widely used option.[7] Other TKIs like pazopanib are also used.

- *Gamma-Secretase Inhibitors (GSIs):* Nirogacestat is the first and only FDA-approved therapy specifically for progressing desmoid tumors. It targets the Notch signaling pathway, which has crosstalk with the Wnt/β-catenin pathway. In its pivotal phase III trial, nirogacestat significantly improved progression-free survival, objective response rates, and patient-reported outcomes such as pain and quality of life.[6]

- **Hormonal Therapy and NSAIDs:** Agents like tamoxifen and non-steroidal anti-inflammatory drugs (NSAIDs) were historically used but are now considered to have limited efficacy and are not recommended as first-line systemic options for progressing disease.[6]

3. **Surgery:** The role of surgery has been relegated from a first-line to a secondary or tertiary option. It is now reserved for specific clinical scenarios, such as debilitating symptoms (e.g., intractable pain, bowel obstruction) where a complete resection can be achieved with acceptable morbidity, or for disease that has failed to respond to systemic therapies.[17] The goal remains complete removal, but mutilating surgeries solely to achieve negative microscopic margins are strongly discouraged.[6]
4. **Locoregional Therapies:**
   - **Radiation Therapy:** This can be an effective option for achieving local control in unresectable tumors or as an adjuvant treatment after a margin-positive resection. However, its use is tempered by concerns about long-term toxicity, including fibrosis, fracture, and the risk of radiation-induced secondary malignancies, particularly in younger patients.[16]
   - **Ablative Therapies:** Techniques such as cryoablation (freezing the tumor) and High-Intensity Focused Ultrasound (HIFU) are emerging as minimally invasive options for local control of smaller, accessible tumors.[17]

Complications and Prognosis

The complications of DTF are a direct result of its infiltrative growth and are highly

dependent on the tumor's location.[8] Intra-abdominal tumors can cause life-threatening complications like bowel obstruction, fistula formation, ureteral obstruction leading to renal failure, or compression of major mesenteric vessels. Extra-abdominal tumors can cause severe pain from nerve entrapment, joint contractures limiting mobility, and significant cosmetic deformity. Head and neck tumors are particularly dangerous due to the risk of airway compromise or cranial nerve palsies.

The prognosis in terms of overall survival is excellent for patients with sporadic DTF, as the disease does not metastasize. However, the morbidity can be substantial and lifelong. For patients with FAP-associated intra-abdominal DTF, the prognosis is more guarded, and DTF is a leading cause of mortality in this population due to its severe local complications.[8]

Risk Factors for Recurrence and Management of Recurrent Disease

Local recurrence after surgery is a defining feature of DTF, with historical rates ranging from 25% to as high as 65%.[11] Several factors have been identified as predictors of a higher risk of recurrence:

- **Younger age** at diagnosis
- **Large tumor size** (typically >5-7 cm)
- **Extra-abdominal location** (especially tumors of the extremities)
- **Positive surgical margins**
- **Recurrent presentation** (vs. primary)
- **Specific *CTNNB1* mutation status** (S45F) [8]

The management of recurrent disease follows the same principles as primary disease, with an even greater emphasis on avoiding further morbid surgery. Active surveillance is often the initial approach for an asymptomatic recurrence. If the recurrence is progressive and symptomatic, systemic therapies (such as nirogacestat or sorafenib) or radiation therapy are generally preferred over re-operation. All cases of recurrent DTF warrant discussion in a multidisciplinary tumor board

with expertise in sarcoma and desmoid tumors to determine the optimal treatment sequence.[6]

CONCLUSION

This report presents the successful management of a solitary juvenile polyp in a 19-month-old male, a common and benign cause of significant rectal bleeding in childhood. The case illustrates a classic clinical presentation, standard surgical treatment, and the importance of vigilance for post-operative complications such as hemorrhage. Simple polypectomy is curative for such lesions, with an excellent long-term prognosis.

The comprehensive review of desmoid-type fibromatosis provided herein serves a critical educational purpose, highlighting the profound evolution in the understanding and treatment of this rare and challenging neoplasm. The paradigm has decisively shifted from a historically aggressive surgical model to a more nuanced, patient-centered, and multidisciplinary approach that prioritizes initial active surveillance. The advent of effective systemic agents, particularly targeted therapies like nirogacestat, has further transformed the treatment landscape for patients with progressive or symptomatic disease. This evolution reflects a broader trend in modern oncology towards balancing disease control with the preservation of function and long-term quality of life—a principle of paramount importance when managing these non-metastasizing yet locally destructive tumors.

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