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Isolated Pediatric Renal Vein Thrombosis Presenting as Acute Flank Pain

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Description

Renal Vein Thrombosis (RVT) in children is an uncommon but clinically significant vascular event that can result in renal dysfunction, hypertension, and long-term nephron loss. The condition involves partial or complete occlusion of the renal venous outflow by thrombus formation, leading to venous congestion, ischemic injury, and, in some cases, infarction of renal parenchyma. Although it is well recognized in neonates, often secondary to perinatal asphyxia, dehydration, or catheter-related trauma, isolated renal vein thrombosis beyond the neonatal period is exceedingly rare. When it does occur, the presentation may be atypical and mimic other causes of acute flank pain, such as renal colic, pyelonephritis, or ureteral obstruction. Understanding its pathophysiology, diagnostic challenges, and management options is crucial to prevent permanent renal damage.

In the pediatric population, renal vein thrombosis may arise as a complication of systemic disorders that promote a hypercoagulable state. These include inherited thrombophilias such as factor V Leiden mutation, prothrombin gene mutation, protein C or S deficiency, and antithrombin III deficiency. Acquired

risk factors, including dehydration, sepsis, nephrotic syndrome, systemic lupus erythematosus, and trauma, also contribute to thrombogenesis. In some children, particularly those presenting with isolated RVT, no identifiable systemic disorder is found, and the event is termed idiopathic. The left renal vein is slightly more vulnerable due to its longer course and possible compression between the superior mesenteric artery and aorta, a phenomenon known as the nutcracker effect, which can predispose to stasis and thrombosis.

The pathophysiology of RVT is best understood through Virchow's triad-venous stasis, endothelial injury, and hypercoagulability. In children, venous stasis may result from dehydration or extrinsic compression; endothelial injury may be secondary to infection, catheterization, or local inflammation; and hypercoagulability may be inherited or acquired. When these factors coexist, thrombus formation occurs within the renal vein, leading to increased venous pressure, renal congestion, and reduced glomerular filtration. Over time, ischemic injury can cause cortical necrosis, atrophy, and chronic renal impairment.

Clinically, isolated renal vein thrombosis in children beyond infancy often presents as acute flank or abdominal pain of sudden onset. The pain is typically localized to one side and may be accompanied by gross or microscopic hematuria. Nausea, vomiting, and mild fever can occur, leading to initial misdiagnosis as pyelonephritis or renal calculi. In contrast to neonatal RVT, where palpable flank mass and hematuria are classical findings, older children usually exhibit subtler signs. Hypertension may develop secondary to activation of the renin-angiotensin system due to renal ischemia. Some cases are discovered incidentally

during imaging for unrelated reasons, especially when the thrombus is non-occlusive. Because of its rarity and nonspecific presentation, delayed diagnosis is common, potentially leading to irreversible renal injury.

Diagnostic evaluation requires a high index of suspicion and a multimodal imaging approach. Ultrasonography with color Doppler is the preferred initial investigation, as it is non-invasive, widely available, and radiation-free. Findings may include an enlarged kidney with reduced cortical echogenicity, absent or reversed venous flow, and echogenic thrombus within the renal vein. In equivocal cases, contrast-enhanced computed tomography or magnetic resonance venography provides superior anatomical detail, demonstrating the filling defect within the renal vein and, occasionally, extension into the inferior vena cava. Laboratory investigations often reveal hematuria, mild proteinuria, and elevated lactate dehydrogenase levels. Renal function tests may show elevated serum creatinine, particularly in bilateral or extensive thrombosis. Screening for thrombophilic disorders should be undertaken in all children to identify inherited or acquired risk factors that may influence therapy duration and recurrence prevention.

The management of isolated pediatric renal vein thrombosis depends on the extent of thrombus, presence of bilateral involvement, renal function status, and underlying etiology. The goals of therapy are to preserve renal function, prevent thrombus propagation, and minimize long-term sequelae. Anticoagulation

forms the mainstay of treatment in most cases. Initial therapy typically involves unfractionated heparin or Low-Molecular-Weight Heparin (LMWH) to achieve prompt anticoagulation, followed by transition to an oral agent such as warfarin for a total duration of three to six months. LMWH is often preferred in children because of its predictable pharmacokinetics, ease of administration, and lower risk of heparin-induced thrombocytopenia. Anticoagulation has been shown to reduce thrombus extension and improve renal outcomes without significantly increasing the risk of major bleeding when appropriately monitored.

Conclusion

Isolated pediatric renal vein thrombosis presenting as acute flank pain is a rare but important diagnostic consideration in children with sudden-onset abdominal or flank discomfort, hematuria, or unexplained renal dysfunction. Prompt recognition and appropriate imaging are critical to differentiate it from more common causes such as infection or calculi. Anticoagulation remains the cornerstone of therapy and is usually sufficient to prevent extension and preserve renal function. Long-term follow-up is necessary to monitor renal health, screen for recurrence, and manage any underlying predisposition. Early diagnosis, multidisciplinary collaboration, and continued surveillance together form the foundation for favorable outcomes in this uncommon but potentially serious pediatric vascular disorder.