Hong Kong J Radiol. 2022;25:136-42 | https://doi.org/10.12809/hkjr2217303

ORIGINAL ARTICLE

Computed Tomography Findings in Wunderlich Syndrome

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ABSTRACT

Background: *Wunderlich syndrome (WS) is defined as renal/perirenal haemorrhage in the absence of trauma. Causative factors include neoplasms, vascular abnormalities, cystic kidney diseases, and coagulation disorders. Computed tomography (CT) is the diagnostic method of choice in the diagnosis of WS. We aimed to investigate CT findings, aetiologies, demographic features, and treatment outcomes in a group of patients diagnosed with WS.*

Methods: *We retrospectively reviewed 113 patients found to have renal-perirenal hematoma on CT between January 2015 and January 2020, and excluded 101 cases with a history of trauma or renal biopsy. The remaining 12 patients constituted the study group.*

Results: *Five (41.7%) of the 12 patients included in the study were female and seven (58.3%) were male. The mean age was 53.2 years (range, 7-81). CT revealed mass lesions in seven (58.3%), a pseudoaneurysm in two (16.7%), and renal vein thrombosis in one (8.3%) patient. Two of the mass lesions (28.6%) detected on CT were angiomyolipomas, one (14.3%) was a haemorrhagic cyst in a patient with adult polycystic kidney disease, and four (57.1%) were solid mass lesions. Three of the four patients with masses were surgically treated. All pseudoaneurysms, the single inoperable solid mass, and one of the angiomyolipomas were treated angiographically.*

Conclusion: *WS is an acute urological emergency with multiple possible aetiologies, some of which are more likely to require surgical management. CT is important in the diagnosis and management of the syndrome to identify haemodynamically unstable cases in need of immediate intervention.*

Key Words: *Angiomyolipoma; Arteriovenous malformations; Carcinoma, renal cell; Hemorrhage; Kidney/diagnostic imaging*

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Submitted: 2 Nov 2020; Accepted: 11 Feb 2021

Contributors: Both authors designed the study and acquired the data. EE analysed data and drafted the manuscript. EG critically revised the manuscript for important intellectual content. Both authors had full access to the data, contributed to the study, approved the final version for publication, and take responsibility for its accuracy and integrity.

Conflicts of Interest: Both authors have disclosed no conflicts of interest.

Funding/Support: This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Data Availability: All data generated or analysed during the present study are available from the corresponding author on reasonable request.

Ethics Approval: This study was approved by the Review Board of Eskişehir Osmangazi University, Faculty of Medicine (Ref: 25403353-050.99-E.38317). The need for patient consent was waived because of the retrospective nature of the study. All patients gave written informed consent for all treatments and procedures.

中文摘要

Wunderlich綜合徵的CT表現

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背景：Wunderlich綜合徵（WS）定義為無創傷腎或腎週出血。致病因素包括腫瘤、血管異常、囊性腎病和凝血障礙。CT是診斷WS的首選診斷方法。本研究旨在檢視WS患者的CT表現、病因、人口統計學特徵和治療結果。

方法：回顧分析2015年1月至2020年1月期間113例CT發現腎或腎週血腫的患者。排除101例有外傷史或腎活檢史患者後，其餘12名患者構成本研究組。

結果：納入研究的12名患者中，5名（41.7%）為女性，7名（58.3%）為男性。平均年齡53.2歲（範圍：7-81歲）。CT顯示7例患者（58.3%）出現腫塊病變，2例（16.7%）為假性動脈瘤，1例（8.3%）患者出現腎靜脈血栓形成。CT檢測到的腫塊中有2例（28.6%）是血管平滑肌脂肪瘤，1例（14.3%）是成人多囊腎病患者的出血性囊腫，4例（57.1%）是實性腫塊。4例有腫塊的患者中，3例接受手術治療。所有假性動脈瘤、1例無法手術的實性腫塊，和1例血管平滑肌脂肪瘤進行血管造影介入治療。

結論：WS是急性泌尿外科急症，有多種可能病因，部分更可能需要手術治療。CT在WS綜合徵的診斷和臨床處理中很重要，可以識別需要立即干預的血流動力學不穩定病例。

INTRODUCTION

Wunderlich syndrome (WS), first described in 1856 as renal and perirenal haemorrhage in patients without a history of trauma, is a life-threatening emergency. Clinically, symptoms are referred to as 'Lenk's triad', comprising acute flank pain, a palpable abdominal mass, and haemorrhagic shock. Haematuria is not an expected finding in patients.[1] Most renal neoplasms may cause WS, with renal cell carcinoma (RCC) and angiomyolipoma (AML) reported as the most common neoplastic causes. Other aetiologies include vascular abnormalities, including polyarteritis nodosa (PAN), aneurysms, arteriovenous fistulas, arteriovenous malformations, renal vein thrombosis (RVT), and coagulation disorders. Hereditary and acquired cystic kidney disease and renal infections are also associated with WS. Although ultrasonography is often the first method used for diagnosis, computed tomography (CT) is undertaken to make a definitive diagnosis. Magnetic resonance imaging (MRI) and angiography are other imaging methods that can be utilised.[2,3]

In this paper, we discuss the CT findings, aetiologies, and demographic and medical characteristics of patients diagnosed with WS, as well as the methods used in their treatment.

PATIENTS AND METHODS

The study was carried out in accordance with the principles of the Helsinki Declaration. Between January 2015 and January 2020, 113 patients with perirenal hematoma were examined using abdominopelvic CT. Excluded from the study were 101 patients with a history of trauma or kidney biopsy. The remaining 12 patients with spontaneous renal/perirenal hematoma constituted the study group. The demographic characteristics, CT findings, aetiologies revealed by CT, and the patients' follow-up and treatment data were recorded. Five (41.7%) of the 12 patients included in the study were female and seven (58.3%) were male. The mean age was 53.2 years (range, 7-81). All patients were diagnosed based on CT findings and clinical and pathological data.

The CT scans were retrospectively evaluated by two radiologists based on consensus. Both radiologists diagnosed that all 12 patients had WS. There was no case of disagreement between the two readers. CT imaging was performed using a 64-slice (Toshiba Aquilion 64, Tokyo, Japan) or 128-slice (Revolution EVO, GE Healthcare, Milwaukee [WI], US) multidetector CT scanner. The subjects were examined in the supine position with their arms extended above their heads. All CT examinations were performed using a routine

abdominal CT protocol, in which the image data were acquired from the dome of the diaphragm to the pubic symphysis after intravenous bolus administration of iodinated contrast medium with a 65-s delay to visualise the portovenous phase. Nine patients were first examined after a 35-s delay to visualise them in cases thought to be an arterial pathology. The intravenous contrast agent (1.5 mL/kg; Iopromide 370, Bayer Schering Pharma AG, Germany or Iohexol 350, GE Healthcare, US) was administered through an antecubital vein with an automatic injector at a rate of 3 mL/s.

Commercial software (SPSS Windows version 22.0; IBM Corp, Armonk [NY], US) was used for statistical analysis. A normality analysis was performed using the Shapiro–Wilk test. Descriptive statistics were presented as mean ± standard deviation for continuous data, and median and range with percentage values for discrete data.

RESULTS

Space-occupying lesions were seen on the CT images of seven patients (58.3%). Pseudoaneurysm formation was detected in two patients (16.7%) and RVT in one case (8.3%). For the remaining two patients (16.7%), the aetiology could not be determined by CT. One patient had end-stage renal disease and was on a routine haemodialysis programme. The other patient's history included coronary angiography, which had been performed 2 days earlier. There were findings consistent with bleeding diathesis in the examination of both patients, and therefore the aetiology of WS was recorded as coagulopathy.

Two of the space-occupying lesions on CT (28.6%) were AMLs (Figure 1), one (14.3%) was a haemorrhagic cyst in a patient with adult polycystic kidney disease (APCKD), and four (57.1%) were solid mass lesions. The demographic and CT data of the patients are summarised in the Table.

On the CT examinations, hematoma was observed to be on the right side in eight patients (66.7%) and on the left side in four patients (33.3%). Two patients had active extravasation. The cause of WS was AML in one of these patients and haemodialysis in the other. The widest diameter in the axial plane of the detected hematomas ranged from 40 to 161 mm (median, 81.5).

Three of the four patients with masses were surgically treated. Radical nephrectomy was performed in two patients, and partial nephrectomy in one patient. The remaining patient was considered inoperable; thus, the bleeding focus was embolised during angiography. The

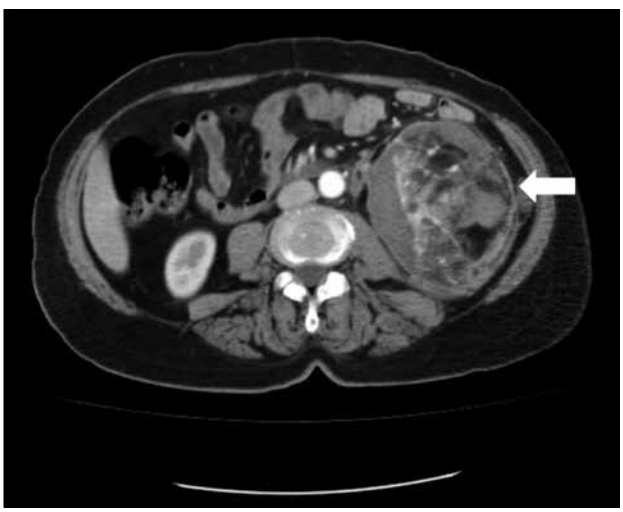

Figure 1. Axial contrast-enhanced arterial phase computed tomography image showing bleeding into the angiomyolipoma.

Table. Demographic data and computed tomography findings of our patients.

Patient No.	Age, y	Sex	Affected side	Aetiology on CT	Definitive aetiology	Mass size (largest diameter), cm
1	7	F	Right	Solid mass	Clear cell carcinoma	6
2	42	M	Left	Pseudoaneurysm	Pseudoaneurysm	
3	43	M	Right	Solid mass	Papillary RCC	5
4	77	M	Right	Solid mass	Clear cell carcinoma	1.5
5	74	M	Right	None	Coagulopathy after angiography	
6	81	F	Left	Angiomyolipoma	Angiomyolipoma	4.5
7	44	M	Right	APCKD	APCKD	4
8	57	F	Left	Angiomyolipoma	Angiomyolipoma	10
9	51	M	Right	Solid mass	Clear cell carcinoma	8.5
10	68	F	Left	None	Bleeding diathesis due to haemodialysis	
11	43	F	Right	Pseudoaneurysm	Pseudoaneurysm	
12	51	M	Right	Renal vein thrombosis	Renal vein thrombosis	

Abbreviations: APCKD = adult polycystic kidney disease; CT = computed tomography; RCC = renal cell cancer.

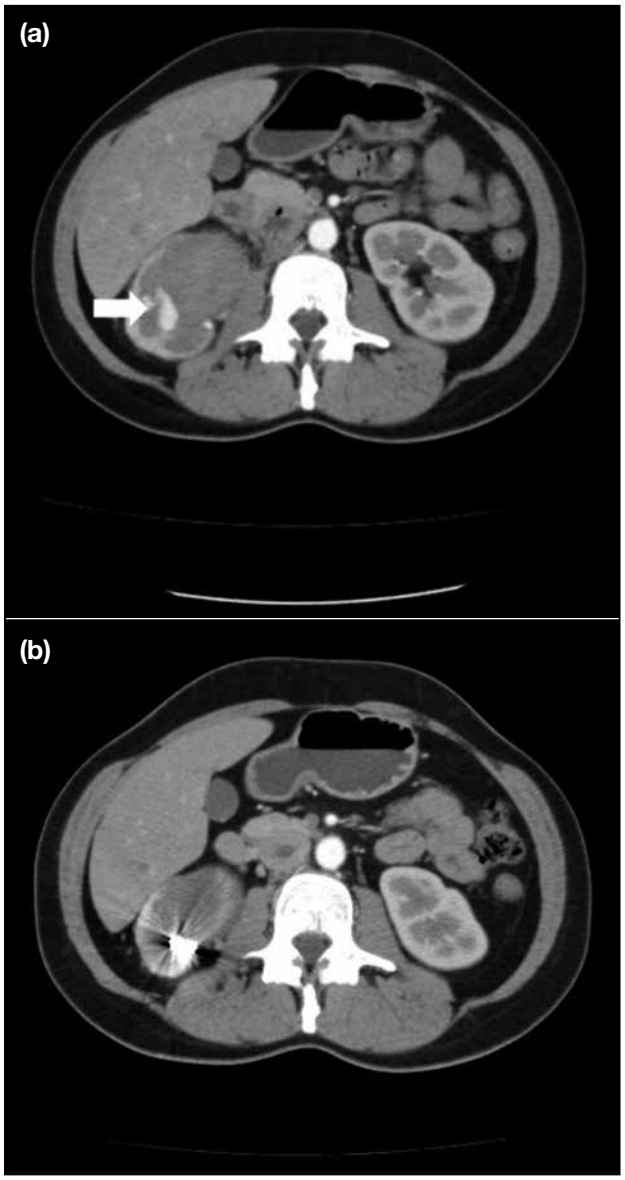

Figure 2. (a) Axial contrast enhanced arterial phase computed tomography (CT) image showing a pseudoaneurysm (arrow) in the right upper pole and associated haematoma. Diagnosis confirmed with digital subtraction angiography. (b) Axial contrast-enhanced arterial phase CT showing pseudoaneurysm after angiographic treatment in the same patient.

bleeding focus of two patients with pseudoaneurysms (Figures 2 and 3) and one of the AML cases were also angiographically embolised. The patients that developed haemorrhage due to cysts or after coronary angiography were discharged once follow-up imaging revealed improved clinical findings. Three patients died; one with end-stage kidney disease and two with haemorrhage due to AML. Thrombectomy was performed during the angiography on the patient with RVT in the interventional radiology unit. Recanalisation was achieved and followed up with anticoagulant therapy.

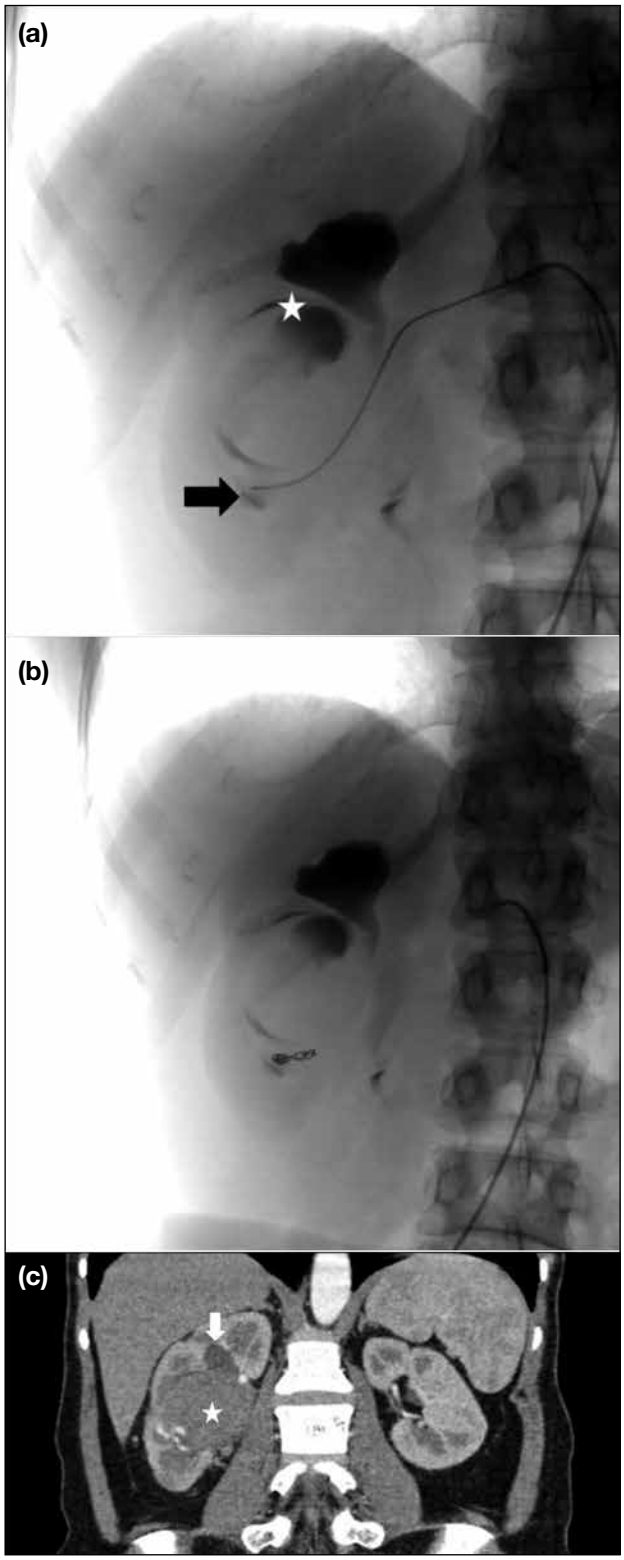

Figure 3. (a) Digital subtraction angiography image showing pseudoaneurysm (arrow) in the lower pole of the right kidney with associated extravasation and hematoma (star). (b) Digital subtraction angiography showing image taken after the procedure; the pseudoaneurysm is completely closed with a coil. (c) Coronal contrast-enhanced computed tomography image showing dilation upper pole calyces (arrow) due to compression of the renal pelvis secondary to hematoma (star).

All four patients with solid masses detected on CT were diagnosed with RCCs in the pathological evaluation (three clear cell carcinomas and one papillary cell carcinoma). According to the radiological, pathological, and clinical evaluations, the aetiology of WS was determined to be pseudoaneurysm in two patients, AML in two patients, RCC in four patients (three clear cell carcinomas and one papillary cell carcinoma), and one patient each with a cyst (patient with APCKD), post-angiography coagulopathy, coagulopathy due to end-stage kidney failure, and RVT.

DISCUSSION

WS is defined as non-traumatic, spontaneous renal/perirenal haemorrhage, which typically presents with flank pain, a palpable mass, and haemorrhagic symptoms. However, it is reported in the literature that only about 20% of patients experience these typical three symptoms together.4 In a previous study, the rates of abdominal/flank pain, haematuria, and haemorrhagic shock were 67%, 40% and 26.5%, respectively.[4]

In other studies, it has been reported that the male-to-female ratio in WS is approximately 6:5.[3,5] Similarly, in our study, WS was more commonly observed among men, with 58.3% males and 41.7% females. The mean age of the patients with WS was 53.2 years. In two studies in the literature, the mean age of the patients has been reported as 48.1 years and 46.8 years, respectively.[3,5] These findings are largely consistent with our study.

Different aetiologies can cause this clinical condition. In a meta-analysis conducted by Zhang et al,[3] tumours (61.5%) were determined to be the most common aetiology, followed by vascular diseases (17%). Similarly, in our sample, tumours were the aetiology in 58.3% of patients (two AMLs, one haemorrhagic cyst, four RCCs) and vascular causes in 25% (two pseudoaneurysm and one RVT). In the present study, RCCs and AMLs ranked first among tumours, which is in agreement with the literature.[3] AMLs are the most common benign solid lesions and RCCs are the most common malign lesions in the general population. WS is an emergency that requires a rapid diagnosis since it causes haemorrhagic shock and can lead to death. As treatment and patient management vary according to aetiology, determination of the aetiology is paramount. To this end, urgent diagnostic imaging is essential. Ultrasonography is often the first imaging method used in WS. However, due to the known inadequacies and disadvantages of ultrasonography in evaluating the retroperitoneum, a cross-sectional method, such as MRI or CT should also be used for a definitive diagnosis.[6] Both of these modalities have been reported to have high sensitivity in diagnosis.[7] In addition to conventional CT, dual-energy CT (DECT) can also be applied to the diagnosis of WS. In DECT, images are obtained using the data obtained by scanning the same anatomical region at two different kVp values (usually 80 and 140 kVp). The contrast resolution of the data obtained at low kVp values is high. It is possible to obtain virtual non-contrast series, iodine perfusion maps, and CT angiography images by taking advantage of the differences in K orbital binding energies of iodine and body tissues.[8,9] DECT angiography images with bone and calcific plaque removed may provide better success in showing active extravasation in WS. It may also contribute to treatment planning by showing compression of the renal parenchyma due to haematoma.[9] However, the necessity of having a double tube or an electronic kVp changeable tube limits its widespread use. CT should be the preferred method for the detection of renal/perirenal bleeding and the underlying cause due to its easier accessibility, shorter examination time during emergencies, and higher patient compliance and tolerance.[2,10] In all of our patients, CT examination was used for the definitive diagnosis.

AML is the cause of WS in about 30% to 35% of cases.[3,11] In our study, AML was detected as the underlying cause in two patients (16.7%). AML is considered to originate from perivascular epithelioid cells located around blood vessels. It contains different proportions of fat, muscle, and abnormal vascular tissue. There are two types of AML: the classic triphasic type and the epithelioid type. The classic type can be sporadic or accompanied by tuberous sclerosis, whereas the epithelioid type is less frequently seen and progresses in a more aggressive manner.[12,13] Studies report no difference between the two types in terms of causing WS.[13] On CT, the classic type of AML is visualised as heterogeneous mass lesion with high fat content and other components that are isodense. The epithelioid type may not be distinguishable from other mass lesions due to its low fat content.[14] Vascular structures with low elastin content in AML tend to form aneurysms. This trend increases as the size of the mass increases. The risk of AML haemorrhage depends on the size of the lesion and the diameter of the aneurysm, increasing when the diameter is >4 cm.[15] In almost all cases of WS secondary to AML, CT and MRI can be used to identify the underlying mass, and treatment is performed with catheter embolisation

or, if embolisation fails, with surgery. Partial or total nephrectomy can be performed depending on the size of the mass.[15] In our study, the mass sizes in the two patients with AML were 4.5 cm and 10 cm. The bleeding focus was angiographically embolised in one of these patients while the other patient died before intervention.

RCC is the second most common cause of WS, reported as being associated with 26% of WS cases.[3] It is noted in the literature that WS is only seen in 0.3% to 1.4% of patients with RCC but due to the high incidence of RCC, it presents as the most common malignancy causing WS.[16] Unlike AML, tumour size is not a good predictor of haemorrhage. When the underlying cause is RCC, treatment is usually radical nephrectomy.[3,17] RCC has three major subtypes: clear cell (70% of all RCCs), papillary (10%-15%), and chromophobe variants (4%-6%). Clear cell RCC is the most common subtype that causes WS due to its hypervascularity and rapid growth. In 60% to 80% of sporadic cases, the von Hippel–Lindau gene is inactivated, which is considered to activate various vascular and somatic growth factors and cause irregular vascularity, thus leading to haemorrhage.[18] In our study, the most common cause of WS was found to be RCC, which was seen in four (33.3%) patients. This rate is largely similar to that in the literature. Similar to the literature on RCC subtypes, the pathology was clear cell carcinoma in three patients and papillary carcinoma in one patient.[2] While three of our patients were treated surgically, the bleeding focus was angiographically embolised in the remaining patient because he was not a surgical candidate.

It is reported that vascular aetiologies are associated with approximately 20% to 30% of WS cases. These include arterial abnormalities, including PAN, renal artery aneurysm, and pseudoaneurysm; and venous factors, such as RVT, renal arteriovenous malformation, and arteriovenous fistula.[11] PAN is the most common vascular cause of WS. PAN is a vasculitis inducing multifocal necrotic areas in medium-size and small arteries, especially the renal arteries. In patients with PAN, diffuse enlargement in the kidneys, loss of the corticomedullary junction, and multiple parenchymal infarcts and microaneurysms can be seen on CT. The demonstration of microaneurysms is important in distinguishing PAN from acute pyelonephritis. Angiography is generally used in the diagnosis and treatment of PAN when it is suspected as the cause of WS.[19]

The incidence of pseudoaneurysms and true renal artery aneurysms has been reported as 0.09%.[20] Iatrogenic causes, inflammation, infection, and vasculitis may play a role in the aetiology of pseudoaneurysms. CT is the preferred method for the diagnosis of ruptured aneurysms because it can reveal massive perinephric haemorrhage and extravasation foci within the hematoma. Patients with ruptured aneurysms are usually treated angiographically.[2] We detected pseudoaneurysms in two of our cases (16.7%) on CT, which was confirmed by angiography that was subsequently used for treatment. Compared to the literature, our rate of pseudoaneurysms was high. RVT is another important vascular pathology that can cause WS. RVT may develop secondary to hypercoagulation, dehydration, and renal masses. In the imaging of patients with RVT, an enlarged kidney or perinephric oedema in the renal sinus can be seen. Filling defects in the renal vein can be demonstrated by contrast-enhanced CT or MRI. Generally, patients with RVT clinically present with haematuria, flank pain, and loss of renal function.[2,21] RVT was detected in one of our patients (8.3%), who had impaired renal function, haematuria, and flank pain, which is consistent with the literature. Studies in the literature report that WS secondary to RVT is rare.[2] The high rate of pseudoaneurysms and the presence of RVT among our patients may be due to our institution being a tertiary health centre, to which such patients are referred for angiographic treatment.

Renal cysts often rupture into the pelvicaliceal system; perinephric rupture is rarely detected. Simple and haemorrhagic cysts are common causes of WS. Intracystic haemorrhage is common in APCKD but secondary perinephric bleeding has rarely been reported.[22] Among the causes of cyst rupture, intracystic infection or bleeding are common, and the risk increases with increasing cyst size.[18] In some cases, large hematomas may make it difficult to detect the underlying cause by compressing the ruptured cyst. Follow-up CT imaging may be required to make a diagnosis. Patients with cyst rupture are usually treated conservatively, and antibiotic therapy can be added to treatment if necessary.[23] In the current study, APCKD was seen in one of our patients, who did not have any complications during the follow-up and did not require any surgical or angiographic procedure.

Coagulation disorders are a heterogeneous group of diseases. The literature contains studies that are mostly in the form of case reports indicating coagulation disorders as a rare cause of WS.[24,25] In addition, there are other case reports showing that oral anticoagulant

therapy, which is widely used today, can cause WS.[26] It is known that patients with chronic kidney disease who undergo haemodialysis have a predisposition to platelet dysfunction and bleeding secondary to endothelial abnormalities.[27,28] Among our cases, WS developed due to coagulopathy in one patient after coronary angiography and in another patient that was receiving haemodialysis.

Other causes of WS include infectious diseases, such as acute pyelonephritis, renal abscess, and emphysematous pyelonephritis. It is reported that infections result in WS at a rate of approximately 5% to 10%. The risk of WS is higher in diabetic patients. Parenchymal necrosis secondary to infection, inflammation-related erosion of the renal vessels, and intravascular thrombosis are considered to play a role in its pathogenesis. Idiopathic WS is responsible for 5% of cases.[2,3]

CONCLUSION

WS is an acute urological emergency with an aetiologically broad spectrum. The two most common causes of WS are renal neoplasms and vascular pathologies. Emergency surgery is required in hemodynamically unstable cases. CT has an important place in diagnosis, determination of the underlying aetiology, and management of patients. Underlying benign pathologies can be successfully detected using CT, thus avoiding unnecessary interventional procedures.

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