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Iterprofessional Clinical Management of Renal Infarction-An Integrated Nursing, Pharmacy, and Laboratory Approach

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Abstract

Background: Renal infarction is an uncommon but clinically significant ischemic condition caused by obstruction of the renal arterial system, often linked to cardioembolic disorders, thrombosis, hypercoagulable states, and traumatic or iatrogenic vascular injury. Its nonspecific symptoms frequently lead to misdiagnosis, delaying timely intervention.

Aim: This study aims to provide an integrated, interprofessional overview of the etiology, clinical presentation, diagnostic approach, and management strategies of renal infarction involving nursing, pharmacy, and laboratory perspectives.

Methods: A comprehensive analysis of current literature and multidisciplinary clinical practices provides an evidence-based synthesis of epidemiology, pathophysiology, diagnostic modalities, and therapeutic interventions, emphasizing interprofessional collaboration.

Results: Renal infarction most commonly results from cardioembolic events, particularly atrial fibrillation, followed by thrombosis, aortic dissection, trauma, hypercoagulable states, and atheroembolic disease. Diagnosis relies on high clinical suspicion, elevated LDH, urinalysis abnormalities, and contrast-enhanced CT imaging. Management includes catheter-directed thrombolysis, anticoagulation, surgical interventions when indicated, and long-term secondary prevention through risk-factor control.

Conclusion: Early recognition and coordinated interprofessional management significantly improve renal and systemic outcomes. Timely diagnosis, targeted therapy, and ongoing patient education are essential to reducing morbidity and preventing recurrence.

Keywords: Renal infarction, thromboembolism, atheroembolism, diagnosis, CT angiography, anticoagulation, interprofessional care.

Introduction

Renal infarction represents an uncommon but clinically significant ischemic condition that arises from partial or complete obstruction of the main renal artery or its segmental branches, leading to impaired renal perfusion and parenchymal ischemia. In most reported cases, the underlying mechanism involves embolic events originating from the heart or thrombus formation within the renal vasculature itself [1]. Cardioembolic sources are particularly prominent, especially in patients with arrhythmias or structural heart disease. Additional etiologies include aortic thromboembolism, blunt or penetrating trauma, spontaneous or traumatic renal artery dissection, inherited or acquired coagulation abnormalities, and atheroembolic disease associated with advanced vascular pathology. Clinically, renal infarction should be suspected in patients who present with the abrupt onset of flank or abdominal pain accompanied by laboratory or functional evidence of renal impairment. Supporting findings often include elevated lactate dehydrogenase levels, hematuria, and proteinuria in the absence of urinary tract obstruction or other identifiable causes. Diagnostic suspicion is further

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strengthened in older individuals, particularly those over 60 years of age, and in patients with preexisting cardiac conditions such as atrial fibrillation. Initial diagnostic evaluation commonly begins with a noncontrast abdominal computed tomography scan to exclude more frequent causes of acute abdominal pain, followed by contrast-enhanced imaging when initial findings are inconclusive [2][3]. Timely identification is critical, as early intervention allows consideration of revascularization strategies that may preserve renal function. Therapeutic management depends on the clinical context and includes intravascular or systemic thrombolysis, anticoagulant therapy, and antiplatelet agents. The extent of renal recovery is influenced by several factors, including the duration of ischemia, the degree of arterial occlusion, patient comorbidities, and the promptness of treatment initiation. Partial occlusions and early intervention are associated with more favorable outcomes. Despite its clinical importance, renal infarction is frequently underdiagnosed due to symptom overlap with more prevalent conditions such as nephrolithiasis or acute pyelonephritis. This diagnostic challenge is reflected in autopsy studies, which demonstrate a higher incidence of renal infarction than that reported in clinical case series. Delayed or missed diagnosis can result in permanent renal damage, as reperfusion therapies offer limited benefit once prolonged ischemia has occurred [4][5].

Etiology

Renal infarction results from a heterogeneous group of pathological processes that ultimately compromise renal arterial blood flow, leading to ischemic injury of renal parenchyma. These causes can be broadly categorized into cardioembolic sources, intrinsic renal artery thrombosis, traumatic or mechanical injury, aortic or renal artery dissection, hypercoagulable states, and atheroembolic disease [6][7][8][9][10]. Understanding these etiological mechanisms is essential for accurate diagnosis, targeted management, and prevention of recurrent events, as each category is associated with distinct risk factors, clinical contexts, and therapeutic implications. Cardioembolic disorders constitute the most prevalent cause of renal infarction, accounting for up to 55% of reported cases. Within this group, atrial fibrillation represents the dominant underlying condition, identified in approximately 64% to 75% of affected patients [7]. In some individuals, renal infarction may serve as the initial clinical manifestation of previously undiagnosed atrial fibrillation. Furthermore, many patients with known atrial fibrillation who develop renal infarction are found to have inadequate anticoagulation, highlighting the critical role of optimal therapeutic monitoring. Cardioembolic renal infarction predominantly affects older adults, most commonly those in the sixth to eighth decades of life. Additional cardiac sources of emboli include rheumatic mitral valve disease, recent myocardial infarction with mural thrombus formation, and infective endocarditis involving either native or prosthetic valves. Less frequently, emboli may arise from intracardiac tumors such as atrial myxomas or from thrombi located in the left ventricular apex, particularly in patients with impaired ventricular contractility [11]. Renal artery thrombosis represents another important arterial cause of renal infarction and is often associated with underlying atherosclerotic disease. Thrombotic occlusion may originate within the renal artery itself or extend from the abdominal aorta. Advanced atherosclerosis promotes endothelial injury and local thrombogenesis, increasing the risk of acute arterial obstruction. In addition, aneurysms of the thoracic or suprarenal abdominal aorta create areas of altered blood flow that favor thrombus formation, thereby increasing the likelihood of embolic or thrombotic renal infarction. Management in such cases emphasizes aggressive control of modifiable cardiovascular risk factors, including hyperlipidemia, hypertension, and smoking cessation, alongside appropriate antithrombotic therapy [12].

Trauma or mechanical injury is a less common but clinically significant cause of renal infarction. Blunt abdominal trauma may result in renal artery dissection, thrombosis, or external compression due to retroperitoneal hemorrhage. These mechanisms can acutely impair renal perfusion, leading to segmental or global renal ischemia. Iatrogenic injury may also occur during abdominal or retroperitoneal surgical procedures. The choice between conservative management, endovascular intervention, or open surgical repair depends on the severity of vascular injury, hemodynamic stability, and the presence of associated injuries. Aortic or renal artery dissection represents another distinct etiological category. Mechanical instrumentation is the most frequent precipitating factor, particularly during endovascular or open surgical procedures involving the aorta or its major branches. Aortic dissection may extend into one or both renal arteries, resulting in compromised renal blood flow. Similarly, endovascular stent placement during aortic repair can inadvertently affect renal artery patency. Although rare, spontaneous renal artery dissection can occur and may lead to severe renal ischemia even in individuals without significant comorbidities. Clinical decision-making in these cases requires individualized assessment, with careful consideration of surgical versus nonsurgical approaches based on anatomical findings, renal function, and overall patient risk [13][14]. Fibromuscular dysplasia represents a unique vascular disorder that predisposes to renal artery involvement and subsequent renal infarction. This condition predominantly affects women younger than 50 years and involves the renal arteries in approximately half of cases, often bilaterally [15]. Fibromuscular dysplasia affects small- and medium-sized arteries, leading to stenosis, aneurysm formation, or arterial dissection. Renal infarction may occur as a result of thrombus formation within poststenotic dilated arterial

segments, with hypertension frequently serving as the initial clinical manifestation [16][17]. Spontaneous renal artery dissection has also been reported in association with fibromuscular dysplasia, as well as in connective tissue disorders such as Marfan syndrome and Ehlers–Danlos syndrome [19][20]. These associations underscore the importance of considering underlying systemic vascular disorders in patients presenting with renal infarction at a young age. Further details regarding fibromuscular dysplasia are discussed in dedicated references [18].

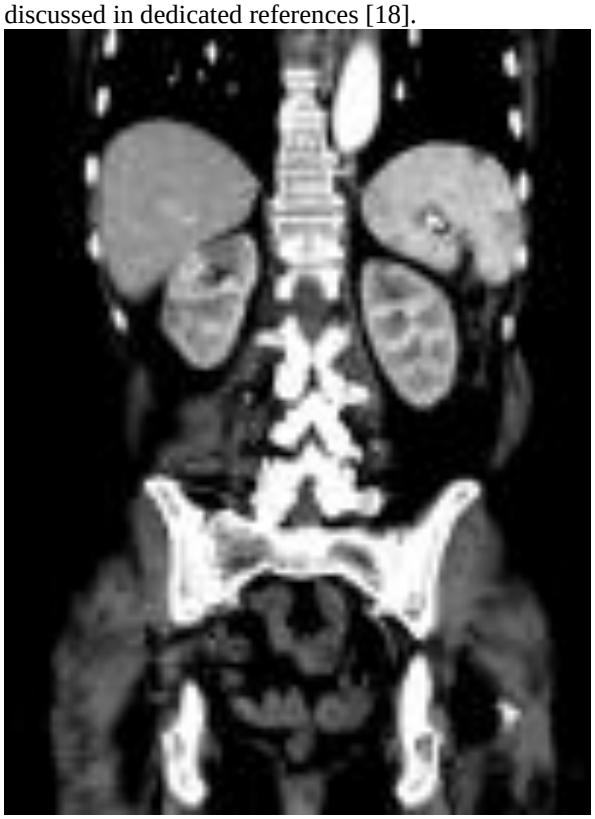

Fig. 1: CT imaging of a patient with renal infarction.

Hypercoagulable states constitute another major etiological group and include both inherited and acquired conditions that predispose to arterial thrombosis. Heritable disorders such as protein C deficiency, protein S deficiency, antithrombin III deficiency, Factor V Leiden mutation, prothrombin gene mutation, and hyperhomocysteinemia have all been implicated in renal artery occlusion. Acquired hypercoagulable conditions include systemic lupus erythematosus, polycythemia vera, malignancy, and sickle cell disease. Renal infarction has also been reported in association with oral contraceptive use, anabolic steroid abuse, cocaine use, and COVID-19 infection, reflecting the prothrombotic milieu associated with these conditions [21][22]. Identification of an underlying hypercoagulable state has important implications for long-term anticoagulation and secondary prevention. Atheroembolic disease represents a distinct mechanism of renal infarction that is particularly relevant in older patients with advanced atherosclerosis. Many individuals undergoing angiographic procedures have significant atherosclerotic plaque burden, placing them at increased risk for cholesterol crystal embolization. These emboli may occlude small renal arteries and arterioles, resulting in renal ischemia and infarction. The incidence of acute kidney injury due to biopsy-confirmed renal atheroemboli in patients older than 60 years has been reported at approximately 7%. Procedural strategies, such as using a brachial rather than an iliofemoral arterial access route, may reduce this risk [23][24]. Unlike thromboembolic renal infarction, atheroembolic disease often presents with a delayed and insidious decline in renal function, sometimes weeks after the inciting event [25]. Symptoms tend to be less severe because atheroemboli are irregularly shaped and rarely cause complete arterial occlusion [26]. Progressive luminal narrowing and cumulative ischemic injury over time contribute to gradual renal dysfunction [27][28]. Despite comprehensive diagnostic evaluation, a substantial proportion of renal infarction cases remain idiopathic. Approximately 20% to 30% of patients have no clearly identifiable cause, with a median age of around 50 years in this subgroup [6][7][10]. Idiopathic renal infarction may reflect underlying subclinical vascular pathology or generalized endothelial dysfunction. In such cases, aggressive management of secondary risk factors, including hypertension, dyslipidemia, and diabetes mellitus, is strongly recommended to reduce the risk of recurrence [29]. Evidence suggests that patients with idiopathic renal infarction are more likely to be younger and active smokers, indicating potential targets for focused preventive strategies [30].

Epidemiology

Renal infarction is considered an uncommon clinical diagnosis; however, available epidemiological data suggest that its true incidence is likely underestimated. Evidence derived from autopsy studies indicates an incidence of approximately 14 cases per 1000 individuals, corresponding to nearly 1.4% of examined populations [6]. In contrast, retrospective analyses of emergency department admissions report substantially lower incidence rates, ranging from 0.004% to 0.007% [30][31]. This marked discrepancy reflects the diagnostic challenges associated with renal infarction, as many cases remain unrecognized or are incorrectly attributed to more common causes of acute abdominal or flank pain. As a result, autopsy-based data may provide a more accurate representation of the actual burden of disease [30][32]. Patterns of renal involvement further characterize the epidemiology of renal infarction. Unilateral renal infarction has been reported in the majority of patients, accounting for approximately 81% of cases, while bilateral renal involvement occurs in about 19% [9]. Bilateral or multiple infarctions appear to be more frequent among patients with underlying hypercoagulable conditions, suggesting a systemic predisposition to widespread arterial thrombosis rather than isolated embolic events [6].

These observations underscore the importance of considering coagulation abnormalities when extensive renal involvement is identified. The demographic profile of patients affected by renal infarction indicates a predominance in older adults. The mean age at presentation has been estimated at around 63 years, reflecting the accumulation of cardiovascular and thromboembolic risk factors with advancing age [9][19]. Studies have not demonstrated a significant difference in incidence between males and females, suggesting that sex alone does not play a determining role in disease occurrence [9]. Instead, comorbid conditions appear to be more influential in shaping epidemiological trends. Several well-established risk factors are consistently associated with renal infarction. Atrial fibrillation is among the most significant, given its strong association with systemic embolization. Additional risk factors include hypertension, diabetes mellitus, and a prior history of embolic infarction, all of which contribute to vascular injury, thrombogenesis, or impaired cardiac function [9][30]. The coexistence of these conditions increases both the likelihood of renal infarction and the risk of recurrent thromboembolic events. Collectively, epidemiological findings highlight renal infarction as an underdiagnosed condition that predominantly affects older individuals with cardiovascular or thromboembolic risk factors, reinforcing the need for heightened clinical awareness.

Pathophysiology

Renal infarction results from an interruption of renal arterial blood flow that leads to ischemic injury and irreversible tissue damage within the kidney. The underlying pathophysiological mechanisms are primarily thrombotic or embolic in nature. Thrombotic renal infarction develops when endothelial injury within the renal artery or its branches triggers platelet activation and coagulation cascades, resulting in in situ thrombus formation. In contrast, embolic infarction most commonly occurs when thromboemboli originating from the heart, particularly in the setting of atrial fibrillation, dislodging and travel through the arterial circulation until they lodge within the renal arterial system. Because renal segmental arteries are functional end arteries with limited collateral circulation, even partial obstruction can significantly compromise tissue perfusion, while complete occlusion leads to abrupt ischemia and focal necrosis of renal parenchyma. The ischemic insult initiates a cascade of cellular injury characterized by ATP depletion, membrane dysfunction, and inflammatory mediator release, ultimately resulting in coagulative necrosis of affected renal tissue. Loss of viable nephrons impairs glomerular filtration, clinically manifesting as elevated serum creatinine levels and reduced glomerular filtration rate. Depending on the extent and duration of ischemia, renal infarction may lead to acute kidney injury, progression to chronic kidney disease, or, in severe cases, end-stage renal failure. Infarction involving the main renal artery threatens the function of the entire kidney, whereas segmental arterial occlusion compromises a substantial portion of renal mass, which is particularly detrimental in patients with preexisting renal dysfunction or a solitary kidney. Occlusion of smaller subsegmental arteries produces the characteristic wedge-shaped perfusion defects observed on contrast-enhanced computed tomography imaging. Atheroembolic renal infarction follows a distinct pathophysiological process. Cholesterol emboli are typically smaller and more irregular than thromboemboli, making them less likely to cause complete arterial occlusion but more prone to lodging in smaller renal arterial branches [26]. Once embedded, cholesterol crystals elicit a foreign body inflammatory response with macrophage infiltration and giant cell formation. Progressive intimal thickening and luminal narrowing ensue, leading to chronic reductions in renal blood flow, renovascular hypertension, and gradual decline in glomerular filtration rate [27][28]. This delayed and progressive nature differentiates atheroembolic disease from the abrupt ischemia seen in thromboembolic infarction.

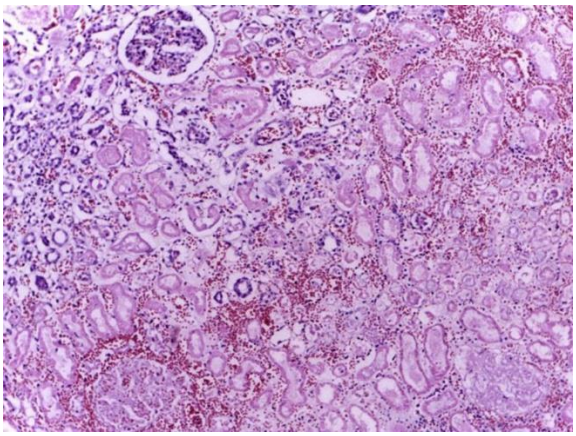

Fig. 2: Renal tissue necrosis.

History and Physical Clinical Features

The clinical presentation of acute renal infarction is variable and frequently nonspecific, contributing significantly to delayed or missed diagnosis. Most patients present with the sudden onset of abdominal or flank pain, often described as severe and persistent, and commonly accompanied by nausea, vomiting, hematuria, and, less frequently, low-grade fever. Physical examination may reveal localized abdominal or costovertebral angle tenderness, although findings are often subtle and nonspecific [5][6]. Cutaneous manifestations are observed in approximately one-third of cases, particularly in patients with atheroembolic disease, and include livedo reticularis or peripheral cyanotic changes such as blue toe syndrome [33][34]. The nonspecific nature of these symptoms often leads clinicians to attribute them to more common conditions, including ureteric colic, recently passed renal calculi, acute pyelonephritis, or gastrointestinal disorders. As a result, fewer than half of affected

patients receive an accurate diagnosis within the first 48 hours of presentation [35]. Laboratory evidence of renal dysfunction may not be immediately apparent, as signs of acute kidney injury can develop days to weeks after the initial ischemic event. Nevertheless, the combination of sudden flank or abdominal pain, markedly elevated lactate dehydrogenase levels, and the presence of hematuria or proteinuria should raise strong suspicion for renal infarction [31]. A high index of clinical suspicion is essential, particularly in patients with established vascular risk factors. These include advanced age, male sex, smoking history, hypertension, diabetes mellitus, and hypercholesterolemia. Atrial fibrillation remains one of the most significant risk factors, serving as a common source of systemic emboli that may precipitate renal infarction [36][37]. In suspected cases of atheroembolic disease, fundoscopic examination is recommended, as retinal cholesterol emboli, known as Hollenhorst plaques, may provide supportive diagnostic evidence [38]. Acute interruption of renal arterial perfusion may also lead to new-onset, renin-mediated hypertension at presentation, reflecting ischemia-induced activation of the renin–angiotensin–aldosterone system. This hypertension often improves with appropriate treatment but is frequently misattributed to pain or stress and therefore overlooked [4][8]. Some patients remain asymptomatic, and renal infarction may be identified incidentally during imaging performed for unrelated indications [39]. In cases of suspected atherosclerotic renal embolization, a history of recent precipitating events such as angiographic procedures within the preceding six months, progressive renal dysfunction, characteristic skin findings, and retinal cholesterol crystals strongly support the diagnosis [33][34][38].

Evaluation

Laboratory Studies

Laboratory assessment plays a central role in the evaluation of suspected renal infarction and often provides the earliest objective clues supporting the diagnosis. Common laboratory abnormalities include leukocytosis and elevated inflammatory markers such as C-reactive protein, reflecting the acute inflammatory response to ischemic tissue injury [35][40]. One of the most characteristic findings is a marked elevation in lactate dehydrogenase, which results from extensive cellular necrosis within the renal parenchyma. In renal infarction, LDH levels may increase to four or five times the upper limit of normal without a parallel rise in hepatic enzymes such as aspartate aminotransferase or alanine transaminase, a pattern that helps distinguish renal infarction from other causes of acute flank pain including renal colic or pyelonephritis [4][41][42]. Importantly, LDH elevation may persist for up to fifteen days following symptom onset, making it a useful diagnostic marker even in delayed presentations [6]. Urinalysis frequently demonstrates macroscopic or microscopic hematuria and proteinuria, reflecting glomerular and tubular injury within the ischemic renal tissue [35][40]. Serum creatinine levels may also be elevated, particularly in cases involving large infarcts or bilateral renal involvement, where nephron loss significantly compromises overall renal function [6]. Creatine kinase may rise in some patients, especially when extensive ischemic injury is present. When common causes such as cardioembolic disease or renal arterial pathology are excluded, or when revascularization is under consideration, a comprehensive evaluation for underlying coagulation disorders is warranted to identify potentially correctable etiologies. In cases of renal infarction due to cholesterol embolization, additional laboratory features may be observed. These include eosinophilia, eosinophiluria, and reduced serum complement levels during the acute phase, although these abnormalities often resolve within one week of the initial insult [43][44][45]. The presence of these findings, in conjunction with clinical features such as focal neurological deficits, mental status changes, localized cyanosis, or retinal Hollenhorst plaques, supports the diagnosis of atherosclerotic embolic disease [1]. Atheroembolic renal injury is typically associated with a slower and more insidious decline in renal function, which may progress over several weeks before stabilizing [27]. Because clinical recognition is frequently delayed or missed, renal biopsy may be required for definitive diagnosis. Histopathological examination identifies cholesterol emboli in more than 75% of confirmed cases, appearing as biconvex or needle-shaped clefts within vessel lumens after cholesterol dissolution during tissue processing [46][47]. Associated findings include marked intimal inflammation, eosinophilic infiltration, and, in some cases, focal glomerulosclerosis [44][47][48].

Imaging Studies

Imaging is essential for confirming the diagnosis of renal infarction and for differentiating it from more common causes of acute flank pain. Initial evaluation typically begins with a noncontrast computed tomography scan of the abdomen and pelvis, as this modality effectively excludes urolithiasis and acute pyelonephritis, which account for the majority of similar clinical presentations [49]. However, noncontrast CT findings in renal infarction are often nonspecific and may demonstrate only mild renal enlargement, perinephric fat stranding, or no detectable abnormalities at all. When clinical suspicion remains high, particularly in patients with thromboembolic risk factors, hematuria, or markedly elevated LDH levels, contrast-enhanced CT imaging should be pursued [30]. Contrast-enhanced CT is the preferred noninvasive modality for identifying renal infarction. Typical findings include well-defined hypodense regions within the renal parenchyma corresponding to areas of reduced or absent perfusion. These defects are often wedge-shaped and are best visualized during the arterial phase of contrast

enhancement. In segmental infarction, a thin rim of normally enhancing cortex may be observed surrounding the infarcted area, a phenomenon known as the cortical rim sign, which is present in approximately half of renal infarction cases [50][51]. This sign usually becomes apparent several days after the ischemic event and reflects preserved capsular perfusion supplied by collateral vessels arising from the suprarenal, ureteral, and lumbar arteries [52]. In cases of complete main renal artery occlusion, contrast imaging may demonstrate total nonenhancement of the affected kidney. Traumatic transection of the renal artery may be suggested by the presence of a hilar hematoma on CT imaging. When the kidney appears chronically atrophic or scarred, revascularization is unlikely to be beneficial.

CT renal arteriography remains the gold standard diagnostic test for renal infarction, as it provides definitive visualization of arterial occlusion and guides therapeutic decision-making [19]. Magnetic resonance angiography offers comparable diagnostic accuracy but is less commonly used in the acute setting due to longer acquisition times. Nuclear medicine techniques, such as dimercaptosuccinic acid scans, can also demonstrate areas of reduced or absent tracer uptake corresponding to infarcted renal tissue. Renal artery Doppler ultrasonography is frequently used to assess renovascular disease but has limited sensitivity for nonstenotic lesions and is highly operator-dependent. Image quality may also be compromised by patient body habitus. Color Doppler imaging can demonstrate reduced or absent blood flow within affected regions but cannot reliably determine etiology [29]. Intravenous pyelography may reveal nonvisualization of infarcted renal segments, although this modality has largely been replaced by advanced cross-sectional imaging [4]. Intravascular ultrasound represents an emerging diagnostic tool that enhances the evaluation of renal arterial pathology by providing detailed information about the vessel wall, which is not fully visualized by conventional arteriography. This technique can help differentiate thrombotic occlusion, arterial dissection, and fibromuscular dysplasia. Although currently used primarily in cardiovascular imaging, further research may expand its role in renal infarction assessment [29][53].

Additional Cardiac Studies

Given the strong association between renal infarction and cardioembolic disease, cardiac evaluation is a critical component of the diagnostic workup. All patients without an immediately identifiable cause should undergo electrocardiographic assessment or prolonged Holter monitoring to detect atrial fibrillation or other arrhythmias that may predispose embolic events. Transthoracic or transesophageal echocardiography is recommended to evaluate for intracardiac thrombi, valvular pathology, or structural abnormalities that could serve as embolic sources [52]. Identification of a cardiac etiology has significant implications for long-term management, including anticoagulation strategies aimed at preventing recurrent embolic events.

Treatment and Management

Management of renal infarction is guided by the underlying etiology, extent of ischemia, renal function at presentation, and the timing of diagnosis. Accurate vascular assessment is essential, and CT renal angiography is the preferred modality for evaluating renal arterial anatomy, identifying the affected vessels, and determining the degree of occlusion. Revascularization is generally considered only if the affected kidney demonstrates viable parenchyma, as evidenced by preserved size or perfusion. Imaging showing a shrunken, atrophic kidney or a dense wedge-shaped scar usually indicates chronic infarction, for which revascularization is not beneficial. Therapeutic strategies can be broadly categorized into catheter-directed thrombolysis, systemic thrombolysis, anticoagulation or antiplatelet therapy, surgical interventions, and supportive management, including antihypertensive therapy [54][55]. Several factors influence the decision to pursue revascularization, including time since onset of ischemia, renal function, infarct size and location, the presence of arterial dissection, contralateral kidney function, and whether occlusion is partial or complete. Establishing the timing of infarction can be challenging. Acute clinical symptoms such as sudden flank pain, nausea, vomiting, and new-onset hypertension generally suggest a recent onset of less than one week. Complete arterial occlusions of six hours or less are generally considered potentially reversible. Even with complete unilateral occlusion, patients with otherwise normal renal function may not initially present with elevated blood urea nitrogen or creatinine. CT angiography is essential to assess the degree of arterial obstruction, as partial occlusions with some perfusion indicate the potential for renal recovery.

Catheter-directed thrombolysis, also referred to as percutaneous endovascular therapy, is the primary intervention for proximal or bilateral renal artery occlusions and is particularly critical for patients with a solitary functioning kidney. Segmental artery occlusions may also be treated selectively using this approach. Techniques include local intra-arterial thrombolysis, mechanical thrombectomy, angioplasty, or stenting, and outcomes are best when interventions are performed promptly after symptom onset. Case reports indicate that reperfusion can be successful even up to 96 hours post-presentation, although earlier intervention correlates with improved recovery [55][56][57][58]. Protocols for catheter-directed thrombolysis often involve initial thrombectomy, followed by a bolus intra-arterial infusion of urokinase (250,000 IU) if thrombectomy is incomplete. Continuous infusion of 50,000 IU per hour for up to 72 hours may be used, with daily angiographic monitoring. If no reperfusion is achieved after 72

hours, therapy is discontinued. Alternative thrombolytic agents, such as streptokinase or tissue plasminogen activator, have also been utilized. When thrombolysis is unsuccessful, angioplasty with or without stenting may be considered, though formal guidelines are lacking [37]. Complications include hemorrhage, pseudoaneurysm formation, and distal embolization. Following intervention, antiplatelet therapy with aspirin and clopidogrel is generally prescribed for three to six months. Novel techniques, such as ultrasound-enhanced thrombolysis, may improve penetration of thrombolytic agents into thrombi [60].

Systemic thrombolysis is an alternative when catheter-directed therapy is unavailable. While effective, systemic therapy carries a higher risk of major bleeding compared to targeted catheter-directed approaches [61][62]. Reperfusion injury remains a potential complication in both systemic and catheter-directed thrombolysis. Compared with systemic therapy, catheter-directed treatment allows for lower total doses and precise delivery of thrombolytic agents directly to the occluded vessels, improving efficacy and reducing systemic adverse effects [60]. Patients presenting several days or longer after infarction, when revascularization is unlikely to restore function, are managed with anticoagulation or antiplatelet therapy. Long-term anticoagulation is indicated for patients with underlying hypercoagulable disorders or cardioembolic sources. Standard therapy typically includes intravenous heparin followed by warfarin with a target INR of 2 to 3, or higher (2.5–3.5) in patients previously on therapeutic anticoagulation. Direct oral anticoagulants, including Factor Xa inhibitors, are increasingly used due to their stable pharmacokinetics and lack of monitoring requirements. Aspirin is recommended for secondary prevention, particularly in patients with prior infarction, even if no overt hypercoagulable state is identified. Open surgical interventions are reserved for trauma-induced renal artery injury, aortic dissection extending into renal arteries, or select cases of fibromuscular dysplasia. Procedures include arteriotomy with embolectomy, aorto-renal or ileo-renal bypass, and vascular reconstructive surgery. Surgery is generally ineffective for thrombotic or embolic infarction and carries significant perioperative risk [37][40]. Referral to specialized centers is advisable for patients requiring complex vascular reconstruction.

Renovascular hypertension commonly develops within the first week following renal infarction due to renin-mediated mechanisms. Agents targeting the renin-angiotensin system are preferred, and blood pressure often normalizes as renal perfusion is restored. Long-term hypertension management should follow standard clinical guidelines if underlying essential hypertension is present [37][39]. Treatment of atheroembolic renal infarction is primarily supportive, focusing on prevention of further embolization. Patients should avoid elective angiographic or vascular procedures during the acute phase. Prognosis is often determined by the severity of underlying atherosclerotic disease, though partial renal function recovery is possible in patients without significant comorbidities, with some studies reporting complete recovery in approximately 24% of cases [27]. Preventive measures include aspirin therapy, strict blood pressure and glycemic control, smoking cessation, and weight management [1][63][65]. Long-term follow-up for renal infarction has not been standardized. Monitoring should include periodic assessment of renal function and surveillance for therapy-related complications, particularly bleeding in anticoagulated patients. Lifelong aspirin therapy is generally recommended, even in patients with a remote or asymptomatic renal infarction [10][66]. Follow-up imaging, preferably using MRI, is suggested at six and twelve months to detect new lesions or assess recovery. Patients with preexisting renal or cardiac conditions require closer monitoring and potentially longer-term surveillance to prevent recurrent events and optimize renal and cardiovascular outcomes.

Differential Diagnosis

Renal infarction presents with nonspecific symptoms that overlap with a variety of abdominal, urologic, and vascular conditions, making accurate differential diagnosis essential. Conditions commonly considered include nephrolithiasis, pyelonephritis, aortic aneurysm, aortic dissection, appendicitis, diverticulitis, gastroenteritis, mesenteric ischemia, renal cell carcinoma, and gynecologic disorders. Nephrolithiasis and pyelonephritis are particularly frequent misdiagnoses because flank pain, hematuria, and nausea are shared features. Failure to include renal infarction as a potential diagnosis can delay treatment, rendering revascularization ineffective and increasing the risk of irreversible renal damage. Laboratory evaluation can aid differentiation, as marked elevation of lactate dehydrogenase (LDH) without corresponding increases in serum transaminases is characteristic of renal infarction and not typically seen in pyelonephritis or nephrolithiasis. Imaging plays a critical role, with contrast-enhanced CT of the abdomen providing definitive evidence of perfusion defects. In patients at risk for systemic emboli, particularly those with atrial fibrillation or hypercoagulable states, clinicians should evaluate for concurrent mesenteric ischemia, which may necessitate urgent angiography to prevent bowel infarction [66].

Prognosis

The prognosis for renal infarction is influenced by both renal and systemic factors. Retrospective studies suggest that approximately 20% of patients diagnosed with renal infarction die within 40 months, although progression to end-stage renal disease (ESRD) is relatively uncommon, affecting only around 2% of cases [29]. Mortality is often

related to underlying cardiovascular disease or embolic events in other organs rather than renal injury alone. Early recognition and prompt intervention significantly improve outcomes, as timely revascularization can restore perfusion and preserve renal function. Factors affecting prognosis include warm ischemia time, collateral blood flow, infarct size, and preexisting renal impairment. Catheter-directed thrombolysis has been associated with improved renal recovery when initiated promptly. Extrarenal embolic events, such as infarction of the spleen, liver, intestines, or lungs, worsening overall morbidity and prolong hospitalization. Prognostic evaluation must therefore consider systemic embolic risk and underlying cardiovascular status, emphasizing the importance of a multidisciplinary approach in both acute management and long-term surveillance [29].

Complications

Renal infarction can lead to both acute and long-term complications, primarily involving renal function and blood pressure regulation. Acute kidney injury (AKI) frequently occurs due to ischemic damage to renal tissue, particularly in cases with extensive or bilateral infarction. If ischemia is severe or prolonged, AKI may progress to chronic kidney disease (CKD), with studies demonstrating CKD development in up to 58% of patients during follow-up renal scintigraphy [67][68][69]. The severity of AKI and the patient's age are significant predictors of CKD progression. Other complications include renovascular hypertension, typically mediated by increased renin release, which may persist even after partial renal recovery. Secondary cardiovascular events may also arise due to the underlying etiologies of renal infarction, such as atrial fibrillation or atherosclerosis. Early detection, revascularization when indicated, and careful monitoring of renal function are crucial to minimizing complications and optimizing long-term outcomes.

Consultations

Management of renal infarction requires interprofessional collaboration and specialist consultations tailored to the underlying etiology and complications. Nephrologists are essential for monitoring renal function, managing AKI or CKD, and guiding long-term renal care. Cardiologists assess and manage cardioembolic sources such as atrial fibrillation or valvular disease, while hematologists evaluate underlying hypercoagulable states that may contribute to infarction. Vascular surgery or interventional radiology may be consulted for structural vascular lesions, including fibromuscular dysplasia, arterial dissection, or thromboembolic occlusions requiring revascularization. Follow-up evaluations are critical, as inflammation during the acute phase may obscure vascular abnormalities. Extra-renal arteries, such as carotid vessels, should also be assessed in patients with systemic embolic risk or suspected vasculopathies, including Ehlers-Danlos syndrome, to prevent recurrent infarctions and optimize patient outcomes [52].

Patient Education

Patient education is a critical component of renal infarction management, particularly regarding anticoagulation and secondary prevention. Patients on warfarin must understand the importance of maintaining therapeutic INR levels, recognizing potential food or drug interactions, and adhering to prescribed dosing schedules. Those with recurrent infarctions despite anticoagulation require counseling on medication compliance and risk factor management. Lifestyle modifications, including smoking cessation, blood pressure and glycemic control, dietary adjustments, and regular exercise, should be emphasized. Education on recognizing early symptoms of recurrent renal or systemic emboli is essential for timely medical intervention. Pharmacists play a key role in reinforcing adherence, monitoring interactions, and providing counseling, while nurses provide direct patient education and follow-up support, ensuring a coordinated and effective preventive strategy.

Other Issues

Renal infarction requires a high degree of clinical suspicion due to its rarity and nonspecific presentation. High-risk patients include older adults, those with atrial fibrillation, or individuals who have recently undergone vascular instrumentation. Acute flank pain with hematuria in the absence of urinary tract infection, hydronephrosis, or calculi should prompt immediate consideration of renal infarction. If initial noncontrast CT imaging is negative, prompt contrast-enhanced CT or CT angiography is warranted to facilitate potential revascularization. Elevated LDH with normal AST and ALT levels is a hallmark laboratory finding. Comprehensive evaluation should include urinalysis, serum aminotransferases, creatinine, LDH, EKG, and coagulation studies. Rapid diagnosis allows timely catheter-directed thrombolysis, ideally within six hours of infarction onset. Systemic thrombolysis may be employed if catheter-based intervention is unavailable. Long-term secondary prevention with anticoagulants and antiplatelet therapy is recommended for all patients [37][55].

Enhancing Healthcare Team Outcomes

Optimal management of renal infarction requires an interprofessional team, including primary clinicians, nephrologists, cardiologists, hematologists, vascular surgeons, interventional radiologists, nurses, and pharmacists. Diagnosis is primarily clinician-driven, while endovascular or surgical interventions involve interventional radiology or vascular surgery. Nephrologists evaluate renal function and manage AKI or CKD, while cardiologists and hematologists address underlying etiologies such as atrial fibrillation or hypercoagulable disorders. Nursing staff provide patient assessment, surgical preparation, and postoperative care, and pharmacists ensure safe

anticoagulation, medication reconciliation, and patient counseling. Effective communication, prompt reporting of patient status changes, and coordinated interventions among all team members are critical to reducing complications, improving patient outcomes, and ensuring timely and safe management of this rare and complex condition.

Conclusion:

Renal infarction remains a frequently underdiagnosed condition due to its nonspecific symptoms and overlap with more common abdominal and urologic disorders. Early identification is essential, as prompt revascularization can preserve renal function and reduce long-term complications, while delays may lead to irreversible injury. High-risk individuals—particularly those with atrial fibrillation, atherosclerosis, or hypercoagulable states—require heightened clinical vigilance. Effective management depends on a multidisciplinary approach involving nephrologists, cardiologists, hematologists, radiologists, surgeons, nursing staff, and pharmacists working collaboratively to provide timely diagnostics, therapeutic interventions, and patient education. Long-term outcomes are optimized through sustained anticoagulation when indicated, aggressive risk-factor modification, control of hypertension, and regular renal function monitoring. Ultimately, improved awareness, interprofessional coordination, and adherence to evidence-based strategies are key to reducing morbidity, preventing recurrence, and enhancing overall patient prognosis in renal infarction.

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