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CKJ Review

CKJ REVIEW

Loin pain haematuria syndrome 1967–2020: a review

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ABSTRACT

The purpose of this retrospective review is to question the validity of the condition 'loin pain haematuria syndrome' (LPHS). We highlight the possibility that most patients regarded as having LPHS have a psychiatric/psychological basis for their symptoms, particularly loin pain. Because of this, and because it recurs despite treatment, the review also questions the use of treatments that are invasive, expensive, and carry considerable morbidity.

Keywords: haematuria, loin pain, psychiatric features, somatisation disorder

Peer-reviewed publications (PubMed) were accessed using the terms 'loin pain haematuria' and 'loin pain haematuria syndrome'. A total of 613 patients diagnosed as LPHS were identified in 75 papers published between April 1967 and March 2017 (Group A). Nine papers published between April 2017 and December 2020 produced 125 patients (Group B). The demographics of all 738 patients in the two groups are described where available. Seven papers (May 2021–March 2023) are not included in this review as they do not affect our conclusions.

In Group A, haematuria was reported in only 274 of the 613 patients. Various forms of imaging, most importantly renal arteriograms, were normal. Histologically, assessment of 331 renal biopsies were normal or showed minor changes. Various treatments for pain relief, ranging from analgesics to kidney auto-transplantation (KAT) showed mixed results. Thirty-one patients had a psychiatric history which was also suspected in another 205 of the 613 patients.

The nine papers in Group B focused particularly on treatment for pain, KAT being the favoured option, rather than investigation of the aetiology of LPHS. The basis for diagnosis was often unclear and, in many cases, a psychiatric factor was not excluded.

This review throws doubt on the existence of LPHS as a specific physical condition. Scrutiny of the literature supports the view that LPHS is a manifestation of a psychiatric or psychological condition.

INTRODUCTION

LPHS has been a feature of nephrological practice since the original report by Little *et al.* [1], who described three women with loin pain and haematuria. Renal histology showed interstitial fibrosis in two and sub-capsular fibrosis in the third. Renal arteriography was described as showing tortuosity of vessels with narrow irregular lumina. Later, more sophisticated imaging failed to confirm these changes. Haematuria, although originally an essential component of LPHS, has been an inconsistent feature, and recently the syndrome has been described as loin pain 'with or without haematuria' [2]. This review assesses the validity of the belief that LPHS is a physical conditiion and explores the evidence for a psychiatric/psychological basis.

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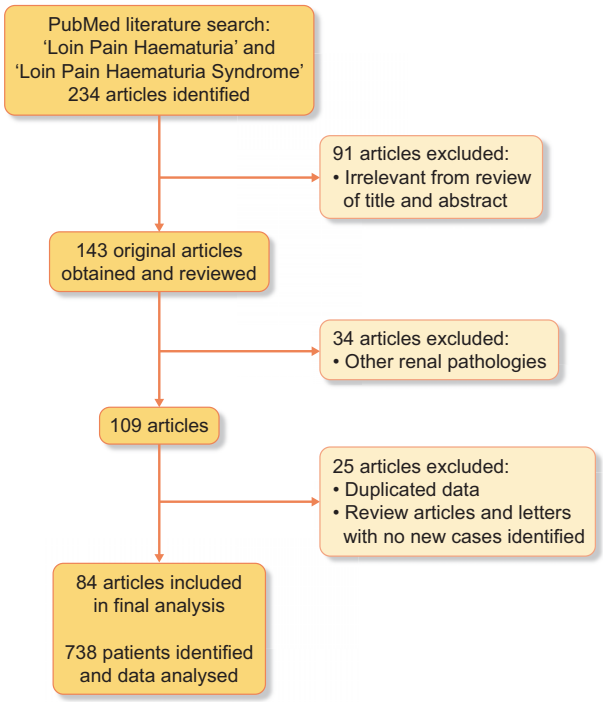

Figure 1: Selection of peer-reviewed papers: March 1967–2021.

Table 1: Profile of the 84 papers assessed in detail (Groups A + B)

Type of paper	Number of papers
Single case reports	27
Case series: 2–10 patients	36
Case series: 11–20 patients	10
Case series: 21–30 patients	7
Case series: 31–40 patients	1
Case series: 41–50 patients	1
Case series: >51 patients	2
Total	84

ANALYSIS OF PUBLICATIONS 1967–2020

The PubMed search produced 234 publications; 150 papers were excluded for reasons given in Fig. 1. The remaining 84 publications comprising 738 patients were analysed (Table 1). Papers were mostly from four countries: UK 34 (367 cases), USA 23 (201), Australia 7 (87), Canada 6 (59); 718 patients (97%) were from English-speaking countries.

The 84 publications (Tables 1 and 2) were split into two groups. Earlier publications encompassed 1967–2017 (Group A) and later publications 2017–2020 (Group B). The rationale for the division was that by 2017 the content of publications had changed from a predominantly nephrological search for a cause for the symptoms to surgical treatments to relieve pain. The latter papers were mostly written by surgeons, anaesthetists, and radiologists and not published in nephrological journals. Unlike many publications in Group A, psychiatrists were not involved and there was an emphasis on relieving pain.

CHARACTERISTICS OF 613 PATIENTS 1967–2017 (GROUP A)

Clinical aspects

Demographic status

There were 126 men and 404 women (83 gender unknown). The age-range (known in 243) was 6 to 80 years (mean 29.9, median 31.0); in 370 patients age was not reported. Racial status was given in a minority: Caucasians 61, African Americans 2, and White Americans 1. Of 92 patients with a reported occupation, 46 were in an occupation related to medicine. Relatives' occupations were reported in 20 cases of whom 16 had relatives whose employment was medically related.

Haematuria, proteinuria, urine microscopy

Macroscopic haematuria was reported in 162 patients. If red cells in urinary sediment and dipstick-positive haematuria are accepted as adequate evidence, 112 patients had microscopic haematuria; 279 patients were recorded simply as 'haematuria'. Of the 613 cases, 44 were reported to have proteinuria. Haematuria and proteinuria unreported in 10% and 75% of cases, respectively.

Blood pressure, kidney function

Blood pressure, measured in 223 patients, was raised in 20. Kidney function was normal in 260 as judged by an eGFR ≥60 ml/min or serum creatinine (SCr) ≤1.2 mg/100 mL (106 μmol/L), or a statement that kidney function or SCr was normal. Eight patients had mildly reduced renal function: 1.24–1.47 mg/100 mL (110–130 μmol/L). Kidney function was not recorded in 345 (56.3%) of patients.

Urolithiasis

Among the 75 papers, 16 reported 161 patients with a history of renal stones. Fifty-nine had a past history or stones present at the time of LPHS diagnosis. Among these 59, in only 22 had stones been detected. In 18 the diagnosis was not corroborated; in 16 the history was probably correct but stones were not found when LPHS was investigated. All patients were diagnosed as LPHS and are included here, though recognising that LPHS was not necessarily an appropriate diagnosis.

Imaging, cystoscopy

Intravenous urography

Of 613 patients, 379 underwent IVU. All were normal.

Renal arteriography

A total of 131 patients underwent this procedure; 102 were reported to be abnormal. Most of the studies were performed between 1967 and 1997; the abnormalities described were similar to those reported by Little *et al.* [1]. With improved imaging, small vessels were better defined and the earlier-described abnormalities were not confirmed. In 482 patients, renal arteriography was either not done or not reported.

Table 2: Details of the 84 peer-reviewed references assessed (Groups A and B) and miscellaneous publications

	LPHS Group A–75 papers						
	Authors	Title	Cases	Journal	Year	Vol	pp
1	Little PJ, Sloper JC and de Wardener HE	A syndrome of loin pain and haematuria associated with disease of peripheral renal arteries.	3	Q J Med	1967	36	253–9
2	Burden RP, Booth LJ *et al.*	Intrarenal vascular changes in adult patients with haematuria and loin pain—a clinical, histological and angiographic study.	12	Q J Med	1975	44	433–47
3	Michael J, Jones NF *et al.*	Recurrent haematuria: role of renal biopsy and investigative morbidity.	13	Br Med J	1976	1	686–8
4	Guyer PB	Radiology of the loin pain haematuria syndrome.	14	Clin Radiol	1977	52	33–42
5	Fletcher P, Al-Khader AA *et al.*	The pathology of intrarenal vascular lesions associated with the loin pain haematuria syndrome.	3	Nephron	1979	24	150–4
6	Burden RP, Dathan JR *et al.*	The loin pain/haematuria syndrome.	9	Lancet	1979	1	897–900
7	Sherwood T	Loin pain/haematuria syndrome.	2	Lancet	1979	1	1033–4
8	Parbtani A, Cameron JS	Platelet involvement in loin pain/haematuria syndrome.	9	Lancet	1979	1	1413
9	Ratajczak T, Steciwko A *et al.*	Recurrent loin pain—hematuria syndrome.	1	Wiad Lek	1980	33	1913–5
10	Habte W, Dobbie JW *et al.*	The loin-pain haematuria syndrome in males.	3	Scott Med J	1981	26	118–20
11	Adiga KM	Loin-pain-haematuria syndrome.	1	J Ind Med Ass	1982	78	151–3
12	Nicholls AJ, Muirhead N *et al.*	Loin pain and haematuria in young women: diagnostic pitfalls.	2	Br J Urol	1982	54	209–11
13	Aber G and Higgins PM	The natural history and management of the loin pain/haematuria syndrome.	51	Br J Urol	1982	54	613–5
14	Bell GM, Williams P *et al.*	Is the loin pain and haematuria syndrome a manifestation of hypersensitivity?	10	Lancet	1984	1	340
15	Sheil AG, Ibels LS *et al.*	Renal autotransplantation for severe loin pain/haematuria syndrome.	3	Lancet	1987	2	1216–7
16	Reifsteck JE, Holder JC *et al.*	Loin pain haematuria syndrome.	1	Urol Radiol	1987	1	155–7
17	Hutchison SM, Doig A *et al.*	Recurrence of loin pain/haematuria syndrome after renal autotransplantation.	1	Lancet	1987	1	1501–2
18	Bergroth V, Konttinen YT *et al.*	Loin pain and haematuria syndrome: possible association with intrarema arterial spasms.	4	Brit Med J	1987	294	1657
19	Sheil AG, Ibels LS *et al.*	Treatment of loin pain/haematuria syndrome by renal autotransplantation.	9	Lancet	1987	2	907–8
20	Smellie SW, Lambert M *et al.*	Factor XII deficiency associated with loin pain/haematuria syndrome.	1	Lancet	1987	2	1330
21	Siegler RL, Brewer ED *et al.*	Platelet activation and prostacyclin supporting capacity in the loin pain hematuria syndrome	1	Am J Kid Dis	1988	12	156–60
22	Hutchison SM, Harkiss G	In-vitro study of immune responses in loin pain/haematuria syndrome.	7	Lancet	1989	1	1451
23	Bloom PB, Viner ED *et al.*	Treatment of loin pain haematuria syndrome by autotransplantation.	1	Am J Med	1989	87	228–32
24	Blacklock AR	Renal denervation with releasing capsule incision in the loin pain/haematuria syndrome.	1	Br Med J	1989	64	202–4
25	Leaker BR, Gordge MP *et al.*	Haemostatic changes in the loin pain and haematuria syndrome: secondary to renal artery spasm?	26	QJM	1990	76	969–79

Table 2: Continued

	LPHS Group A–75 papers						
	Authors	Title	Cases	Journal	Year	Vol	pp
26	Chin JL	Loin pain-haematuria syndrome: role for autotransplantation.	10	J Urol	1992	147	987–9
27	Dimski DS, Hebert LA *et al.*	Renal autotransplantation in the loin pain-hematuria syndrome: a cautionary note.	2	Am J Kid Dis	1992	20	180–4
28	Lucas PA, Leaker BR, Neild GH	Psychiatric aspects of loin pain/haematuria syndrome.	15	Lancet	1992	340	1038
29	Kelly B	Psychological aspects of loin-pain/haematuria syndrome.	7	Lancet	1992	340	1294
30	Woolfson RG, Lewis CA *et al.*	Ureteric peristalsis studies in loin pain and haematuria syndrome: another diagnostic disappointment.	30	Brit J Urol	1993	72	291–2
31	Gibson P, Winney RJ *et al.*	Bilateral nephrectomy and haemodialysis for the treatment of severe loin pain haematuria syndrome.	1	NDT	1994	9	1640–1
32	Harney J, Rodgers E *et al.*	Loin pain-hematuria syndrome: how effective is renal autotransplantation in its treatment?	4	Urology	1994	44	493–6
33	Miller F, Lane BP *et al.*	Loin pain-hematuria syndrome with a distinctive vascular lesion and alternative pathway complement activation.	1	Arch Path Lb M	1994	118	1016–9
34	Talic RF, Parr N, Hargreave TB	Anephric post graft nephrectomy in patient treated with renal autotransplantation for bilat metachronous LPHS.	1	J Urol	1994	152	1194–5
35	Prager JP, DeSalles A *et al.*	Loin pain haematuria syndrome: pain relief with intrathecal morphine.	1	Am J Kid Dis	1995	25	629–31
36	Bultitude MI	Capsaicin in treatment of loin pain/haematuria syndrome.	4	Lancet	1995	345	921–2
37	Lucas PA, Leaker BR *et al.*	Loin pain and haematuria syndrome: a somatoform disorder.	15	QJM	1995	88	703–9
38	Burke JR, Hardie IR	Loin pain haematuria syndrome.	2	Paed Nephrol	1996	10	216–20
39	Karvelas JP, Ramsey EW	Renal autotransplantation in patients with loin pain hematuria syndrome.	4	Can J Surg	1996	39	121–5
40	Parnham AP, Low A *et al.*	Recurrent graft pain following renal autotransplantation for loin pain hematuria syndrome.	11	Br J Urol	1996	78	25–8
41	Spitz A, Huffman JL *et al.*	Autotranplantation as an effective therapy for LPHS: case reports and a review of the literature.	2	J Urol	1997	157	1554–9
42	Lall R, Mailis A, Rapoport A	Hematuria-loin pain syndrome: its existence as a discrete clinico-pathological entity not supported.	7	Clin J Pain	1997	13	171–7
43	Andrews BT, Jones NF *et al.*	The use of surgical sympathectomy in the treatment of chronic renal pain.	18	Br J Urol	1997	80	6–10
44	Bultitude M, Young J *et al.*	Loin pain haematuria syndrome: distress resolved by pain relief.	26	Pain	1998	76	209–13
45	Sheil AG, Chui AK *et al.*	Evaluation of LPHS treated by renal autotranplantation or radical renal nephrectomy.	46	Can J Surg	1998	32	215–20
46	Chin JL, Kloth D *et al.*	Renal auto-transplantation for the loin pain-hematuria syndrome: long-term follow-up of 26 cases.	22	J Urol	1998	160	1232–6
47	Gusmano RG	Could this be loin pain hematuria syndrome?	1	Pediatr Nephrol	1998	12	800

Table 2: Continued

	LPHS Group A–75 papers						
	Authors	Title	Cases	Journal	Year	Vol	pp
48	Blacklock AR, Raabe AL *et al.*	Renal auto-transplantation with interposed PTFE arterial graft: not necesarily a cure for LPHS.	1	JR Coll Surg Ed	1999	44	134
49	Francis DM	Renal auto-transplantation with interposed PTFE arterial graft: not necesarily a cure for LPHS.	5	JR Coll Surg Ed	1999	44	412
50	Armstrong T, McLean AD *et al.*	Early experience of intra-ureteric capsaicin infusion in loin pain haematuria syndrome.	10	BJU Int	2000	85	233–7
51	Gill IS, Uzzo RG *et al.*	Laparoscopic retroperitoneal live donor right nephrectomy for allotransplantation and autotransplantation.	2	J Urol	2000	164	1500–4
52	Haddad E, Lapeyraque AL *et al.*	Clinical quiz. Loin pain haematuria syndrome.	1	Pediatr Nephrol	2002	17	217–9
53	Playford D, Kulkarni H *et al.*	Intra-ureteric capsaicin in loin pain haematuria syndrome: efficacy and complications.	4	BJU Internat	2002	90	518–21
54	Pukenas BA, Zaslau S	Loin pain hematuria syndrome: case series.	3	WV Med J	2003	99	192–3
55	Greenwell TJ, Peters JL *et al.*	The outcome of renal denervation for managing loin pain haematuria syndrome.	24	BJU Internat	2004	93	818–21
56	Spetie DN, Nadasdy G *et al.*	Proposed pathogenesis of idiopathic loin pain-haematuria syndrome.	34	Am J Kid Dis	2006	47	411–27
57	Diwakar R, Andrews PA	Renal transplantation in a patient with loin pain hematuria syndrome.	1	Clin Nephrol	2006	66	144–6
58	Dube GK, Hamilton SE *et al.*	Loin pain hematuria syndrome.	1	Kid Internat	2006	70	2152–5
59	Bass CM, Parrott H, Jack T *et al.*	Severe unexplained loin pain [loin pain haematuria syndrome]: management and long-term outcome.	21	QJM	2007	100	369–81
60	Uzoh CC, Kumar V *et al.*	The use of capsaicin in loin pain-haematuria syndrome.	3	BJU Int	2009	103	236–9
61	Canales BK, Windsperger A *et al.*	Endoscopic findings in loin pain hematuria syndrome: concentric clot in calyceal fornices.	2	Diag Ther Endo	2008	Epub	721 850
62	Goroszeniuk T, Khan R *et al.*	Lumbar sympathetic chain neuromodulation with implanted electrodes for long-term pain relief in LPHS.	4	Neuromodul....	2009	12	284–91
63	Ahmed M, Acher P, Deane AM	Ureteric bupivicaine infusion for loin pain haematuria syndrome.	17	Ann RCS Engl	2010	92	139–41
64	Elkins JR, Koep LL *et al.*	Hypnotherapy intervention for loin pain hematuria: a case study.	1	Int J Clin Exp H	2012	60	111–20
65	Almaiman H, Serre JE *et al.*	A mini-invasive approach to renal autotransplantation in the management of loin pain hematuria syndrome.	4	Prog Urol	2013	23	389–93
66	Gambaro G, Fulignati P *et al.*	Percutaneous renal sympathetic nerve ablation for loin pain haematuria syndrome.	1	NDT	2013	28	2393–5
67	Kadi N, Mains E *et al.*	Transperitoneal laparoscopic renal denervation for the management of LPHS.	9	[a]	2013	22	346–51
68	Moeschler SM, Hoelzer BC *et al.*	A patient with LPHS and chronic flank pain treated with pulsed radiofrequency of the splanchnic nerves.	1	Clin J Pain	2013	29	26–9
69	Taba Taba Vakili S *et al.*	Loin pain hematuria syndrome.	1	Am J Kid Dis	2014	64	460–72
70	Dekker TJ *et al.*	Loin pain haematuria syndrome.	1	Ned Tidj Gen	2014	159	A8101
71	Russell A, Chatterjee S, Seed M	Does this case hold the answer to one of the worst types of pain in medicine—that of LPHS.	1	BMJ Case Rep	2015	26	2015
72	Rohrer F, Déglise S *et al.*	Bilateral renal autotransplantation may be an effective and definitive treatment in case of LPHS.	1	Urol Int	2017	99	118–20

Table 2: Continued

	LPHS Group A–75 papers						
	Authors	Title	Cases	Journal	Year	Vol	pp
73	Cowan NG, Banerji JS *et al.*	Renal autotransplantation: 27 year experience at two institutions.	17	J Urol	2015	194	1357–61
74	Zubair AS *et al.*	Clinical and safety outcomes of laparoscopic nephrectomy with renal autotransplantation for LPHS.	21	J Ev Based Med	2016	9	84–90
75	Prasad B, Giebel S *et al.*	Renal denervation in patients with loin pain hematuria syndrome.	4	Am J Kid Dis	2017[b]	69	156–9
			613				

	LPHS Group B–9 papers						
	Authors	**Title**	**Cases**	**Journal**	**Year**	**Vol**	**pp**
76	Zuidema X *et al.*	Dorsal root ganglion stimulation: a treatment option for chronic pain due to refractoty LPHS.	1	Neuromodulation	2017	20	841–3
77	de Jager RL, Casteleijn NF *et al.*	Catheter-based renal denervation as therapy for severe chronic kidney-related pain.	6	Nephrol Dial Tran	2018	33	614–9
78	Prasad B, Giebel S, Garcia F *et al.*	Successful use of denervation in patients with Loin Pain Haematuria Syndrome—the Regina LPHS study.	8	Kid Internat Rep	2018	3	638–44
79	Decastaecker K, Van Parys B *et al.*	Robot-associated kidney auto-transplantation: a minimally invasive way to salvage kidneys.	1	Eur Urol Focus	2018	4	198–205
80	Sollinger HW, Al-Qaoud T *et al.*	The 'HW-LPHS test': a new test to predict the outcome of Renal Autotransplantation for LPHS.	6	Exp Clin Transp	2018	16	651–5
81	Richter B, Bergman J *et al.*	Spinal cord stimulation for Loin Pain Hematuria Syndrome: clinical report.	1	Pain Pract	2019	19	440–442
82	Dale L-A, Silva MA *et al.*	Therapeutic living donor nephrectomy.	2	Clin Transplant	2019	33	e135
83	Babazade R, Devajaran J *et al.*	Management and 30 day outcome after Renal Autotransplantation.	16	Ochsner J	2020	131	822–9
84	Campsen J, Pan G *et al.*	Renal autotransplantation for Loin Pain Haematuria Syndrome using a multidisciplinary team model: intermediate term results.	84	Cureus	2020	12	e12379
			125				

Miscellaneous publications cited; either not reviewed or without patients.

	Author	**Year**	**Reference**
1	Meadow R	1982	Munchausen syndrome by proxy. Arch Dis Child. 1982; **57**: 92–98.
2	de Wardener HE	1985	'The Kidney' 5th ed. Longman Group. pp 468–469.
3	Kelly B	1994	Psychological aspects of the loin pain/haematuria. Lancet. 1992; **340**: 1294.
4	Coffman KL	2009	Loin pain haematuria syndrome: a psychological and surgical conundrum. Curr Opin Organ Transplant. 2009; **14**: 186–190.
5	Internat. Classification of Disease	2016	International Classification of Disease version 10 [2016]. The ICD-10 classification of mental and behavioural disorders 2010, WHO.
6	Lugo-Gavidia LM, Nolde SM, Kiuchi MG *et al.*	2020	Interventional approaches for loin pain haematuria syndrome and kidney-related pain syndromes. *Curr Hypertens Rep*. 2020; **22**:103
7	Prasad B, Sharma A, Lanktree MB *et al.*	2023	Kidney Biopsy and Type IV Collagen Gene Sequencing Fail to Explain Hematuria in LPHS. *Kidney Int Rep*. 2023; **8**: 1013–1021.

[a]**Reference 67:** Minim Invasive Ther Allied Technol.[journal]:
[b]**Reference 75:** Previously 2016 on PubMed

Ultrasound

In total, 101 patients had normal scans. The remaining 512 were not scanned. The earliest mention of ultrasound in relation to LPHS was in 1987 [3].

Isotope renography

Of 70 studies, all but seven were normal.

Renal CT scan

Of 68 scans, 66 were normal. One showed adhesions between the kidney and abdominal wall, and another a resolving renal vein thrombosis.

Cystoscopy, retrograde pyelography

A total of 228 of 235 patients had normal cystoscopies: four had ureteric bleeding, two had abnormal mucosa and one had both. In 50 patients retrograde pyelography was normal.

Renal histology

The results of 240 of the 331 biopsies were available.

Light microscopy

Ninety-five specimens (39%) were normal; 145 abnormal. Abnormalities included vascular and/or fibrotic changes, increased mesangial matrix, and mesangial cell proliferation. One case showed membranous glomerulonephritis. There were no consistent findings.

Immunofluorescence

The commonest findings were C_3 deposition in the mesangium or vessels (55 cases). Six cases showed IgA nephropathy and another IgM nephropathy. No consistent diagnostic features emerge.

Electron microscopy

Thin basement membrane disease (TBMD) was present in two cases. Spetie *et al.* [4] undertook 34 biopsies: nine showed 'unusually thin GBM' and 11 'unusually thick GBM'. The authors postulated that haematuria in LPHS is glomerular in origin and the pain due to tubular obstruction causing renal parenchymal swelling and stretching of the renal capsule. However, they questioned whether the extent of blood seen in tubules resulted from the biopsy procedure itself. They concluded; 'other factors are needed to explain the severe pain in patients with LPHS'.

In a recent biopsy study of 14 patients, Prasad *et al.* [5] found that the GBM was normal in 12 and thicker than normal in one (one biopsy inadequate). This paper is not included in our review.

Therapeutic interventions and outcomes

Many interventions were used: analgesic combinations usually containing opioids, nerve block, denervation, KAT, and nephrectomy (Table 3). Patients frequently had multiple interventions but, in many, the type and/or the outcome were unclear or not reported.

Analgesics

Of 613 patients, 289 (47.1%) were treated with analgesics; in the remaining 324 analgesic data were missing. The commonest analgesics were opioids used singly or with other drugs amounting to 274 patients (44.7% of the 613). Among these, 18 were considered to have overused their drug.

Table 3: Efficacy of treatments potentially curative of pain in LPHS patients (Group A)

Treatment	Success	Failure	Unknown, partial, or short-lived	Total patients
Anticoagulants	0	9	5	14
Denervation	26	28	49	103
Stent	0	6	6	12
Nephrectomy	9	14	19	42
Auto-transplant	74	40	54	168
TOTAL [%]	**109 [32.2]**	**97 [28.6]**	**133 [39.2]**	**339**

Anticoagulants, anti-platelet drugs

Five patients, described by Burden *et al.* [6], received warfarin and had short-term pain-relief; another five received both drugs or just anti-platelet agents. In four the drugs used were not specified. In the latter nine patients there was no consistent evidence of benefit. Leaker *et al.* [7] reported 25 patients, some of whom were given anti-platelet agents without success.

Capsaicin

In 61 of 75 patients treated, capsaicin was instilled into the ureter on the side of the loin pain; the site was not stated in 14. In 44 patients with adequately reported results, 17 were successful, 15 failed and in 12 pain control was partial or short-lived. Coffman [8] undertook a meta-analysis of LPHS patients treated with intra-ureteric Capsaicin. Pain-relief was achieved in 57.5% of patients but its duration was unclear and side effects often intolerable.

Local anaesthetics

Various anaesthetics injected at different sites were used in 29 (4.7%) patients, some being treated more than once. Only four patients had successful outcomes, 12 had partial/temporary improvement in pain and six had no response. In seven the outcome was not clear.

Denervation

This was carried out in 103 (16.8%) patients. Discernible outcomes in 67 were: successful 26; failed 28; partial or short-lived 13. Outcomes were not clear in 36 patients. Coffman's meta-analysis [8] found that pain was relieved in 25.4% of cases but recurred in 75% of these patients.

Nerve block

Various nerve-blocking manoeuvres were attempted, some on multiple occasions. Nerve block was used in 91 (14.8%) patients. The effects on pain relief were disappointing or uninterpretable.

Ureteric stenting

This was performed in 12 patients. Six had no pain relief, one had partial relief and in five the outcomes were unclear.

Primary nephrectomy

Forty-two (6.8%) patients underwent nephrectomy, one being partial. Only nine (21%) are recorded as having complete pain

Table 4: Results of auto-transplantation (Group B)[a]

Follow-up [months]	Success [no pain]	Partial/temporary pain relief	Failure	Total
1	1	12	4	17
3–4	2	0	0	2
6	0	0	1	1
12	16	16	0	32
TOTAL	19	28	5	52

[a]Eighteen patients, not included here, were operated on but the data were either incomplete or the patient lost to follow-up.

relief; in 14 (33%) pain recurred on the other side. In 19 (45%) it is unclear whether pain relief was achieved or not.

Kidney auto-transplantation

As summarised in Table 4, 168 patients underwent single KAT with pain relief in 74. In 40 of the 168, pain recurred on the same or contralateral side. The outcome with regard to pain in the remaining 54 is uncertain. After KAT, 28 of the 168 underwent auto-transplant nephrectomy for recurrent symptoms. Another 25 (15%) had the second native kidney auto-transplanted; of 25 bilateral auto-transplantations, seven were considered a success, five a failure, but the outcome was unknown in 13. Coffman's meta-analysis [8] showed that 62.5% of patients were pain-free one year post-operatively.

Miscellaneous treatments

Thirty patients were treated by trans-cutaneous nerve stimulation and 26 received physical therapy. A wide range of other treatments were carried out in very small numbers of patients. None resulted in significant or long-term benefit.

Histology of native nephrectomies and biopsies of auto-transplants

There were 17 histological reports of native nephrectomies: normal (4), normal vessels (2), C_3 in vessel walls (3), significant scarring (3), and a variety of other non-specific abnormalities. The histology of three auto-transplant nephrectomies was normal; a fourth showed chronic interstitial nephritis and renal infarction.

Psychiatry and somatisation

In 23 of 75 papers, there was confirmation prior to LPHS diagnosis of a past psychiatric history in 31 patients and a possible such diagnosis in 25. While LPHS was being investigated or treated, a psychiatric diagnosis was suspected in 205. Psychiatric assessments were reported in 314 patients; nine had no assessment; in 290 there was no information on assessment. In 170 patients the nephrologist or psychiatrist thought the loin pain was an expression of underlying psychiatric illness.

Leaker *et al.* [7] noted that 6 of 25 LPHS patients had received psychiatric care prior to diagnosis. Three had addiction problems, one anorexia nervosa, and two were said to have hysteria. Lucas *et al.* [9] found, in 15 LPHS patients, three times the number of medically unexplained symptoms and more analgesic consumption, than a control group. In 8 of the 15, onset of pain was associated with an adverse psychological life-event in the preceding 6 months. This had not occurred in any of the controls. Lucas *et al.* [9] also noted serious parental illness/disability during the patients' childhood. The ranking of adverse life-events was assessed using the American Psychiatric Association's method (Diagnostic and Statistical Manual of Mental Disorders-Revised). Similar views were expressed by Kelly [10] and Lall *et al.* [11]. The latter paper reported that four of seven patients responded successfully after 1 year to a combination of counselling, anti-depressants, and anxiolytics. Bass *et al.* [12], assessing the psychological factors associated with LPHS in 21 patients, using the Hospital Anxiety and Depression Scale (HADS), considered that 12 had a somatoform disorder, two had dissimulation and six admitted opioid abuse. Taba Taba Vakili *et al.* [13] noted a wide range of psychological and psychiatric factors known to contribute to the development of symptoms in LPHS patients.

Not all authors acknowledge a psychiatric/psychological aetiology in LPHS. Bultitude *et al.* [14], recognising that not finding a physical cause for the syndrome suggests a psychiatric disorder, studied patients using a wide range of patient questionnaires: McGill Pain Questionnaire (MPQ), Pain Rating Index, Present Pain Intensity, General Health Questionnaire, HADS. The assessments were made pre- and post-treatment with Capsaicin. No patient had a past history of psychiatric illness and pre-treatment there were no differences between treatment and control groups. However, post-treatment the Capsaicin group showed significant improvement. The authors concluded that LPHS has an organic basis. Dube *et al.* [15] reported depression in LPHS patients but was unable to decide whether this was a pre-existing condition or a consequence of long-standing pain.

Dependency on opioids

As mentioned above, 18 patients were considered to overuse opioids. Many others were found to be opioid-dependent. Other papers comment on this aspect of patients' histories [7, 12, 13]. That some patients labelled as LPHS may have exaggerated their symptoms to obtain opioids cannot be excluded.

Patients' and relatives' occupations

The finding that patients and near relatives often had occupations related to medicine was noted by Little *et al.* [1] and other authors [6, 9, 12, 14, 16]. In Group A, 46 of 92 patients with known occupations worked in a medical field; this was also true for 16 of 20 close relatives.

Self-induction of clinical features

Little *et al.* [1] reported falsification of body temperatures by patients. Similarly, Aber and Higgins [16] reported interference with urine tests for blood (see Discussion).

LPHS in children

Of the 613 patients (Group A) reviewed, 26 were children, aged 6–16. The paper by Burke and Hardie [17] presented data in children with LPHS and specifically in two children aged 6 and 10. They comment: 'a significant number show psychological and psychopathological features', and that 'A detailed psychological history is mandatory'. Evaluation should include any psychiatric history in relatives and any history of emotional, physical, or sexual abuse in the child. Factitious haematuria should also be borne in mind. The two children reported had haematuria and loin pain but kidney biopsies showed proliferative glomerulonephritis. Both children had experienced adverse life events. They should not be regarded as having LPHS because of

the clear histological findings. An account of one child, aged 7, is in the Discussion.

CHARACTERISTICS OF 125 PATIENTS 2017–2020 (GROUP B)

Clinical aspects

Demographic status

There were 53 women and 19 men; gender was not stated in 53. The age range was 19–62. Caucasians comprised 68 of 70 patients. Occupation was recorded in only two of the 125 patients.

Haematuria, proteinuria, urine microscopy, and culture

Macroscopic haematuria was reported in 10 patients. In one paper LPHS was accepted as a diagnosis 'with or without' haematuria [2]. Proteinuria was absent in the 12 patients tested. Urine microscopy and culture was carried out in one study (12 patients); no results were provided.

Blood pressure, kidney function

Blood pressure was normal in 17 patients in whom it was measured. Kidney function (eGFR) was normal in 19 patients in whom it was measured.

Imaging, cystoscopy

Radiological

Few diagnostic radiological investigations were carried out in contrast to over 750 in Group A. Renal angiography was used not for diagnostic purposes but for pre-operative or pre-denervation assessment of vascular structures.

Cystoscopy, urine cytology

In contrast to Group A, cystoscopy was reported in only one paper (six cystoscopies) but without details. Urine cytology was mentioned in one paper without results.

Renal histology

There were no reports on renal histology.

Therapeutic interventions and outcomes

Most patients in Group B were heavy users of opioids. Some had undergone other treatments similar to those in Group A.

The nine papers in Group B comprise treatments by KAT [5], renal denervation [2], electrical neurological stimulation [2] (dorsal root ganglion 1, spinal cord 1), and nephrectomy [2]. The outcomes of these treatments were reported at various times after the procedures; in some, follow-up was as brief as one month. However, there are clear, longer-term data, especially for KAT at one year (Table 4). Overall, outcome descriptions were often imprecise.

Kidney auto-transplantation

Seventy patients were auto-transplanted. In 18 (26%), there were no follow-up data. The outcomes in the remaining 52 patients are summarised in Table 4. Nineteen had complete pain relief, sustained for varying lengths of time. In 28 the pain improved but was not eradicated after 1 year; in five patients KAT did not relieve pain.

Renal denervation

There were 14 LPHS patients treated by denervation. In one paper describing eight patients [18], seven had pain reduction of >30% at 6 months using the MPQ. Another paper [19] reported six patients who had a significant reduction in analgesic consumption at 3 months and a further fall by 12 months.

Dorsal root ganglion stimulation (DRGS), spinal cord stimulation (SCS)

In one patient DRGS resulted in abolition of pain after 1 month. At 36 months the pain was less severe compared with pre-treatment [20]. The assessment used a Numeric Rating Scale which fell from 9 to 0. Similarly, the one SCS patient reported less pain than initially after 26 months [21] as assessed by a Quality-of-Life Scale which improved from 5 to 10.

Nephrectomy

Two papers record five nephrectomies. In one series of 60 patients deemed suitable for KAT [2], three elected to undergo nephrectomy rather than KAT; the outcome was not recorded. In another paper [22] an auto-transplanted LPHS patient continued to have pain, and the graft was removed at 6 months. The patient remained pain-free at 11 years. Interestingly, the 'LPHS kidney' removed was transplanted into a non-LPHS patient; the recipient also had no pain at 11 years.

Psychiatry and somatisation

Among Group B papers, the importance of psychiatric assessment is referred to. However, there were no psychiatrists among the authors or in the clinical work-up. In one paper [19], all six LPHS patients were seen by 'a pain specialist or psychologist' but without a record of findings despite evidence of opioid dependency. The largest study in this review is by Campsen *et al.* [2] and related to KAT. Pain and quality-of-life were assessed preoperatively and at 1, 3, and 5 years post-operatively. Assessments included the Beck Depression Inventory (BDI). In 88.9% of patients a 50% reduction in pain was found at 1 year; the BDI fell from 23.7 to 8.2. Only five patients were re-evaluated at 3 years, and three at 5 years. Pain scores had improved but the pain remained. In eight papers in Group B there was no discussion concerning the possibility that some of the patients' symptoms may have had a psychiatric basis.

COMPLICATIONS OF TREATMENT IN GROUPS A AND B

Complications ranged from minor (chest or wound infections) to very serious (nephrectomy of a native or auto-transplanted kidney). Bilateral nephrectomy was considered necessary in some patients, leaving them dependent on life-long renal replacement therapy.

DISCUSSION

In the first publication of what came to be known as loin pain haematuria syndrome, Little *et al.* [1] used investigations available at the time to arrive at a diagnosis. Renal arteriography suggested abnormalities of smaller intra-renal arteries. Burden *et al.* [6, 23] supported this opinion and also noted psychological traits similar to some noted by Little *et al.* [1]. Importantly, Sherwood in 1979 [24] observed that during arteriography 'transient

patches of renal cortical ischaemia' can be seen. In the 1980s, improved imaging showed that the renal vessels were, in fact, normal and the putative abnormalities were artefacts caused by the procedure itself.

With reference to haematuria, the patients we reviewed sometimes reported visible blood or it was detected microscopically or by urine test strips. In 279 of 613 cases the type of haematuria was ignored and in 60 (9.8%) was not reported at all. Similarly, many investigations, often crucial in excluding specific renal diseases, were not performed even when the patients' histories merited it. Fifteen patients had investigations suggesting kidney diseases potentially explaining their haematuria; 10 were glomerular diseases.

There was sometimes failure to report the treatments used or their outcomes. This applied even to radical treatments such as nephrectomy, auto-transplantation, and auto-transplant nephrectomy. Despite employing these forms of surgery many patients continued to complain of pain, sometimes immediately post-operatively, making an underlying physical disorder unlikely.

Little *et al.* [1] commented on the psychological attitude of their patients including anxiety, repeated demands for medical attention and occasionally fabrication of evidence. Eighteen years later, in his book 'The Kidney' [25], de Wardener expanded on these findings.

Our review shows that a psychiatric aspect of the aetiology of LPHS cannot be ignored. We agree with Kelly [10] who commented in his paper on LPHS: 'The roles of illness behaviour, patterns of somatisation and psychiatric disorder associated with chronic pain syndromes, need to be addressed, as do the patterns of medical and surgical response to these patients'. Kelly [10] concluded that in 'at least a subgroup (of LPHS patients) psychological and psychopathological factors are paramount and perhaps explanatory. Nevertheless, these approaches to treatment have been neglected' despite the known association with chronic pain. In Group A, 56 patients had a definite or possible psychiatric illness prior to the onset of their loin pain/haematuria; another 170 patients were considered by their nephrologist or psychiatrist to have pain that was an expression of an underlying psychiatric or psychological condition.

Some patients considered to have LPHS might be examples of Munchausen syndrome, or Munchausen Syndrome by Proxy. This term, coined by Meadow [26], related to a study in 19 children whose mothers fabricated their children's clinical histories and signs. Many of the mothers had been nurses, some having fabricated symptoms or signs in themselves; three were regarded as having Munchausen Syndrome. Factitious haematuria was a feature in seven of the 19 children, some bearing a remarkable similarity to one patient in this review. Haddad *et al.* [27] described a 7-year-old with recurrent loin pain and haematuria as having Munchausen Syndrome by Proxy. Her mother, previously a nurse, had had similar symptoms when younger. After multiple negative investigations, the child's condition was labelled as LPHS. It was then discovered that the blood in the child's urine was not of her blood group but matched the mother's group. The problem was the mother, not the child. Twelve months after the 'cause' of haematuria was found, the child had neither haematuria nor loin pain.

Lucas *et al.* [9] reported that LPHS patients had significantly more unexplained somatic symptoms than matched controls and that the loin pain was significantly related to 'an adverse psychological life event'. They concluded that LPHS might be 'a type of persistent somatoform pain disorder'. The WHO definition of this term is 'persistent, severe and distressing pain that cannot be explained fully by a physiological process or a physical disorder' [28].

Related to the patients' psychiatric state is the high incidence, in both groups of patients, of opioid misuse and overuse amounting to dependency. While not directly related to the patients' psychiatric status, several authors [7, 29, 30] refer to patients whose occupations related to medicine as noted by Little *et al.* [1].

That relief of loin pain was achieved by denervation or auto-transplantation suggests that in some individuals the pain has a genuine physical cause. The explanation for successful pain relief is either interference with the renal nerves (denervation) or removal of the nerves by nephrectomy or auto-transplantation. Such theories leave unanswered the question of why many such procedures have failed to relieve pain.

From the 1970s onwards, efforts were largely directed at better defining the syndrome as described in 1967. After more than 50 years, neither a distinct syndrome nor a convincing aetiology have emerged. By 2017, the content of LPHS publications was changing from a predominantly nephrological search for a cause for the symptoms to surgical treatments for pain. Papers were mostly written by surgeons, anaesthetists and radiologists and not published in nephrological journals. Descriptions in the earlier (Group A) and later (Group B) papers also differ in the absence of psychiatric assessment in the latter group. Psychiatric assessments were often undertaken in earlier studies; psychiatrists were involved in patient management and appeared as authors in many papers. In the 2017–2020 group the need for psychiatric assessment was acknowledged, but there is mention of only one psychologist being involved with patients. It is surprising that many patients continue to undergo major surgery, especially auto-transplantation. Many are vulnerable, often with a psychiatric background, yet are subjected to major surgery in the absence of concrete evidence of somatic pathology.

Our review demonstrates that the earlier investigative search by nephrologists and radiologists for LPHS as a specific diagnosis has been replaced by invasive surgical treatments. A possible psychiatric or psychological basis for LPHS is largely being ignored. Furthermore, the categorisation of LPHS into Types I and II and Types a and b [31, 32] obfuscates an already contentious subject, and should be abandoned.

CONCLUSIONS

The diagnostic approach to patients with suspected LPHS has been haphazard. The strong evidence that it is a somatoform disorder has been overlooked, particularly since 2017. Many investigations and treatments have been invasive and expensive. Some treatments have carried significant morbidity and have often been ineffective in relation to pain control. Patients have been left with long-term ill health, especially patients rendered anephric. Approaches to treatment, especially the use of radical surgery, have been embraced despite lack of a definitive aetiology and despite evidence that LPHS is both recurrent and possibly a psychiatric/psychological disorder. Recent data relating to KAT and less invasive treatments such as renal nerve ablation and neurological stimulation offer hope that patients with severe loin pain, irrespective of its aetiology, may have their symptoms relieved. It is surprising, perhaps even disturbing, that so many patients, particularly in the last 5–10 years, have been submitted to major surgery, especially KAT, without evidence that LPHS exists as a physical disorder or that there is effective long-term treatment.

The assessment of patients presenting with loin pain (sometimes with haematuria) should include thorough psychiatric and occupational histories, examination, and appropriate investigations. While opinions as to whether LPHS is a somatoform pain disorder differ, there can be no doubt that a thorough psychological and psychiatric assessment is mandatory. The increasing use of invasive major surgery in LPHS merits consideration of a randomised controlled trial of the most promising therapies. The rarity of the disorder would, however, necessitate a multi-centre trial.

FOOTNOTE

In 2010, JBE spoke with two authors of the original paper on LPHS (P J Little and H E de Wardener, Ref,1 1967). Both commented on the continued absence over the years of a convincing aetiology for the syndrome.

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DATA AVAILABILITY STATEMENT

No new data were generated or analysed in support of this research.

CONFLICT OF INTEREST STATEMENT

None declared

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