Features and complications of nephroptosis causing the loin pain and hematuria syndrome

A preliminary report

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ABSTRACT

Objective: Loin pain with and without hematuria is a serious clinical problem that is most difficult to diagnose and treat. The underlying symptomatic nephroptosis was disparaged long ago, the loin pain hematuria syndrome is doubted and the link between the 2 conditions remains unknown. The lack of demonstrable pathology on all supine imaging has caused disbeleive. This article aims to demonstrate underlying symptomatic nephroptosis features and complications including loin pain hematuria syndrome and discuss the patho-etiology mechanism.

Methods: Observational study and thorough investigations of patients presenting with loin pain and hematuria showed that all supine standard and ancillary imaging was normal. Upright intravenous urography and isotope renography, however, showed features of symptomatic nephroptosis causing the initial intermittent renal pain. Retrograde pyelography demonstrated late organic complications of symptomatic nephroptosis causing pain and hematuria of loin pain hematuria syndrome. Observations detected the illusive overlooked anomalies demonstrable on photographs.

Results: All supine standard and ancillary imaging appeared deceptively normal in patients presenting with recurrent episodes of loin pain with or without hematuria. Upright imaging demonstrated gross nephroptosis with pelvi-ureteric junction kink, causing the initial intermittent and later organic obstruction pain. Renal pedicle stretch or twist was also demonstrable on upright intravenous urography and isotope renography as causes of ischemic renal pain. Retrograde pyelography demonstrated the organic renal damage of symptomatic nephroptosis when complicated into loin pain hematuria syndrome. Pyelocalyctaisis with eroded papillae, peritubular backflow and intrarenal extravasation of contrast medium with venous leakage, showed the renal site and cause of hematuria fulfilling the definition of loin pain hematuria syndrome. "Auto-nephropexy" and "sympathetic nephroplegia" were illusive neuro-ischemic findings that took years of follow up observation to affirm in cases of symptomtic nephroptosis complicated into loin pain hematuria syndrome. Other complications included segmental infarction and renal atrophy "auto-nephrectomy".

Conclusions: The presented photographs demonstrate that loin pain and hematuria have real heterogeneous patho-etiology of ureteral kink obstruction and pedicle stretch or twist ischemia of symptomatic nephroptosis with intermittent and irreversible stages. The overlooked anomalies on all supine imaging are demonstrable on upright imaging and retrograde pyelography with pyelocalyctaisis that may affect both kidneys via sympathetic neuropathy. The reproducible evidence affirms that pain is genuine and symptomatic nephroptosis may be complicated into loin pain hematuria syndrome.

Keywords: Loin pain hematuria syndrome, nephroptosis, obstruction, ischemia, neuropathy, auto-nephropexy, auto-nephrectomy, sympathetic nephroplegia.

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Loin pain with or without hematuria is most serious and a difficult clinical problem in Najran, Kingdom of Saudi Arabia. It affects mostly young females of Saudi and Yemeni origin at their 2nd-4th decade of life. However, few females from other provinces, expatriates and males have been encountered among a series of 190 patients prospectively studied over the last 9 years, representing the largest single group of patients' referred to the Urology Department, King Khalid Hospital, Najran, at a rate of 1.76 cases per month. Najran is in the southern province of the Kingdom of Saudi Arabia at the border of Yemen with mixed population from both countries and expatriates. The hospital provides the main urology service in the region. This report reflects the author's experience based on reviewing the findings of the long observational study that aimed at understanding the disorder, verifying its genuineness and identifying its real patho-etiology.

The main management problem of loin pain was the lack of demonstrable pathology on repeated imaging, when supine. The underlying symptomatic nephroptosis (SN) though well known,[1] was disparaged[2] and loin pain hematuria syndrome (LPHS) though well documented,[3] its existence may be doubted[4] and both are extremely problematic to manage.[3-6] Demonstrable renal pathology of loin pain and hematuria was invariably lacking on all supine imaging of the received protocol.[3-6] Urinary tract infections (UTI) may affect a few patients during the occasional episodes but UTI, stones and organic causes play no role in the pathogenesis of LPHS.[3-6] Many complex ramifying management problems of SN and LPHS are well known[2-6] but have no solutions. Some of the problems were communicated[2,7] and the illusive overlooked link of SN with LPHS was pointed out recently.[8]

This article aims to demonstrate the patho-etiology features of SN and complication into LPHS as genuine causes of loin pain and hematuria. The photographic evidence demonstrable on upright imaging and retrograde pyelography is visible and reproducible by other researchers and colleagues. Identifying the patho-etiology of loin pain and hematuria may revive interest to help future adequate management of young patients suffering from the incapacitating genuine pain of SN and LPHS.

Methods. All patients presenting with loin pain with or without hematuria during the last 9 years were entered into a prospective observational study and underwent thorough clinical, laboratory and imaging investigations. Repeated standard imaging was invariably normal, when supine. However, 190 patients demonstrated SN of > 2 vertebrae on upright imaging. Of whom 36 (18.9%) patients developed recurrent episodes of painful hematuria for which no organic pathology was detected on all standard and ancillary imaging, when supine. The study aimed to affirm genuineness of loin pain and hematuria and identify its real patho-etiology. Reviewing the data of 9 years study revealed many clinical and radiological findings that is indeed incredible for a discarded disorder but may be easier to believe when the underlying patho-etiology of pain and hematuria is demonstrated on imaging photographs.

Imaging included grayscale ultrasound (US) and intravenous urography (IVU) and were carried out repeatedly on all patients. Ancillary imaging was carried out on all cases suffering from severe pain and hematuria episodes and included computer axial tomography (CAT), magnetic resonance imaging (MRI) or arteriography (MRA), doppler ultrasound, and ^{99}mTc DTPA isotope renography (IR) scans. Grayscale ultrasound, IVU and IR were carried out at supine and upright postures. Cystoscopy and retrograde pyelography (RGP) were carried out for localizing the side and site of hematuria in cases who gave informed consent. Upright IVU and IR imaging and RGP demonstrated the overlooked patho-etiology features and complications causing pain and hematuria while all other imaging missed the detectable pathology. Long term follow-up observations identified the illusive overlooked anomalies of SN complicated into LPHS. Investigations included regular urine analysis and culture that were mostly negative for UTI, so were the tests for Tuberculosis and Brucellosis. Renal function tests were always normal. Serum immunoglobulines, compliment factors C3 and C4 were normal in all but 5 of the 36 LPHS cases. Consumption coagulopathies affected 3 cases presenting with life-threatening hematuria episodes and requiring massive blood transfusions. All cases were thoroughly investigated at multiple specialist clinics, both at our hospital and elsewhere, for the bizarre multiple associated splanchnic symptoms (MASS) that accompany loin pain and hematuria. Attending physicians excluded all relevant organic causes of pain and hematuria, and possible causative personality and psychiatric disorders.

Results. All standard and ancillary imaging appeared normal, when supine. Comparing supine to erect IVU films demonstrated nephroptosis of >3 vertebrae **(Figures 1-4).** Nephroptosis constantly affected the right kidney either alone **(Figures 1 & 2)** or as part of a bilateral drop **(Figures 3 & 4).** Upright imaging also demonstrated features and complications of SN causing pain and hematuria. Ureteral kink at the pelvi-ureteric junction (PUJ) caused urinary stasis, distended renal pelvis and obstruction pain **(Figures 3 & 4)** that may progress into organic PUJ obstruction in a few patients many years later. However, ureteral kink obstruction did not explain the most agonizing loin pain that was not relieved by lying supine and took several days of bed

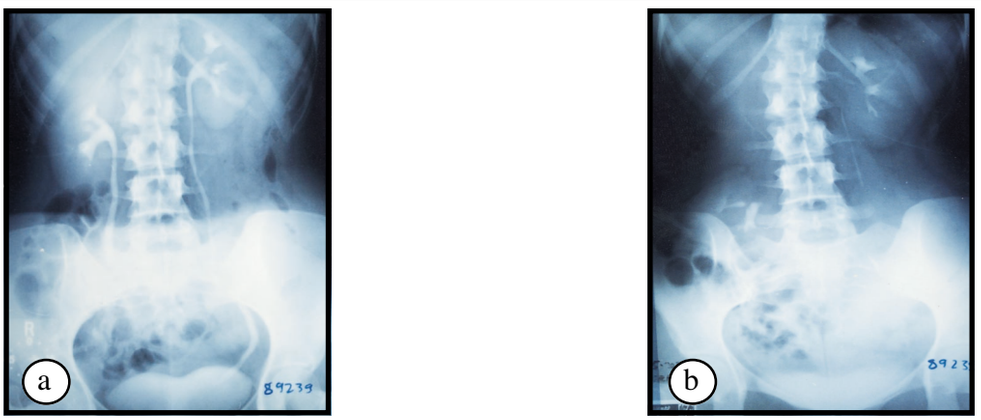

Figure 1 - Intravenous urography films showing patient **(a)** supine and **(b)** erect. Supine film shows normal kidneys. Erect film shows normal left kidney but demonstrates right nephroptosis of 4 vertebrae with rotation twist of the right kidney around its pedicle depicted by the changed appearance of renal contour and calyx pattern. the neuro-vascular renal pedicle is unseen but pedicle stretch and twist are depicted when the right kidney's normal position at (a) is compared to nephroptosis at (b).

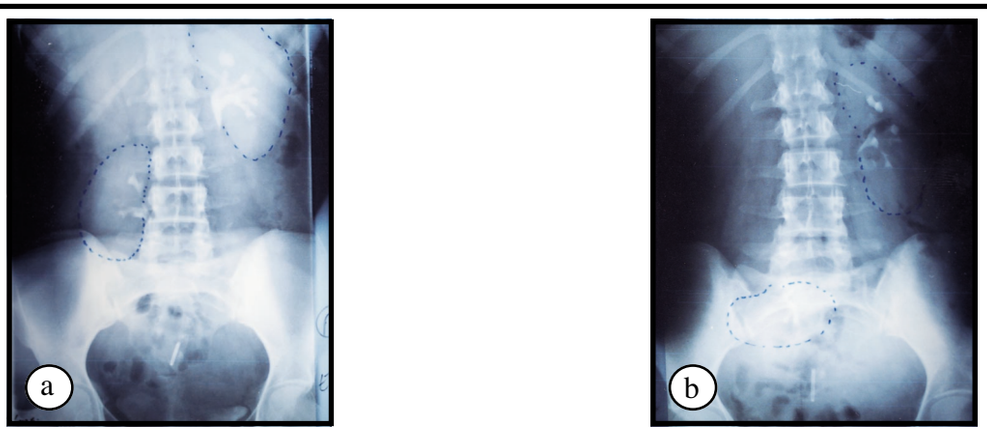

Figure 2 - Intravenous urography films showing patient **(a)** supine and **(b)** erect. Both kidneys were reported normal at (a) but the right kidney postion is fixed 2 vertebrae lower than normal position shown on a previous IVU. Upright gross nephroptosis of 4+ vertebrae dropping the right kidney to pelvis with twist rotation around pedicle is demonstrable on (b), while the left kidney remains at normal position. The mobile right kidney can no longer move upward to normal renal bed but the demonstrable mobility differentiates nephroptosis from an ectopic kidney.

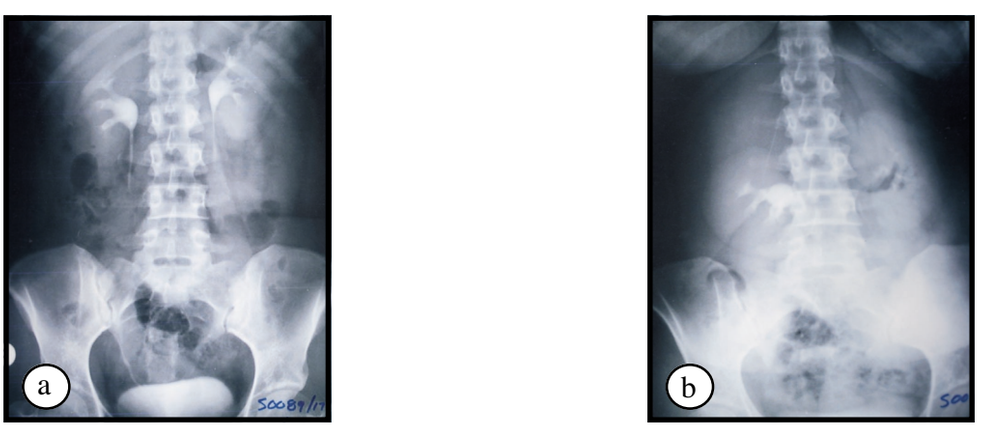

Figure 3 - Intravenous urography films showing patient **(a)** supine and **(b)** erect. Normal renal position at (a) and bilateral nephroptosis of 3 vertebrae at (b). The right kidney shows pelvi-ureteric junction kink obstruction with distended renal pelvis on the upright film (b). Pelvi-ureteric junction obstruction disappears on supine posture (a) but a few cases may progress to the classical picture over the years.

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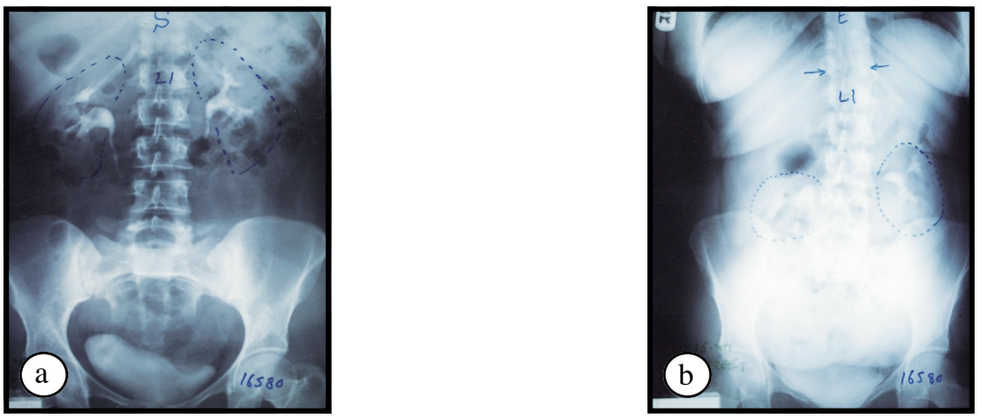

Figure 4 - Intraveneous urography films showing patient **(a)** supine and **(b)** erect. Upright film (b) demonstrates bilateral nephroptosis of 3 vertebrae and renal rotation while the supine film (a) is perfectly normal.

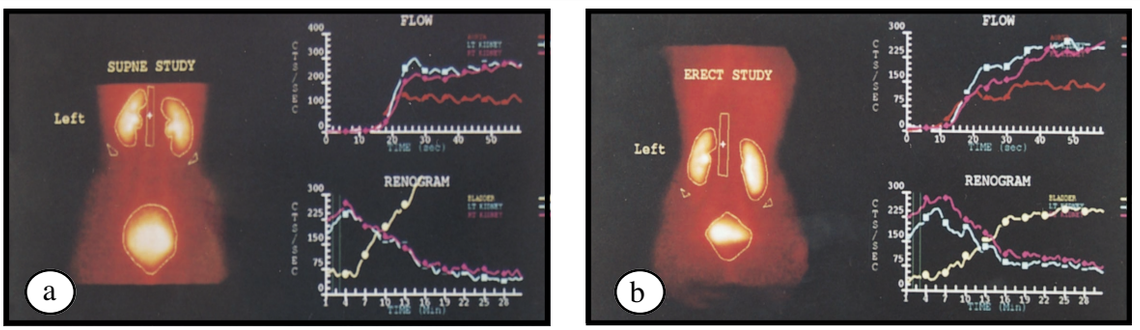

Figure 5 - 99Tc DTPA isotope renography scan showing patient **(a)** supine and **(b)** erect. Normal despite bilateral nephroptosis with over 21% split function impairment of blood flow and glomerular filtration with a delayed wash out phase at the sitting up posture (b).

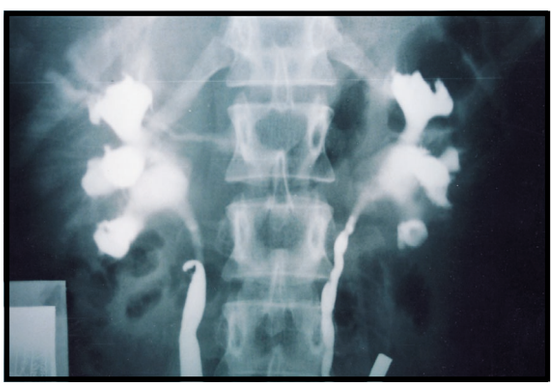

Figure 6 - Retrograde pyelography (RGP) shows pyelocalyctaisis of the right kidney with papillary erosion, peri-tubular backflow and intra-renal extra-vasation of contrast medium with leakage into the renal vein. Early involvement of the left kidney is shown. The right kidney had nephroptosis of 3 vertebrae but became spontaneously fixed during the course of illness "auto-nephropexy". The patient sufffers from most agonizing left loin pain while hematuria episodes originate from the right kidney. "Sympathetic nephroplegia" may explain this finding (see text).

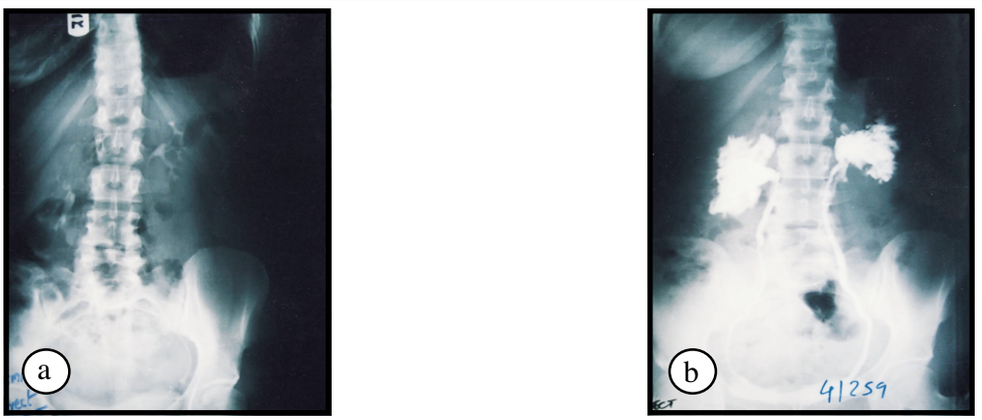

Figure 7 - (a) Intravenous urography and **(b)** retrograde pyelography of the same patient suffering from bilateral loin pain and gross hematuria episodes. The intravenous urography (a) and all other imaging appeared normal while and retrograde pyelography (b) demonstrates this gross bilateral pyelocalyctaisis damage. This patient had right ptosis only. Note that retrograde pyelography (b) was carried out on the standing conscious patient using 5-10 ml of contrast medium without undue injection pressure. Such contrast extra-vasation neither occurs in normal kidneys nor in an obstructed hydronephrotic kidney.

rest and opiates to resolve, suggesting neuropathic ischaemic renal pain induced by renal pedicle stretch. Though ptotic pedicle stretch of renal vessels and nerves did not show directly on IVU, it was demonstrable by the renal drop measured as the number of lumbar vertebrae or cm **(Figures 1-4).** Renal twist was demonstrable by the changes of renal contour and calyx pattern of a ptosed kidney rotated around its pedicle that mostly involved the right kidney **(Figures 1-4).** The demonstrable obstructive, ischemic and neuropathic causes of renal pain may remain intermittent "functional" for years, and were totally missed on all supine standard and ancillary imaging. Although torsion is a known mechanism of ischemia, pedicle stretch remains a subjective cause of ischemic pain requiring further objective affirmation. Isotope renography also demonstrated the obstruction and ischemia of SN. Split renal function with impaired blood flow, decreased glomerular filtration rate and obstruction urinary stasis were demonstrable on IR imaging on comparing sitting up to supine posture. The response to frusemide and results of IR were mostly reported normal despite a drop of >21% affecting the right ptosed kidney **(Figure 5).** Upright ischemia and obstruction variably contributed to the split results of IR. The ischemic changes were most prominent on other IR imaging and were also demonstrable on doppler ultrasound by the increased resistive index despite the novelty of the investigation at upright posture. Retrograde pyelography demonstrated the renal complication long before it appeared on

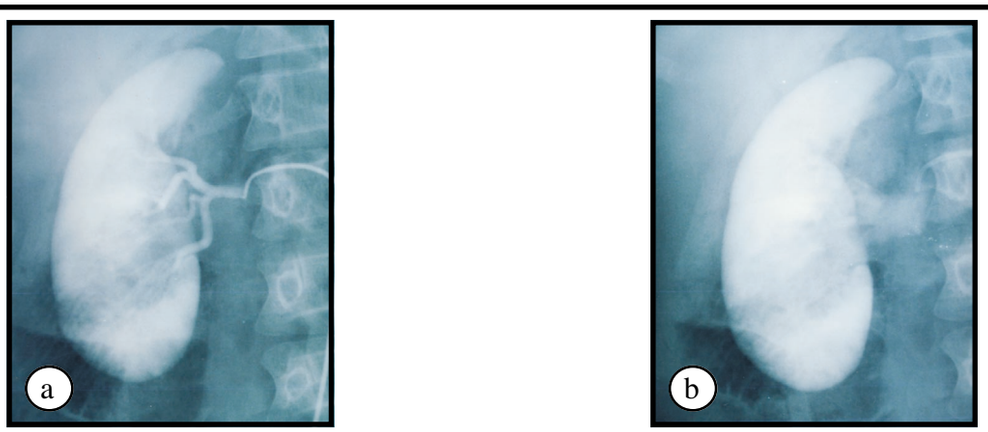

Figure 8 - Conventional renal arteriography in **(a)** arterial and **(b)** venous phase of a conventional renal arteriography demonstrating right upper segment infarction in a patient who presented with a life-threatening hematuria episode after years of recurrent right loin pain of symptomatic nephroptosis. Imaging using ultrasound, intravenous urography, retrograde pyelography missed this finding.

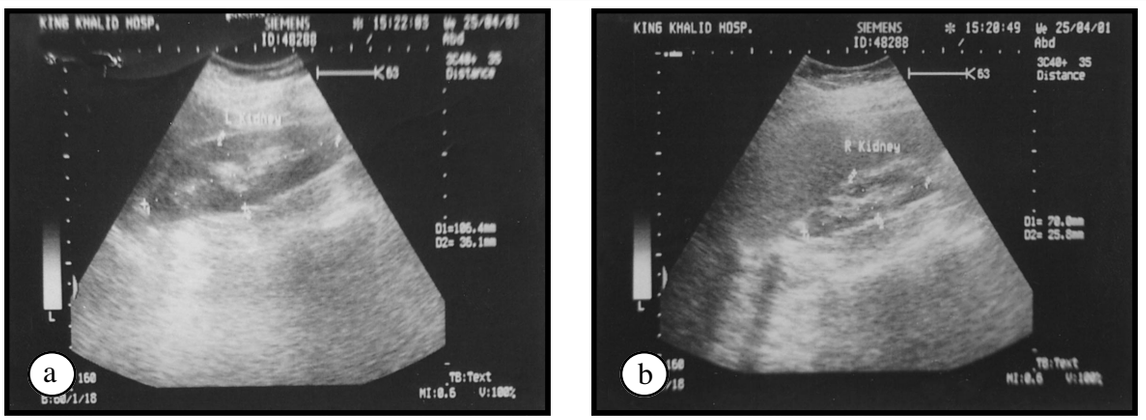

Figure 9 - Grayscale ultrasound of **(a)** left and **(b)** right kidneys of a 34-years-old female, showing 50% reduction of renal mass of a previously right ptosed kidney that has undergone "auto-nephropexy" and is undergoing insidious atrophy "auto-nephrectomy".

arteriography while other imaging including MRA appeared deceitfully normal, overlooking the remarkable anomalies. The organic renal damage of pyelocalyctaisis initially affected the upper pole of right ptosed kidney localizing the site of hematuria of LPHS complicating SN. **Figure 6** demonstrates pyelocalyctaisis that may progress to affect all the renal calyces of the right ptosed kidney with papillary erosion atrophy, peri-tubular backflow and intrarenal extravasation of contrast medium. The ischemic damage of the medulla papillae communicates the vascular and collecting renal systems, causing venous leakage of contrast medium into veins as well as the crises of intermittent hematuria in the opposite direction.

Pyelocalyctaisis may be gross on RGP **(Figure 7)** while US, IVU, CAT and MRI scans appear normal. The figure also demonstrates similar damage involving the contra-lateral left kidney that was not mobile at all. As all supine standard and ancillary imaging appeared normal, the painful hematuria of SN fits the known definition criteria of LPHS. The demonstrable gross bilateral renal damage on RGP **(Figures 6 & 7)** is new evidence explaining the cause of painful hematuria of LPHS complicating SN though it has been long known as Dietl's crisis.

The complication of SN into LPHS became progressive, serious and most illusive over the years. The involvement of the normally situated or mildly ptosed left kidney with pain and pyelocalyctaisis, identical to that of the right ptosed kidney **(Figures 6 & 7),** initially defied clinical explanation. Long-term follow-up observations affirmed this phenomenon and suggested that "sympathetic nephroplegia", akin to the well-known condition that affects the eye and testes, may explain the contra-lateral pathology.

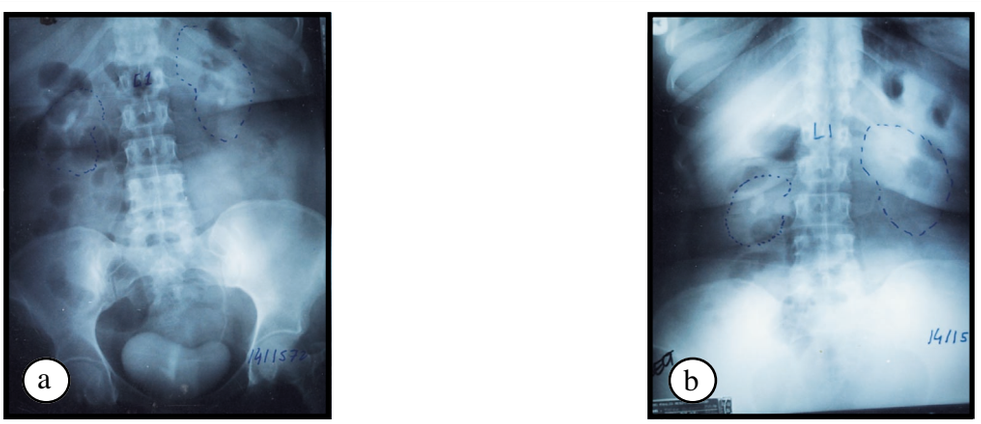

Figure 10 - Intravenous urography at **(a)** supine and **(b)** erect postures showing renal atrophy of a mobile right kidney affecting a male patient who suffered from recurrent painful hematuria episodes for over 2 decades.

Repeated episodes of painful hematuria required frequent hospital admissions, opiate therapy and prolonged bed rest for patients suffering from known SN complicated with hematuria of LPHS. One 3rd of 36 LPHS complicating SN cases, with demonstrable right mobile kidney on previous IVU, have their right kidneys spontaneously fixed either at its normal, midway or ptosed position **(Figures 2, 6 & 7).** The right kidney has undergone spontaneous fixation "auto-nephropexy" during the course of illness. "Auto-nephropexy" erased the evidence on SN but has neither cured renal pain nor hematuria. Despite the spontaneous fixation of ptosis, organic neuro-ischemic renal medulla papillae and nephron damage **(Figures 6 & 7)** became irreversible cause of painful hematuria of LPHS. Other ischemic complications of SN included acute upper pole segmental infarction demonstrated on conventional arteriography of a patient presenting with life threatening painful hematuria **(Figure 8).** Renal atrophy "auto-nephrectomy" was detected in SN patients who remain on conservative management **(Figures 9 & 10).**

Discussion. The reported photographs demonstrate heterogeneous organic causes of SN pain in which ureteral kink obstruction and stretch or torsion ischemia on upright IVU **(Figures 1-4)** and split function on upright IR **(Figure 5)** are reversible and variably contribute. Retrograde pyelography demonstrates the organic pyelocalyctaisis as the patho-etiology of painful hematuria **(Figures 6 & 7),** caused initially by intermittent renal ischemia of pedicle ptotic stretch or torsion.[9-11] This affirms that SN may complicate into LPHS. Definition of LPHS is fulfilled as standard imaging still lacks a demonstrable pathology,[2-3] when supine. Other ischemic renal complications included renal infarction **(Figure 8),** nephron loss and atrophy **(Figures 9 & 10).** Renal atrophy of ptosed kidneys occurred insidiously over a few years, affirmed by serial imaging.

The reported patho-etiology of SN pain is heterogeneous with obstruction, ischemia and neuropathy components that have reversible and organic stages. Although, these causes, and rarely UTI, may contribute to the establishment of pyelocalyctaisis,[12,13] the lesion seems primarily ischemic.[9-11] The link of SN with LPHS was illusive and overlooked, particularly when the ischemic complications of auto-nephropexy, auto-nephrectomy and sympathetic nephroplegia involving the contra-lateral kidney occurred insidiously over many years. Auto-nephropexy, made the link of SN with LPHS most illusive by erasing the evidence on renal mobility, but demonstrated that surgical[13-15] or spontaneous nephropexy alone may neither cure the loin pain of SN nor abort its complication into LPHS. The demonstrable pathology on upright IVU,[1,2,8-10,12-15] arteriography[9,10] and IR9,[2,9,11] are well documented on SN that was discarded long ago.[2] Upright imaging is currently undone and has not been reported previously in LPHS.[3-6] Retrograde pyelography findings **(Figures 6 & 7)** have not previously been documented in either condition. The use of IVU started early in the 20th century while clinical evidence on the genuineness of SN pain dated back to the 15th century.[1,12-15] Loin pain hematuria syndrome was reported in 1967[3] while Dietl's crisis is known for centuries. Organic reno-vascular complications demonstrated on conventional arteriography of SN[9,10] and LPHS[3-6] are of advanced cases. The demonstrable link of SN with LPHS, other ischemic complications of infarction and atrophy "auto-nephrectomy" and the most illusive "auto-nephropexy" and "sympathetic nephroplegia" are reported here.

This report does not attempt to resolve all the complex management problems of SN[2] and LPHS.[3-6] It only aimed to report demonstrable patho-etiology of renal pain and hematuria that affirm genuineness of loin pain, its renal origin and the link of SN with LPHS. Being based on hospital studies of SN patients, it cannot answer questions on prevalence or incidence of SN and LPHS neither in Najran nor make a comparison with it elsewhere. The presented features and complications of SN explain the bizarre heterogeneous renal pain, hematuria and MASS. These were illusive and overlooked due to the same management problems that caused disparagement of SN.[2,7]

The problems of SN are chronic with multiple and complex ramifications. Disputes of historical interest have led to many rises and falls[12] until SN was disparaged and nephropexy was abandoned decades ago.[2,7,12,13] Discarding SN from current textbooks has made it a forgotten and overlooked diagnosis. Upright imaging is not routinely carried out and chance diagnosis of SN and its link with LPHS is impossible to detect on supine imaging.[7,8] The bizarre MASS may present SN patient to many specialist clinics where repeated multiple investigations prove entirely normal. Hence, this report concerns not only urologists but also physicians and surgeons managing these cases. The symptoms are explained when the anatomy of blood and sympathetic nerve supply of the kidneys is considered.[16] The presented imaging evidence resolves the main problem of SN and LPHS concerning the lack of a demonstrable patho-etiology on supine imaging. Other renal SN features and complications[9-15] as well as management problems of SN[2,7,12] and LPHS[3-6,8] are documented but require objective evaluation and resolution. Symptomatic nephroptosis was discarded when pain was thought imaginary and the disease was thought an invention of knife happy urologists.[1,13-15] The reported evidence is reproducible when upright imaging and RGP are

considered in the management of loin pain with and without hematuria, and calls for reconsideration. Organic causes of SN and LPHS pain at its early intermittent stage are demonstrated here on upright imaging. Reno-vascular complications of SN shown on arteriography of advanced SN[9] and LPHS[3] cases appear much earlier on RGP. However, conventional arteriography is considered obsolete in the current era of MRA and spiral CT scans that are possible only at supine postures. The reported features and complications of SN were thus illusive and overlooked due to the obscure role of "Gravity and Time" detectable only on upright imaging, RGP and long term follow up observations. The intermittent ischemia of ptosis[9,10,15] insidiously progressed into chronic reno-vascular damage, in which the role of sympathetic neuropathy is important but requires clarification. The sympathetic reflex of acute splanchnic pain causing nausia, vomiting, constipation and anorexia is well known. It has made SN pain sound bizarre, unreal and hard to believe. It also confused loin pain presentation with acute abdomen, causing many unnecessary surgeries.[2,7] Chronic MASS of SN included gastro-intestinal symptoms of acid peptic disease and irritable bowel syndrome or constipation, backache with sciatica-like pain and cystitis.[7] These symptoms were documented in SN[1,14,15] and may represent features of sympathetic over-activity or neuropathy that add to the dilemma of diagnosis. Sympathetic neuropathy is known to play an important role in the pathogenesis of LPHS,[6] which is identified[8] and affirmed here to complicate SN. Surgeons who operated early on SN addressing its intermittent heterogeneous patho-etiology before the onset of organic pyelocalyctaisis reported results of >90% pain cure.[1,14,15] When surgery was indicated on the basis of demonstrable pyelocalyctaisis on IVU, however, nephropexy cured renal pain in <50% of patients.[13] This demonstrates that accurate assessment of the patho-etiology of renal pain and timely interference are mandatory for successful surgical management. The only chance of cure is dependent upon timely dealing with the anomalies at the reversible stage of SN or early LPHS. Addressing sympathetic neuropathy seems important for achieving a cure.

Simple nephropexy, reported by Hahn,[17] may cure the pain of early intermittent obstruction and pedicle stretch and twist schemia,[1,9,11,14,15] but does not abort sympathetic over-activity and neuropathy or reverse the organic damage of chronic disease. It remains unknown when neuropathy sets in. In 1989, Blacklock reported renal sympathetic denervation for the treatment of LPHS.[18] Unfortunately, neither sympathectomy nor renal auto-transplantation cured LPHS.[3-6] This may be due to the fact that surgery was delayed until irreversible renal damage, shown in **(Figures 6 and 7)** had occurred. Sympathetic neuropathy is particularly important for explaining the ischemic complication of SN into LPHS affecting the contra-lateral kidney. Loin pain hematuria syndrome is known to affect both kidneys.[3-6] It is demonstrated here that LPHS may affect both the ptosed right kidney as well as the contra-lateral normally situated left kidney. "Sympathetic nephroplegia" is neuropathic damage of a contra-lateral normal organ with its twin organ pathology, akin to the known condition that affects the eye and testes, offers the only scientific explanation for the involvement of a normal kidney with LPHS. Many patients reported left loin pain contra-lateral to the right ptosed kidney, well before organic renal complications affected either kidney. The bizarre pains of SN and its MASS become easier to understand when sympathetic patho-physiology is considered.

Sympathetic nerves synapse at the celiac plexus[16] and over-stimulation may explain the cross-referred renal pain and MASS, in addition to the fact that some patients also have visceroptosis.[1,14] The problem of which kidney is the original site of painful hematuria is resolved by cystoscopy and RGP, but to determine which kidney is the origin of pain, and when sympathectomy should be carried out with nephropexy, is extremely difficult. Forthcoming reports will objectively address such management problems of SN pain and painful hematuria of LPHS with data and statistical analysis.

The patients' main problem was being disbelieved and falsely labeled,[4,7,8] mainly due to the lack of demonstrable pathology on the invariably normal supine imaging. Lack of objective tests to affirm pain genuineness, renal origin and severity has also compounded the problems of diagnosis.[2,7] The bizarre MASS made SN pain sounds unreal. Patients look absolutely normal after an agonizing episode of functional "renal angina" pain is settled.[7] Some may use hospital time to make up for their lost social life outside, leaving an impression of malingering but this does not explain the hematuria.

Doctors' perception of malingering, opiate dependency and psychological pain[4] has lead to labeling with these disorders that may occur as iatrogenic complications later perhaps as a result of prolonged suffering from the undiagnosed and untreated disorders.[7] Believing that these patients suffered from genuine pain implanted a belief that initiated this research to find and demonstrate a patho-etiology of loin pain that is evidenced by the presented photographs. The importance of evidence based on imaging photographs is to overcome medical and patients problems of disbelief and false labeling. Increased awareness may help such unfortunate patients both in the Kingdom of Saudi Arabia and elsewhere to be taken seriously, properly investigated and appropriately treated. This may

prevent adding the insult of false labeling to injury of the incapacitating pain, allowing adequate analgesia until an effective cure for the diseases is found.

Awareness of the precise patho-etiology of renal pain at its reversible and organic stages may moderate expectations of colleagues, patients and relatives of a successful therapy that may cure pain and hematuria but cannot reverse an established pyelocalyctaisis or renal atrophy. For predicting precise prognosis, objective evaluation before therapy must segregate intermittent features from organic irreversible complications of SN. In order to achieve the highest possible chance of cure, therapy must address the mixed patho-etiology of pain at an early stage of SN and LPHS. This has important implications on taking the consent, particularly for explaining any post-therapy residual symptoms or possible renal atrophy. Renal atrophy was reported in 2 of 10 patients after intraureteric capsaicin injection[4] and the loss of 75% kidneys of LPHS patients after surgery[6] may in fact be complications of advanced renal disease rather than surgery as renal atrophy may occur spontaneously in patients who remain on conservative therapy **(Figures 9 & 10).** Upright imaging and RGP may resurrect management issues of SN and LPHS by the demonstrable patho-etiology. Further research work is required and a Saudi National Survey is recommended.

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