REVIEWS

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Renal Vein Thrombosis – Etiology, Diagnosis, Therapeutic Options

Zakrzepica żyły nerkowej – etiologia, rozpoznanie i opcje terapeutyczne

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Abstract

The majority of venous thrombotic events involve the extremities, although venous thrombosis may occur at any location, including organ veins. The appropriate evaluation and treatment has been well defined for venous thromboembolism of the legs, but it remains less clear for venous thrombosis involving other venous segments. Renal vein thrombosis (RVT) is a rather uncommon but possibly very serious clinical entity characterized by a number of underlying causes and risk factors, a very broad spectrum of clinical manifestation, unknown outcome, and poorly defined treatment strategy. The purpose of this review is to summarize the corporate literature regarding etiology, diagnostic modalities, clinical manifestation and, particularly, therapeutic options on which to base recommendations for anticoagulant therapy (**Adv Clin Exp Med 2006, 15, 5, 917–924**).

Key words: renal vein thrombosis, recurrence, survival, anticoagulation.

Streszczenie

Zdecydowana większość przypadków zakrzepicy żylnej dotyczy kończyn, proces zakrzepowy jednak może objąć każde naczynie żylne, w tym również żyły narządów wewnętrznych. Zalecenia diagnostyczne i lecznicze w przypadku zakrzepicy żylnej kończyn zostały dokładnie określone, ale dla zakrzepicy obejmującej inne części układu żylnego pozostają nadal niejasne. Zakrzepica żyły nerkowej (RVT) jest jednostką chorobową występującą rzadko i może być bardzo groźna. Charakteryzuje się obecnością wielu czynników sprawczych, bardzo szerokim zakresem objawów klinicznych, nieznanym rokowaniem i bardzo niejasno sformułowanymi zaleceniami leczniczymi. Celem pracy jest podsumowanie dostępnej literatury na temat etiologii, technik diagnostycznych, objawów klinicznych, a szczególnie możliwości terapeutycznych, na podstawie których można by sformułować zalecenia co do leczenia przeciwzakrzepowego w tej chorobie (**Adv Clin Exp Med 2006, 15, 5, 917–924**).

Słowa kluczowe: zakrzepica żyły nerkowej, nawroty, rokowanie, leczenie przeciwzakrzepowe.

Renal vein occlusion is a rather uncommon but intriguing clinical entity caused by a variety of different factors and having a very broad spectrum of clinical manifestation from asymptomatic to acute renal failure [1–5], unknown outcome, and poorly define treatment strategy. After occlusion by thrombus, renal veins develop two primary routes of collateral drainage, one through the subcapsular plexus and the other, only applicable to the left kidney, involving the ureteral, gonadal, and adrenal veins [6–8]. The ultimate response of the kidney to venous occlusion is determined by the balance between the extension and acuteness of occlusion, the development of collateral circulation, the involvement of one or both kidneys, and the origin and character of the underlying disease(s) [8–11].

The first description of renal vein thrombosis (RVT) is attributed to Hunter [12], but Rayer was the first to recognize its relationship to the

nephrotic syndrome [13]. Early studies focused primarily on the *post mortem* diagnosis of RVT found at autopsy, as was the large Mayo Clinic analysis of 29,280 unselected adult autopsies performed from 1929 to 1961 [14]. Only 17 cases of bilateral renal vein thrombosis were found, i.e. an incidence of approximately 0.6 per 1000 autopsies; among these 17 RVT cases, males predominated (2.5:1 over females), the average age was 47 years, and only 2 had nephrotic syndrome. Several prospective studies have evaluated the incidence of RVT in nephrotic syndrome and reported incidences ranging from 1.9 to as high as 42% [15, 16]. Llach prospectively evaluated 151 patients with nephrotic syndrome and found RVT in 22% [17]. The reported incidence of renal vein occlusion in children varies from 0.26 to 0.7% [2].

Etiology

Etiology varies depending on the composition of the general and local causes, and these determine different presentation, treatment, and prognosis. Several classification schemes have been proposed [14, 18, 19] for dividing contributing variables into systemic and local categories. Primary renal disease is likely the most common cause, particularly if renal malignancy is added to this group [14]. Both acute and chronic nephropathic conditions can be complicated by venous thrombosis, particularly membranous glomerulonephritis. The prevailing theory is that RVT is a results of the underlying kidney disease rather than its cause. Renal vein thrombosis complicates nephrotic syndrome one third of the time on average. There are several proposed mechanisms which may contribute to the hypercoagulable state of nephrotic syndrome. These include increased levels of factors V and VIII [20, 21], reduced AT-III concentration [22, 23], and altered protein S metabolism with reduced free protein S and elevated C4b-binding protein, the carrier protein for protein S [24]. Elevated fibrinogen concentrations and thrombocytosis may also contribute. Immunological injury induced by subepithelial deposition of various antigen and antibody complexes may result in endothelial injury, providing a nidus for thrombus formation. In general, any acute or chronic renal disease can be complicated by renal vein thrombosis [3–5, 25–26]. The prevalence appears to be greater in primary as opposed to secondary glomerular disease [27–30].

Renal cell carcinoma has the propensity to invade the venous system and is associated with venous tumor thrombus in 4% to 25% of cases [31–33]. When present, tumor thrombus heralds a worse prognosis, with larger malignancy of more advanced grade and stage and more frequent metastases. Inferior vena caval thrombus propagation with retrograde involvement of renal veins is always a concern, particularly in those patients with a supra-renal IVC filter or cephalad filter migration.

Congenital and acquired thrombophilias have been reported, including deficiencies in antithrombin III, protein C, protein S deficiencies, hyperhomocysteinemia, and the antiphospholipid syndrome; however, these are primarily limited to case reports [34–38]. Oral contraception, pregnancy, and the puerperium have also been described as causative factors [39–40].

Renal blood flow is robust, comprising 20% of cardiac output. Therefore, rapid efflux of blood from the renal veins may be protective and limit the formation of venous clots [41]. Indeed, alterations in renal hemodynamics alone may precipitate RVT, thus underlining the relevance of this variable. Dehydration induced by febrile illness, diarrhea, or sepsis has been shown to cause RVT particularly in the infant and neonate. Other causes of hemodynamic instability, such as adrenal hemorrhage, hypoglycemia, seizure disorders, cyanotic congenital heart disease, and right heart failure, have been implicated [3–5, 42].

RVT may be induced by extrinsic and/or intrinsic involvement of the renal vascular pedicle. A primary tumor of the venous wall, usually leiomyosarcoma arising in the inferior vena cava, serves as an example of an intrinsic obstruction. Of 86 vascular leiomyosarcomas in one study, 33 involved the inferior vena cava and nearly all were accompanied by thrombus [43]. Renal venous ostia and the retrohepatic portion of the inferior vena cava were involved in the majority. Retroperitoneal processes, such as tumor, lymphoma, retroperitoneal fibrosis, or abscess, may precipitate RVT by extrinsic compression or involvement of the renal veins [44–45]. The "nutcracker phenomenon" involves compression of the left renal vein between the abdominal aorta and the superior mesenteric artery [46]. This syndrome produces left RVT and provokes symptoms of pelvic congestion, which include dysmenorrhea, dysuria with microhematuria, dyspareunia, vulvar and pelvic varices in the female, and varicoceles in males [47].

RVT may occur in transplanted renal allografts which, unlike the native kidney, have a single drainage system. Bakir et al. [48] reviewed 558 consecutive cadaveric kidney transplants and noted a 6% incidence of RVT, which accounted for one third of all early (90 days) graft failures. Living related donor renal transplant is associated with a significantly lower rate of RVT [49].

Predisposing factors may include the use of OKT3 and cyclosporine therapy [50–51]. Vascular injury due to trauma or surgery may also precipitate RVT. Renal vein thrombosis secondary to trauma is usually accompanied by renal artery thrombosis and is suggested by the history and a palpable flank mass on examination [52–53].

Clinical Presentation

Since the spectrum ranges from acute complete to chronic incomplete renal vein(s) occlusion, the clinical spectrum varies from acute severe illness with renal failure to completely asymptomatic course [3–4, 13–16, 43]. In the acute setting, seen more often in neonates and dehydrated infants, the affected kidney(ies) becomes markedly congested, the capsule distends, and hemorrhagic infarction develops, resulting in a nonfunctional kidney. Even though the clinical presentation in a newborn less than 72 hours old is usually sudden onset of abdominal distension, a flank mass, hematuria, and proteinuria, the condition may remain asymptomatic, only to be detected later in life by the presence of an atrophic kidney [1–12, 14–19, 25–26].

In gradual occlusion, collateral vessels can develop effectively, providing adequate venous drainage and thus preventing renal damage. Clinical signs and symptoms may be minimal or absent, with predominant presentation of underlying diseases, and the presence of renal vein occlusion remains unsuspected (undiagnosed). Chronic, unspecific complaints (nausea, apathy, weakness, or generalized edema) or upper abdominal and/or flank pain may also be the presenting symptom [1–12].

Laboratory studies show no characteristic changes for renal vein occlusion. The urine may contain an increased number of red cells and, in the absence of primary renal disease, the urine shows little protein and few, if any, abnormal elements. No notable changes in blood count or serum enzymes have been recognized [1–12].

Diagnostic Techniques

Radiographic methods constitute the principal means of confirming the diagnosis. The sonographic diagnosis of RVT is primarily based on direct visualization of thrombi within the renal vein and inferior vena cava, demonstration of renal vein dilatation proximal to the point of occlusion, and increase in renal size during the acute phase of venous congestion, then, if infarction and/or atrophy progress, demonstration of a small nonfunctional kidney of abnormal renal structure. However, conventional sonographic findings of RVT are largely unspecific [54]. The right renal vein is usually readily shown when the liver is used as an acoustic window, but the left renal vein is often obscured by overlying bowel gas. Renal scan is another noninvasive method for diagnosing renal vein occlusion. Persistent concentration on delayed scans with enlargement of the involved kidney is consistent with renal vein thrombosis on dynamic and static 99m technetium-pentetic acid study [55].

Excretory urography reflects interstitial swelling from venous congestion, which causes stretching and compression of the collecting system, by decreased or absent opacifications. The acute symptomatology of RVT in association with this radiologic appearance establishes the diagnosis. Excretory urography may show notching of the ureters or renal pelvis from impingement of dilated, tortuous collaterals; however, notching is not specific to renal vein occlusion. It should be noted that excretory urography may appear entirely normal in some patients with renal vein occlusion [56–59].

Originally, renal arteriography with a focus on the nephrographic and venous phase was considered by some investigators [60] to be preferable to renal venography because of the better assessment of the renal parenchyma, blood flow, and presence of collaterals with no risk of dislodging venous thrombi. However, lack of detail in the venous phase makes the study alone not infrequently insufficient for a definitive diagnosis of RVT. Only delayed venous phase films will show larger renal veins and the site of obstruction. Acute RVT will show in arteriography an enlarged kidney, poor filling of cortical arteries, and no renal vein drainage if the renal vein is completely occluded. If renal vein obstruction has been gradual, a normal arterial phase and profuse collaterals in the venous phase will be obtained. Renal arteriography is generally preferred in patients being evaluated for RVT associated with renal trauma or tumor [56–60].

Inferior venacavography with selective catheterization of the renal vein establishes the diagnosis of RVT most reliably [61, 62]. Selective renal venography shows partial defects indicating clot or stenosis if occlusion is incomplete (Fig. 1) or an image of amputation in cases of complete occlusion of the renal vein. In general, the use of epinephrine is not necessary, but if all contrast material is washed out of the renal vein within three seconds or less, the use of intra-renal arterial epinephrine by decreasing blood flow enhances retrograde venous filling and allows later visualization

of the smaller intra-renal veins. The collateral network is often easily demonstrated with this procedure, usually reflects the chronicity of the RVT, and explains the lack of renal functional deterioration. Recently, RVT has been diagnosed by digital subtraction venography, which is a simple, safe, noninvasive and, in some series, efficient method [63].

Computerized tomography (CT) can be very valuable method in the diagnosis of RVT. Definitive CT diagnosis is based on visualization of the clots within the vein and sometimes extension into the inferior vena cava [64]. Indirect CT signs include enlargement and distention of the affected renal vein (Fig. 2), with kidney enlargement, persistent parenchymal opacification, capsular venous collaterals, thickening of Gerota's fascia, and pericapsular "whiskering", i.e. stranding of the perinephric fat and perinephric hemorrhage [65]. Magnetic resonance imaging (MRI) produces high-contrast images between flowing blood, vascular walls, and surrounding tissues with the best vascular potency (Fig. 3) and is becoming the diagnostic procedure of choice in the diagnosis of RVT [66]. MRI is used routinely in the detection and assessment of the extent of neoplastic renal vein thrombosis in the preoperative staging of renal cell carcinoma [67]. However, MRI has also been used successfully in assessing nonneoplastic renal vein thrombosis [68, 69].

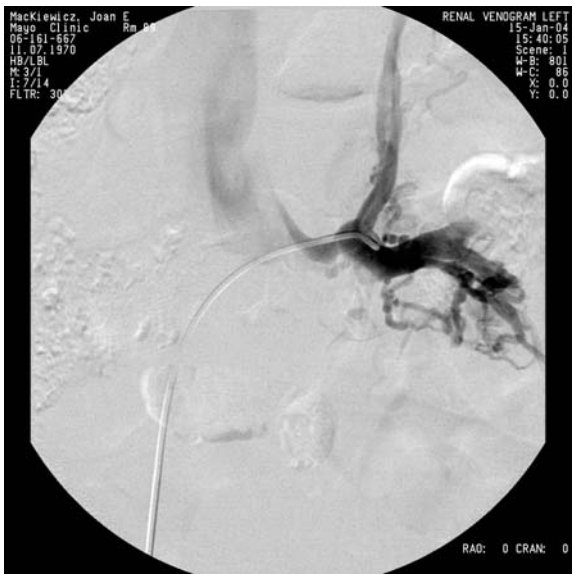

Fig. 1. Selective venogram of the left renal vein reveals stenosis of the proximal segment and collateral drainage via the adrenal vein.

Ryc. 1. Wybiórcza wenografia lewej żyły nerkowej ukazuje zwężenie naczynia przy ujściu do żyły czczej oraz dobrze rozwinięte krążenie oboczne przez żyłę nadnerczową

Treatment and Prognosis

The main elements of therapy can be summarized in the following list of goals: 1) specific treatment of RVT (anticoagulation, thrombolytic, intravascular mechanical thrombus disintegration, thrombectomy), 2) treatment of acute complications (an acute, life-threatening hemorrhage as a result of capsular rupture) and the long-term sequelae (hypertension, infection), 3) specific treatment of underlying renal disease (glomerulopathy, tumors), 4) specific treatment of systemic disease (lupus erythematosus, polyarteritis nodosa, diarrhea, sepsis, adrenal hemorrhage, congestive heart failure), 5) specific treatment of extrarenal causes (excision of retroperitoneal tumors, aortic aneurysm repair, vena caval prima-

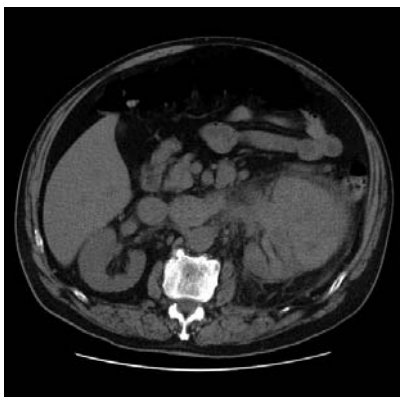

Fig. 2. Abdominal CT picture shows distended thrombosed left renal vein and enlarged, edematous kidney with tumor.

Ryc. 2. Zdjęcie tomografii komputerowej jamy brzusznej przedstawia rozdętą, objętą skrzepem lewą żyłę nerkową wraz z powiększoną, obrzękniętą nerką zawierającą guz nowotworowy

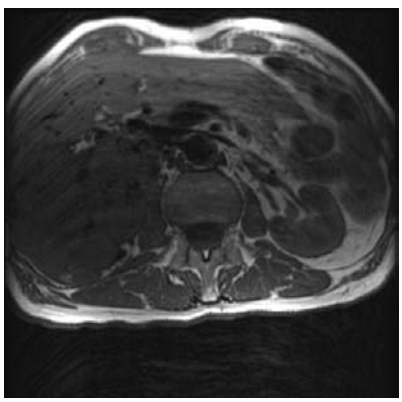

Fig. 3. MR image of left renal vein with distended medial portion that contains clearly seen thrombotic material

Ryc. 3. Obraz lewej żyły nerkowej uzyskany z użyciem rezonansu magnetycznego ukazuje rozszerzoną środkową część naczynia z wyraźnie widocznym materiałem zakrzepowym

ry tumors resection, decompression of the left renal vein squeezed between the abdominal aorta and superior mesenteric artery, treatment of retroperitoneal fibrosis, abscesses), 6) nonspecific treatment directed toward the physiological and biochemical abnormalities (reverse hydration, hypoglycemia, seizure disorders, use of hemodialysis, diuretics).

To date, nephrectomy has been abandoned except in an acute, life-threatening hemorrhage as a result of capsular rupture, renal cell carcinoma, trauma, and extrinsic compression of the vascular pedicle [70–72]. A conservative approach with anticoagulation as the main therapeutic modality seems to be the best for most cases in adults. So far there has been no prospective randomized study looking into the effectiveness of anticoagulation in RVT. Most clinical observations of thromboembolic complications and anticoagulation effect are from nephritic patients. These data show a high incidence of thromboembolic phenomena and poor prognosis [14, 17–19, 73, 74]. Andrassy and Ritz [75] observed 84 nephrotic cases and found over a period of three years 37 episodes of thromboembolic complications in 30 patients. Among these were 23 events of deep vein thrombosis, an incidence of 44%, probably the highest number encountered in medical patients. Other investigators reported lower incidence of thromboembolic complications in children and adults patients with nephritic syndrome (27% to 8.5%), which is still relatively high compared with classic thromboembolic disease [27–29, 76–79]. Lower-extremity deep vein thrombosis and pulmonary embolism were the most frequent complications. A prospective study also revealed 17% incidence of thromboembolic complications in these patients, with abnormal ventilation-perfusion scans in 21% of cases [17]. Renal function may dramatically improve in patients with acute RVT treated with anticoagulant therapy [3–5, 14, 17, 79, 80]. Relapses with new episodes of acute RVT have been observed after cessation of anticoagulant therapy [80].

These clinical observations seem to indicate that patients with RVT should be treated with heparin and warfarin and that warfarin needs to be continued for at least 6 months, but some recommend anticoagulation to continue as long as the predisposing factor exists, which also refers to nephrotic syndrome [3–5, 14, 17, 77–80]. Recently, thrombolytic agents such as urokinase, streptokinase, and tissue plasminogen activator have been used in the management of acute and even chronic renal vein thrombosis, with restoration of renal venous drainage and immediate improvement in function of the affected kidney within 1 to 4 days of the initiation of fibrinolytic therapy [81–84]. To date, however, there are no published data on any large series of patients undergoing thrombolytic therapy for RVT. Moreover, the relative efficacies of heparin vs. fibrinolytic agents in RVT remains undefined. Markowitz et al. [85] analyzed the outcomes of 21 patients with renal thrombosis treated with thrombolytic therapy between 1968 and 1993 and found that 3 (14%) died of bleeding complications. Laville et al. [86] reviewed 27 cases of renal-vein thrombosis: 10 patients treated by anticoagulation alone, 9 by surgical thrombectomy, 7 by thrombolysis, 2 receiving no specific treatment, and one undergoing successively thrombectomy and then thrombolysis. Eight of the 10 patients on anticoagulation lived beyond two months. In contrast, only 4 of the 7 patients treated with thrombolytic agents followed by anticoagulation lived beyond two months; the cause of death in the other 3 patients was hemorrhagic complications. Three of the 9 patients treated with surgical thrombectomy did not survive beyond two months. The limited data seem to indicate that thrombolytic therapy probably offers a more rapid and complete resolution than anticoagulation, but at a much higher risk of hemorrhagic complications and even death. The most recent technique of percutaneous mechanical thrombectomy, which in case reports has proved effective in the treatment of acute RVT, with rapid blood flow restoration followed by a significant improvement in renal function [87], should significantly decrease the risk of hemorrhagic complications.

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