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Wunderlich syndrome secondary to acute pyelonephritis: A case report

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Abstract
Wunderlich syndrome is a rare clinical entity. It is characterized by an acute onset of spontaneous hemorrhage into subcapsular, perirenal and/or pararenal spaces. Lenk triad (flank pain, flank mass and hypovolemic shock) which is classically associated with this condition is seen only in a small subset of cases. Common conditions leading to Wunderlich syndrome includes Renal neoplasms& Renal vascular anomalies. Other rare causes include Renal infections, cystic diseases, calculi, renal failure and coagulation disorders. Multiphasic CT & MRI imaging is used in better detection, localization and characterization of the underlying cause and facilitate optimal management. Imaging modalities like USG is used as follow up investigation. Treatment modalities include Conservative management, Arterial embolization and surgical procedures. In this article we are presenting a case of Acute pyelonephritis presenting as Wunderlich syndrome.

Keywords: Wunderlich syndrome, renal artery aneurysm, trans arterial embolization

Introduction
Wunderlich syndrome is a clinical condition where there is a spontaneous hemorrhage without any antecedent trauma into subcapsular, perirenal, and/or pararenal spaces. Bonet was the first to describe this clinical condition as nontraumatic spontaneous hemorrhage. Carl Wunderlich defined it as spontaneous renal capsule apoplexy resulting in subcapsular or perirenal hemorrhage in patients without antecedent trauma [1, 2]. Lenk triad which is characteristic of this condition is present only in 20% of cases. Lenk triad consist of flank pain, flank mass and hypovolemic shock. Flank pain is seen in 67% of patients, hematuria in 40% cases, flank mass and hypovolemic shock (27% cases) may occur in isolation or in concert [3].
Renal neoplasms and Vascular anomalies are predominant causes of Wunderlich syndrome [2, 5]. Angiomyolipoma among benign neoplasm and Clear cell Renal cell carcinomas among malignant neoplasm are most common causes. Among vascular anomalies Renal artery aneurysms, pseudoaneurysms, vascular malformations, vasculitis syndromes are common causes [2, 5]. Other rare causes include Hereditary & acquired renal cystic disease, renal failure, calculi, infections, pregnancy, coagulation disorders and iatrogenic. Extrinsic compression of kidney will lead into condition called PAGE kidney which is secondary hypertension due to activation of Renin angiotensin aldosterone system.
Multidetector CT & MRI is used for detecting hemorrhage and identifying cause [6, 7]. Diagnostic modalities like USG, is used for follow up care. Interventional Radiologist have important role in Vascular procedures like Arterial embolization in case of active bleed [8]. Treatment modalities include Conservative management, arterial embolization, surgery.

Case presentation
Index case is a 47yr old male patient with history of Type 2 Diabetes Mellitus since past 10 years on Oral hypoglycemic drugs. He had history of Left lower limb cellulitis 1 year back for which he underwent medical management. For similar complaint he had readmission 4 months back and underwent debridement. He had Right Epididymo-orchitis 3 months back, for which he was treated with intravenous antibiotics and supportive treatment.

In present admission patient had complaints of Right loin pain, low grade fever and generalized weakness. Patient was initially evaluated in Emergency room, his vitals were BP-100/70 mmHg, Pulse rate-100 /min, Respiratory rate-20cpm, SpO2-98% on room air. Physical examination showed tenderness in Right renal angle. Chest X ray was unremarkable. CT KUB plain showed acute perinephric hematoma of 30 mm thickness seen (80x30 mm (CC x TR)) causing compression over mid & lower poles of right kidney, Gall bladder calculus, Bilateral perinephric fat stranding. His laboratory values showed Hb- 12.9 gm/dl, TLC- 14000 cells/ microliter, Platelet count- 1.78 lacs/ microliter, Urea- 67 mg/dl, Creatinine- 1.1 mg/dl, INR-1.03. LFT was normal, Urine routine examination showed Albumin2+, Sugar 3+, Pus cells- Plenty/hpf, RBC- 20-25/hpf, Leucocyte, Granular cast & Bacteria present. Nephrology and Urology opinion was taken. Patient was started broad spectrum intravenous antibiotics after sending appropriate cultures.

CT Intra Venous Urography was done on Day 3 (Fig 1,2,3) which showed Non enhancing acute perinephric subcapsular hematoma of 30 mm thickness seen (80x30 mm (CC x TR)) causing compression over mid & lower poles of right kidney. Bilateral perinephric fat stranding. Gall bladder calculus 9mm seen in neck. Right adrenal lipoma 10mm. Pain abdomen has settled by day3. Patient vitals were stable and Renal parameters were within normal range. Blood cultures were sterile. Urine culture showed growth of Enterococcus faecium and antibiotics were changed as per culture sensitivity.

On Day 4 patient had Hb-11.9 gm/dl and repeat USG Abdomen was done which showed 56x25x50 mm, vol = 37 ml, focal, collection in subcapsular region of mid-lower pole with perilesional inflammatory change (Mild increase in hematoma size). Repeat INR was 1.05, Bleeding time & Clotting time was normal. ANA profile & p- ANCA was negative. Patient vitals and urine output were closely monitored and plan was to surgical intervention in case of worsening Renal parameters (PAGE kidney). On Day 5 patient vitals were stable with Hb- 12.5 gm/dl and no loin pain and normal renal function tests. Patient was discharged and plan was to do follow up in Outpatient department.

Patient was followed up on Outpatient department. His BP recordings were within normal range. Blood investigations done after 2 weeks showed Hb 13.4 gm/dl, TLC 11640 cells/ microliter, Platelet count 3.72 Lacs/ microliter, Blood urea 22.92 mg/dl, Serum Creatinine 0.6mg/dl. Repeat Non contrast CT Abdomen (Fig 4,5,6) was done 4 weeks after discharge which showed Liquefication of Right perinephric hematoma.

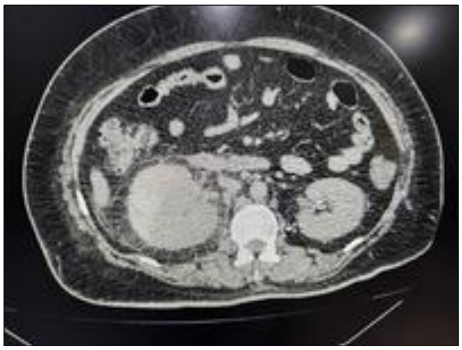

Fig 1: Axial non enhanced image of Abdomen shows hyperdense right perinephric hematoma compressing kidney

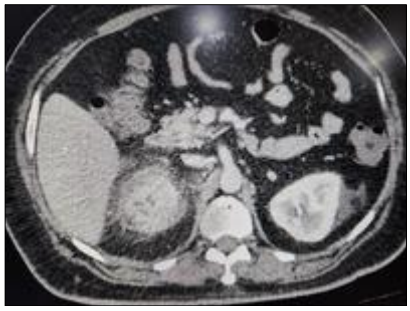

Fig 2: Axial enhanced image shows Right non enhancing perinephric hematoma. Few tiny hypodense striated foci seen in upper pole

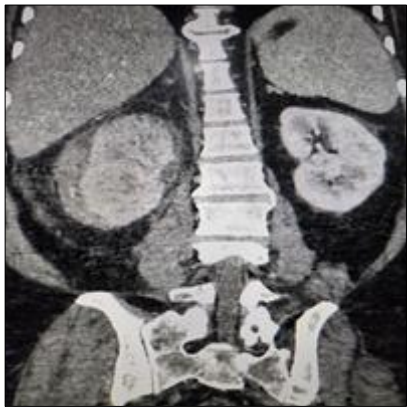

Fig 3: Coronal contrast image of Abdomen showing Right perinephric hematoma compressing kidney

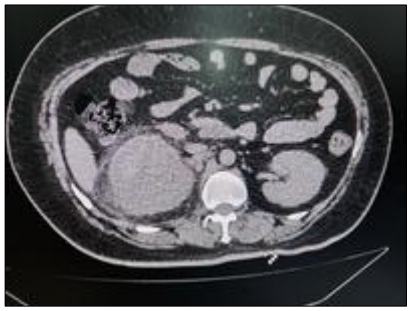

Fig 4: Follow up Axial non contrast image of Abdomen shows liquefication in Right perinephric hematoma

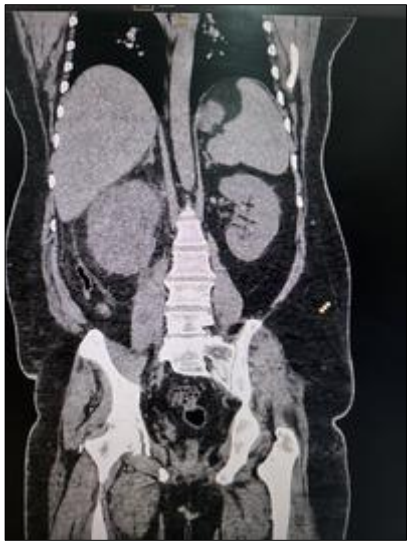

Fig 5: Follow up Coronal non contrast image of Abdomen shows liquefication in Right perinephric hematoma

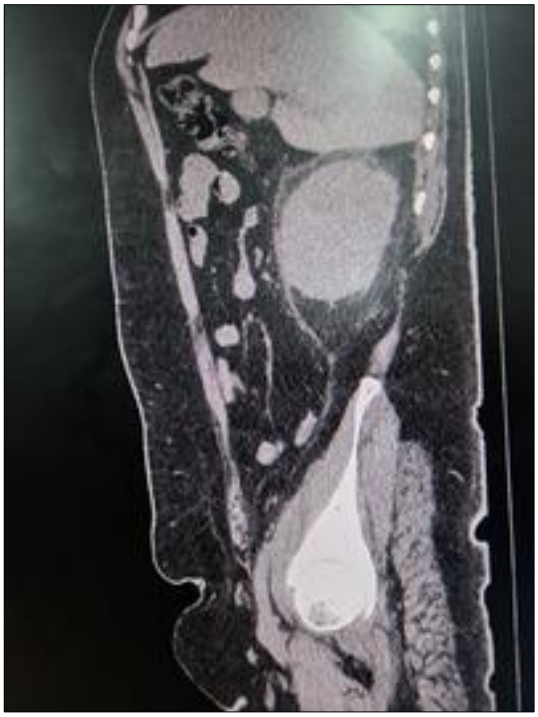

Fig 6: Follow up Sagittal non contrast image of Abdomen shows liquefication in Right perinephric hematoma

Discussion

Wunderlich syndrome as stated above is an acute onset of spontaneous renal hemorrhage into subcapsular, perirenal, and/or pararenal spaces without history of antecedent trauma. Lenk triad that is flank pain, flank mass and hypovolemic shock is characteristic of this condition, it is seen only in 20% of cases. Causes for this condition includes Renal neoplasms, renal vascular diseases, infections, cystic disorders, calculus, hematological & coagulation abnormalities, renal failure, pregnancy, recreational drug use and idiopathic.

Neoplastic causes accounts for nearly 60-65% of total cases of Wunderlich syndrome. Angiomyolipoma contribute to 40% of total cases of Wunderlich syndrome. 15-20% of Angiomyolipoma cases manifest as spontaneous perirenal hemorrhage. Tumor size more than 4cm and aneurysm size more than 5mm has higher risk of rupture [9]. All histologic subtypes have increased risk of Wunderlich syndrome. Multidetector CT or MRI is helpful in diagnosis. Trans arterial embolization is useful in treatment [10]. In case of recurrence after embolization other procedure like nephron sparing surgery can be considered. In cases where there is profuse bleeding total nephrectomy can be considered [11].

Renal cell carcinomas contribute to 30-35% of total WS cases. Only 1% of RCC have tendency to bled spontaneously [12, 13]. Hyper vascular Clear cell RCC is more likely to cause WS than other non-clear cell subtypes. Inactivation of von Hippel- Lindau gene in clear cell RCC leads to unregulated activation of somatic and vascular growth factors leading to hypervascularity [14]. Additional predisposing factors for spontaneous rupture are tumour related arteriovenous shunting, large tumour size, intra tumoral necrosis with increased pressure, extension into adjacent vessel and an acute increase in renal vein pressure. Treatment of RCC with perirenal haemorrhage depends on clinical status of patient. Stable cases are treated conservatively, TAE can be considered to control bleeding, Definitive treatment depends of stage of RCC, options include cutaneous ablation and partial or radical nephrectomy [15].

Vascular causes contribute to 20-25% of total WS cases. Polyarteritis nodosa accounts for 4-13% of total WS cases [16, 17]. CT &MR Angiography are helpful in diagnosis. TAE is considered as preferred treatment option. TAE is prophylactically performed where RAA > 2cms. RAA less than 2 cm need to follow up on regular intervals to check for progression. Catheter angiography is helpful in patients with renal arteriovenous malformations where CT & MR angiograms are nonconclusive. Absolute alcohol for small AVM and coil embolization for large AVM is done. Renal vein thrombosis is treated with anticoagulation, catheter directed thrombectomy and IVC filters [18].

Renal infections account for 5-10% of total cases of WS. These include Acute pyelonephritis, Emphysematous pyelonephritis and renal abscess. Renal infections in background of Diabetes increase the risk for developing WS. CT or MR findings typical for renal infection are heterogenous renal parenchyma or striated appearance, renal abscess and renal parenchymal or pelvicalyceal air foci and an associated perirenal or subcapsular hematoma. Treatment options include conservative management with antibiotics, percutaneous drainage of abscesses or hematomas, TAE or surgery if having active ongoing bleed.

Systemic anticoagulation and conditions like haemophilia are associated with WS. These hematomas are generally massive large volume bleeds. Treatment options include Anticoagulation reversal, removal of offending drug. Renal cystic diseases and their rupture is also associated with WS. Other rare causes include Hydronephrosis secondary to ureteral obstruction, recreational drugs like cocaine, cannabis &amphetamines, and idiopathic.

Conclusion

Wunderlich syndrome is a rare condition. Lenk triad is not seen in majority of cases. There are multiple clinical conditions which can lead to WS. Multidetector CT or MRI is key in diagnosing, localizing and planning for management. Interventional radiology is key for planning Endovascular procedures. USG is useful as a follow up investigation. Treatment options like TAE are preferred. In case of failure of conservative management and TAE, nephron sparing surgery and radical nephrectomy are done.

Declarations

Abbreviations: RCC= Renal cell carcinoma, RAA= Renal artery aneurysm, CT= Computed Tomography, MRI= Magnetic resonance imaging, WS= Wunderlich syndrome, TAE= Trans arterial embolization, AVM= Arteriovenous malformation.

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