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Review Article

New developments in chyluria after global programs to eliminate lymphatic filariasis

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Abbreviations & Acronyms
DEC = diethyl carbamazine
WHO = World Health Organization

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Abstract: Chyluria, commonly seen in south Asian countries, is mainly a manifestation of lymphatic filariasis as a result of infestation with *Wuchereria bancrofti*, although many other causes can contribute. Many patients can be effectively treated with dietary modifications and drug therapy. The most widely used drug is diethyl carbamazine. The recurrences are common after such treatment. Such patients would benefit from sclerotherapy to obliterate the lympatico-renal fistulae located mainly in the renal pelvicalyceal system. The commonly used sclerosing agent is a combination of 5% povidone-iodine and 50% dextrose instilled through a ureteric catheter. A small percentage of patients who recur after sclerotherapy and those with systemic complications, such as hypoproteinemia and edema, might require surgery in the form of renal hilar lymphatic disconnection. Although it is a major operation, the success rates are >90%. Laparoscopic and robotic techniques have minimized the morbidity related to such surgery. With the advent of the global program for eradication of filariasis initiated by the World Health Organization, the incidence of the disease is decreasing. Mass chemotherapy with diethyl carbamazine is the mainstay of this global program. Many years after eliminating filariasis, chyluria continue to occur in such populations, though in dwindling numbers. Future research should aim at finding more efficacious sclerosing agents with minimal recurrences.

Key words: lymphatic filariasis, protienuria, renal hilar lymphatic disconnection, sclerotherapy, *Wuchereria bancrofti*.

Introduction

Chyluria is the presence of chyle in urine. Chyle is a milky fluid composed of lymph and chylomicrons taken up by lymphatic vessels (lacteals), conveyed to the thoracic duct and then drained into the subclavian vein. Normally, the lymphatic vessels do not communicate with the urinary tract. Whenever there is an abnormal communication between these two systems, chyle leaks into the urine. The point of leakage could occur at the kidney, ureter or bladder level. It is an ancient disease described by Hippocrates in 400 BC.[1] The Indian philosopher Charak described it as "suklameha" in 300 BC.[2]

With the implementation of global programs for elimination of lymphatic filariasis by the WHO, the epidemiology of chyluria is bound to change in the near future. Non-parasitic causes might be seen more commonly as a result of rising numbers of nephron-sparing surgery. Increasing trends in global travel and migration of populations might lead to the occasional occurrence of filarial chyluria in non-endemic areas. The present review on chyluria aims at updating the knowledge as well as discussing the potential challenges urologists would face in the future.

Epidemiology

Chyluria is endemic in India, Bangladesh, Myanmar, Malaysia, Indonesia, China, Philippines, Taiwan and Sri Lanka, and in some parts of Australia and Africa.[2,3] The distribution of chyluria corresponds to this filarial belt of the world map between the latitude of 40° north and 30° south (Fig. 1). It is rare in the West. Chyluria was seen commonly in southern parts of Japan including Okinawa and Kyushu several decades ago.[4] The National Filariasis Control Program, based on treatment with DEC, was introduced in 1962 and helped to eliminate chyluria from Japan in the early 1980s. Even within a country, its distribution is variable. In

Fig. 1 Countries where chyluria is common correspond to the filarial belt of the world map.

India, chyluria is rampant in and around the Ganges river belt, whereas in Sri Lanka it is more common in the urbanized coastal belt.[2] This is related to the increased presence of *Culex* mosquitoes in those areas, which transmit the parasite *Wuchereria bancrofti*. *W. bancrofti* is the cause of chyluria in 95% of cases in tropical countries.[2] It is transmitted by mosquitoes of the genera *Culex*, *Aedes* and *Anopheles*, though *Culex* is the commonest vector involved. Therefore, lymphatic filariasis is seen both in urban as well as rural areas.

Etiology

The etiology of chyluria could be parasitic or non-parasitic. Parasitic causes include filariasis, cysticercosis, echinococcosis, malaria and ascariasis.[5] Out of these, filariasis is far more common than others. In Asian countries, chyluria is almost exclusively as a result of filariasis with *W. bancrofti* infestation.[6] Classically, filariasis can manifest as lymphedema (genital or lower limbs) and tropical pulmonary eosinophillia (Fig. 2). Other presentations would be lymphadenitis, hydrocele and epididymo-orchitis. Approximately 2–10% of those with filariasis are believed to develop chyluria.[7,8] Usually it occurs several years after infestation with *Wuchereria*. Parasites obliterate the lymphatics and cause impairment of the lymphatic flow. Dilation and proliferation of lymphatics in the lumbar and pelvic regions leads to abnormal retrograde or collateral flow between cisterna chyli and renal lymphatics and the renal pelvicalyceal system.[9] Normally renal lymphatics follow the renal vein, lateral aortic nodes, lumbar trunk and cisterna chyli.

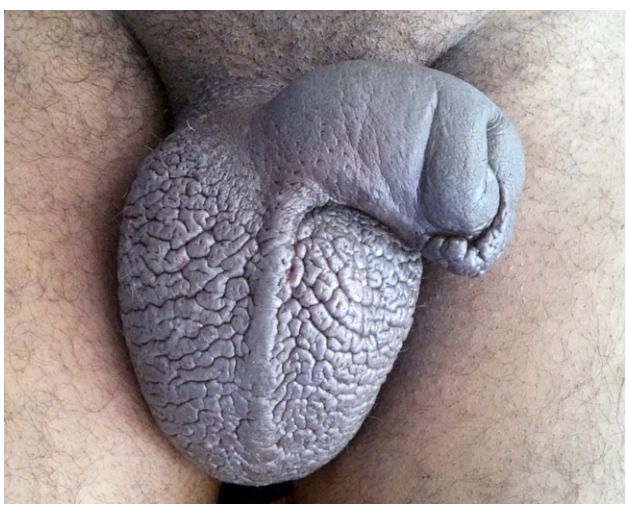

Fig. 2A patient with genital lymphedema as a result of lymphatic filariasis.

Non-parasitic causes include trauma (mainly after partial nephrectomy), lymphatic malformations, infections (tuberculosis, leprosy and mycosis), tumors, radiation, pregnancy, aorto-iliac bypass surgery, lymphangioma of kidney, and bladder and stenoses of the thoracic duct.[5,10,11] The tumors include acute myeloid leukemia and testicular malignancies. Although chyluria should be differentiated from heavy

proteinuria of nephrotic syndrome to avoid mismanagement, the coexistence of the two diseases has been rarely reported.[10,12,13]

Chyluria is rare in the USA and Europe, as lymphatic filariasis is unheard of in those regions. Occasional cases of chyluria seen in the USA and Europe are mainly secondary to surgical trauma and lymphatic malformations.

Pathogenesis

The passage of intestinal lymph in the urine occurs as a result of obstruction or dilation of the retroperitoneal lymphatics.[14] The retroperitoneal lymphatics include those draining the intestine, pancreas and spleen, as well as those draining the kidneys. All these ultimately drain into the cisterna chyli. The exact mechanism of chyluria is not well established; however, it is believed to be as a result of obstruction or regurgitation. According to the obstructive theory, the parasitic infections cause obliterative lymphangitis, which leads to opening up of collaterals leading to backflow of intestinal chyle through alternative pathways.[15] Alternatively, valvular insufficiency of lymphatics causes regurgitation of chyle from the cistern chyli or large intestinal lymph trunks into the renal lymphatics, which in turn causes rupture of these lymphatic channels into the renal pelvicalyceal system (regurgitative theory).[16]

The severity of chyluria is arbitrarily graded into three grades for purposes of easy communication. Grade 1 is the passage of milky urine, whereas grade 2 is the presence of clots. Hematochyluria is considered as grade 3.[17] However, some others believe that a mild degree of hematuria alone is not a predictor of worse severity and poor prognosis. Hence, they classify the presence of hematuria as grade II, and having clots as grade III.[18] The number of calyces of the renal pelvicalyceal system involved at retrograde pyelography can be incorporated into the classification.[1] Involvement of one calyx is considered as mild, whereas two or more is taken as a moderate degree. A severe degree is involvement of all calyces with or without the upper ureter.

There is some evidence that chyluria cause immunological incompetence, as evidenced by a reduction in peripheral T lymphocytes and an even higher incidence of malignancies.[4]

Clinical features

Excretion of milky or cloudy urine is the principal clinical feature (Fig. 3). Flank pain resembling ureteric colic due to ureteral clots and difficulty in passing urine due to bladder clots occurs in severe cases.[19] Chyluria is a rare cause of acute urinary retention.[20] Symptoms are prominent after a fatty meal. Sometimes there can be associated with hematuria (hematochyluria). Severe cases can develop malnutrition, hypoproteinemia, immunodysfunction and hypercoagulability.[2] Chyluria has an unpredictable clinical course with periods of remission. Spontaneous remission can occur in approximately 50% of patients.[21] Although filariasis is the commonest cause of chyluria in tropical countries, most of them do not show any other stigmata of filariasis. However,

Fig. 3 A sample of milky urine from a patient with chyluria.

in one study, 46% of those with chyluria had complications related to the genitourinary tract.[4] In one study, epididymo-orchitis was seen in 19% of the patients with chyluria.[22] This inconsistency might be due to the difficulty in attributing the causality of certain conditions to filariasis. Most studies show a male preponderance, though few report involvement of more females than males.[1,3,18,22,23] The majority of patients with chyluria are aged between 15 and 30 years.[22] Interestingly, the disease involves the left side of the urinary tract, commonly in approximately 71% of patients.[22] In 5% of patients, there is bilateral involvement.

Sometimes chyluria can be mistaken for proteinuria of nephrotic syndrome.[10,24,25] Hypocholesterolemia is a characteristic feature of chyluria, and helps to differentiate it from proteinuria of nephrotic syndrome, where hypercholesterolemia is a feature.[26] This differentiation is critical to avoid unnecessary renal biopsy in patients with heavy proteinuria as a result of chyluria rather than nephrotic syndrome. There are many instances where this has led to unnecessary morbidity. In cases of non-parasitic chyluria, clinical features of the primary illness would be apparent.

Diagnosis

The differential diagnoses of milky urine include phosphaturia, gross pyuria and passage of caseous urine in renal tuberculosis. Turbidity of phosphaturia clears with 10% acetic acid.

Chyluria can be confirmed by a Sudan III test to identify fat globules in the urine. This is a simple and non-expensive test. As this test is not requested commonly, it might be useful to inform the laboratory early to ensure the proper procedure is adhered to. Adding ether to a urine sample is a simple bedside test that can be used to diagnose chyluria.[21] Ether will make the turbidity of chylous urine disappear, as it dissolves fat globules in chylous urine. A fatty meal ingested before the test will increase the yield of the results. A meal containing Sudan III-mixed butter taken 4–6 h before might produce red colored urine in the presence of chyle. The estimation of the urine triglyceride level after a fatty meal is highly specific for chyluria, and is a good way of assessing the response to treatment in a semiquantitative manner.[4,18] A urine triglyceride level >15 mg/dL is indicative of chyluria.[18]

Cystoscopy might reveal milky urine spurting from the ureteric orifice of the affected side. Rarely, chylous efflux might be from the bladder or even posterior urethra.[1] Ureteroscopy, magnetic resonance imaging urogram and lymphoscintigram would help to locate the site, though it is not essential for treatment. Magnetic resonance imaging would show the clusters of dilated lymphatic channels.[3] Lymphangiography can show the site, caliber and number of fistulous communications, but is rarely used nowadays, as it is invasive, time-consuming and technically demanding. Lymphoscintigraphy is much less invasive, and uses Tc-99m diethylenetriamine pentaacetic acid radionucleotides to delineate lymphourinary fistulas.[14] It is the investigation of choice if one wants to identify the exact site, side and extent of the chylous leak. Retrograde pyelography is not specific enough, as contrast leakage can be seen in normal individuals too. Many believe these tests aimed at identifying the leaking site are not cost effective, and would not alter the course of treatment.[5] Identification of the leaking side at cystoscopy before endoscopic sclerotherapy would be sufficient for effective treatment in most cases.

Wuchereria bancrofti shows nocturnal periodicity. Therefore, blood smears and urine should be examined for microfilariae between 22.00 hours and 04.00 hours. In a retrospective study from India, microfilariae were identified in the blood smears of 9% of patients, and in the urine of 11% of patients with chyluria.[27] Filarial serology is not very useful because of its low specificity in endemic areas, as filarial immunoglobulin G antibodies are positive in 3% of the population in such areas.[27] Filarial antigen test using the enzyme-linked immunosorbent assay technique or immunochromatographic card test are useful for a more specific diagnosis.[28] A blood count can show eosinophilia, as in many other parasitic infestations. Recently, in technologically advanced settings, filarial DNA of the adult worms in the blood or lymphatic system has been used to make a diagnosis.[29]

Treatment

Dietary modifications

The reduction of triglycerides in the diet minimizes the production of chylomicrons, and hence reduces the chyluria. Medium chain triglycerides are absorbed directly into blood vessels in the intestinal villi, and hence do not contribute to chylomicrons and chyluria. This could be a reason for the changing severity of chyluria seen in different countries with different dietary habits. Hence, populations that consume coconut milk for cooking, which is rich in medium chain triglycerides (<12 carbon atoms), will have less severe chyluria compared with those who consume ghee as the cooking medium. This could be why chyluria is much less severe in Sri Lanka, where coconut milk is used widely compared with northern India, where ghee is the cooking medium. Replacing long chain fatty acids with medium chain fatty acids in the diet reduces the severity promptly. There might be a place for total parenteral nutrition to provide absolute enteric rest in intractable chyluria.[1]

The restriction of fat might precipitate malnutrition in communities with poor socioeconomic conditions where filariasis is generally common and sources of medium chain triglycerides might be more expensive, leading to poor compliance.[18] Data related to the optimum period of dietary modifications required is sparse. Most recommend dietary modifications as an adjunct to drug therapy. Therefore, the success rate of dietary modifications alone is not well established. Avoiding exertion of the body, and binders to increase the abdominal pressure on lymphatics are beneficial to reducing the severity of chyluria.

Drug therapy

Most cases of chyluria as a result of filariasis respond to treatment with DEC 6 mg/kg/day for 21 days, and DEC is the most commonly used drug. The alternative drugs are albendazole 400 mg/day for 14 days, and ivermectin 6–12 mg as a single dose and repeated after 3 weeks.[1] Doxycycline also has been tried with success in situations where response to DEC has been poor.[29,30] Longer and repeated courses of treatment might be required before symptoms disappear in some cases. DEC acts against the larval stage of the parasite, known as microfilaria. It makes the microfilariae susceptible to phagocytes. The adverse effects of DEC are proportionate to the microfilaremic burden, and in severe cases an acute febrile reaction might occur. Hence, DEC can be combined with an antihistamine agent to minimize such adverse events. The success rate of dietary and drug therapy is approximately 60%.[22] Treatment with DEC can be repeated after 6–8 weeks if the response to the initial course is poor. Those who do not improve with two to three courses of drug therapy will require invasive procedures – endoscopic sclerotherapy or renal pedicle lymphatic disconnection. Use of ACE inhibitors in the treatment has been described, but has not been proved to be successful subsequently.[10]

Endoscopic sclerotherapy

In most cases of chyluria, the abnormal lympho-urinary connections are located in the renal pelvicalyceal system. Instillation of sclerosing agents into the pelvicalyceal system blocks the channels by inflammatory edema initially, and by fibrosis later. Agents that have been used for endoscopic sclerotherapy are 0.1–0.5% silver nitrate solution, 0.2–5% povidone-

iodine, 50% dextrose, 3% hypertonic saline, 10–25% potassium iodide and contrast media used in radiology.[1,5,17] Some claim superior efficacy with a combination of the sclerosing agents; for example, povidone-iodine and dextrose.[31]

The volume of the pelvicalyceal system is approximately 8 mL. Hence, approximately 8–10 mL of the sclerosing agent is instilled into the affected renal pelvis through a ureteric catheter of 5–6-Fr caliber, and then drained off.[32,33] The success rate is approximately 80%, and the procedure can be repeated to increase the success rate. There is no consensus on when and how often should it be repeated. However, many urologists would try at least three attempts before abandoning the procedure and embark on more invasive options. One study found that 3-day, 8-hourly instillation was more convenient to the patient than weekly instillation of silver nitrate solution for 6 weeks.[34] Here, the ureteric catheter is kept in place anchored to a Foley urethral catheter, and every 8 h the patient is tilted to a 15° head low position before a sclerosant is instilled. The affected side is determined by observation of the ureteric orifice for chylous efflux during cystoscopy. It can take approximately 15 min for it to be apparent. Pressure on the kidney might expedite the chylous efflux. When there is bilateral involvement, a gap of at least 2 months is recommended between the two sessions.[1]

As brand preparations of ready to use silver nitrate are not available, supply of sterile solutions of sclerosing agents could be a practical problem and might need special requests. In preparation of such solutions, it is important to ensure that the exact percentage and strength is maintained during the process. Inadvertent use of excessive strength solutions might cause deleterious side-effects, such as acute necrotizing ureteritis, which could even be fatal.[35–37] The need for freshly prepared solution on every occasion and storing in a dark bottle to avoid exposure to sunlight have made silver nitrate a less popular sclerosant.

Povidone-iodine has become a popular agent at present, as it is easily available, water soluble, less reactive, cheap and stable at room temperature. Mixing 2 mL of 5% povidone-iodine with 8 mL of sterile water will produce the required fresh solution suitable for sclerotherapy. A mixture of 5% povidone-iodine and 50% dextrose has been used effectively and safely over a long period of time with a 6-month cure rate of 90%.[2] This combination has recently become the most widely used sclerosing solution.

Those who develop recurrences within a short period of time will have a poor response to repeat sclerotherapy too.[32] The common adverse effects of sclerotherapy are transient, and include nausea, vomiting, flank pain and hematuria. Chyluria has been successfully treated with endoscopic coagulation of leak points, but is not widely used, as it is technically demanding and time-consuming.[38] Those who develop urinary retention as a result of chylous clots would require bladder washouts.

Surgical treatment

Occasionally, patients with refractory chyluria who do not respond to the aforementioned measures, especially those with systemic complications, such as extensive weight loss, hypoproteinemia and edema, recurrent clot retention, and immunodeficiency, require surgical intervention.[1,2] The surgical procedures for chyluria aim at disconnection of lymphatics to the kidney and ureter or creating a lymphovenous anastomosis. The lymphatic disconnection includes nephrolympholysis, hilar stripping, ureterolympholysis, fasciectomy and nephropexy.

Nephrolympholysis includes separation of the kidney from the surrounding perirenal fat. Skeletonization of the renal pedicle vessels, and ligation and division of ensuing lymphatics constitute hilar stripping. Dissection of the ureter away from periureteric tissues to disconnect lymphatics is uretrolympholysis. It can be carried out for 3–5 cm of the upper ureter or up to iliac vessels initially, and then the response can be assessed. If the leak continues and occurs from the lower parts of the ureter, it should be completed later. Fasciectomy is removal of the separated perirenal fat and tissues. The mobilized and isolated kidney by the aforementioned measures is anchored to the psoas muscle to avoid torsion in nephropexy. Many would not embark on all five steps. The aforementioned procedures can be carried out as open procedures or by laparoscopy. Laparoscopic surgery has the advantages of magnified view, minimal morbidity, shorter hospital stay and better cosmesis. Recently, robotic surgery has been used as an adjunct to improve the outcome.[1,39] Although renal hilar lymphatic disconnection surgery is a major operation, it has the best outcome in terms of cure with success rates >90%.

Autotransplantation is another way of achieving lymphatic disconnection. Nephrectomy is no longer considered an option in treatment of chyluria unless the kidney is non-functioning or damaged after other interventions.

Lymphovenous anastomosis aims at reducing the intralymphatic pressure, which is believed to cause chyluria. The lymphatics are of thin caliber, and the procedure requires microsurgical instruments. It can be done retroperitoneally at the renal hilar level by anastomosing a hilar lymphatic to the gonadal vein, in the inguinal region or in the dorsum of the foot.[40–42] A less technically demanding alternative is to anastomose the inguinal lymph node to a tributary of the adjacent saphenous vein.[43] The success rates of these procedures are approximately 50%.

Chyluria secondary to partial nephrectomy resolves with time.[44] In the interim period, the dietary measures mentioned above might be useful. Early invasive interventions might cause further morbidity, and patience will pay off ultimately. Chyluria after radical nephrectomy is rarer than after partial nephrectomy, and can be treated with injection of cyanoacrylate glue.[45] Post-traumatic chyluria as a result of lymphorenal fistula has been treated with somatostatin or octreotide therapy with success.[46,47] In other instances of chyluria, where there is an identifiable cause, the correction of the underlying etiological factor would help to resolve the chyluria.

Prognosis

Most patients with chyluria respond to treatment and have a good prognosis (Table 1). The long-term cure rate is highest after renal hilar lymphatic disconnection surgery, which is >90%. Endoscopic sclerotherapy has success rates reaching 80%, though recurrences occur.[22,34] Drug and dietary measures have a lower success rate of approximately 50–60%.

Table 1 Outcome of different therapeutic interventions

Intervention	Success rate (%)	Recurrence rate (%)
Drug therapy and dietary measures	50–60	40–50†
Endoscopic sclerotherapy	80	10–40†
Lymphovenous anastomosis	50	–
Renal hilar lymphatic dissection	90–100	0–10

†These interventions can be easily repeated to treat recurrences.

However, the latter two therapeutic options can be repeated with ease. Those who have had previously failed treatment and high urinary triglyceride levels are more likely to have a poor response to treatment. Duration of the disease is not a risk factor for failure with drug treatment.[18] Extreme cases of heavy chyluria can lead to severe malnutrition, hypoproteinemia and even death. This phenomenon is rare. Those with an underlying serious cause might develop morbidity related to the cause rather than chyluria.

Prevention

Elimination of bancroftian filariasis is associated with a reduction of incidence of chyluria, as seen in some countries, such as Japan. The reduction of chyluria incidence becomes evident after a latent period of several years. Traditionally, control of filariasis was achieved by improving environmental cleanliness, thereby eliminating breeding sites of the *Culex* mosquitoes. In recent years, mass chemoprophylaxis has been found to be effective in endemic areas by reducing the reservoir of the parasite. This is achieved by administering a single dose of DEC 6 mg/kg with or without albendazole 400 mg. It has been found that a single dose of DEC alone is as good as a combination of DEC and albendazole.[48] The target is to provide chemoprophylaxis cover to 90% of the vulnerable population.[49]

Lymphatic filariasis, which was endemic in 81 countries by the turn of the new millennium, was a neglected disease in the developing world, until it was recognized as the leading cause of physical disability worldwide.[50,51] Hence, in 2000, the WHO started a global eradication campaign to eliminate filariasis. In Sri Lanka, five rounds of DEC and albendazole were given from 2002 to 2006, and this helped Sri Lanka to receive filariasis elimination status by the WHO in 2016.[52]

Conclusion

The necessity for major surgery to treat chyluria might become less in the future due to changes expected in the etiology and epidemiology of chyluria after lymphatic filariasis elimination programs and increasing international travel. New research should be aimed at identifying more efficient sclerosing agents to treat chyluria with lower recurrence rates. Chyluria will be encountered by urologists for a few decades to come, even after elimination of lymphatic filariasis, and those working in the filarial endemic regions should be aware of the new developments related to it.

Conflict of interest

None declared.

References

1 Sharma S, Hemal AK. Chyluria – An overview. *Int. J. Nephrol. Urol.* 2009; **1**: 14–26.
2 Sinha RK, Ranjan N, Singh N, Amit K. Chyluria: a scourge of our region. *BMJ Case Rep.* 2015; bcr2014209188.
3 Sunder S, Jayaraman R, Mahapatra HS *et al.* Analysis of case series of milky urine: a single center and departmental clinical experience with emphasis on management perspectives: a prospective observational study. *Urol. Ann.* 2014; **6**: 340–5.
4 Kimura E, Itoh M. Filariais in Japan some 25 years after its eradication. *Trop. Med. Health* 2011; **39** (Suppl 2): 57–63.
5 Dalela D. Issues in etiology and diagnosis making of chyluria. *Indian J. Urol.* 2005; **21**: 18–23.
6 Taylor MG, Hoerauf A, Bockarie M. Lymphatic filariasis and onchocerciasis. *Lancet* 2010; **376**: 1175–85.
7 Diamond E, Scahapira HE. Chyluria-a review of literature. *Urology* 1985; **26**: 427–31.
8 Brunkwall J, Simonsen O, Bergqvist D, Jonsson K, Bergentz S. Chyluria treated with renal autotransplantation: A case report. *J. Urol.* 1990; **143**: 793–6.
9 Yu HHY, Ngan H, Leong CH. Chyluria – a 10 year followup. *BJU* 1978; **50**: 126–33.
10 Graziani G, Cucchiari D, Verdesca S, Balzarini L, Montanlli A, Ponticelli C. Chyluria associated with nephritic-range proteinuria: pathophysiology, clinical picture and therapeutic options. *Nephron Clin. Pract.* 2011; **119**: c248-54.
11 Stalens JP, Falk M, Howmann-Giles R, Roy LP. Milky urine-a child with chyluria. *Eur. J. Pediatr.* 1992; **151**: 61–2.
12 Lewsuwan S, Kanjanabuch T, Avihingsanon Y, Praditpornsilpa K, Eiam-Ong S. A rare case of chylous ascites and chyluria in an adult nephrotic syndrome with focal segmental glomerulosclerosis. *J. Med. Assoc. Thai.* 2006; **89** (Suppl 2): S253–6.
13 Kano K, Arisaka O. Chyluria due to retroperitoneal lymphangioma producing nephritic syndrome. *J. Pediatr.* 2003; **143**: 685.
14 Singh I, Dargan P, Sharma N. Chyluria-a clinical and diagnostic stepladder algorithm with review of literature. *Indian J. Urol.* 2004; **20**: 79–85.
15 Ut A, Aung STT. Chyluria. *Clin. Rad.* 1975; **26**: 237–42.
16 Ngan H, Leong CH. A lymphographic study of chyluria. *Br. J. Radiol.* 1977; **50**: 863–70.
17 Suri A, Kumar A. Chyluria-SGPGI experience. *Indian J. Urol.* 2005; **21**: 59–62.
18 Goyal NK, Goel A, Sankhwar S *et al.* Factors affecting response to medical management in patients of filarial chyluria: a prospective study. *Indian J. Urol.* 2014; **30**: 23–7.
19 Abeygunasekera A, Bulegoda H, Nirupika H. A case of chyluria. *Ceylon Med. J.* 2003; **48**: 34.
20 Darrad M, Basu S, Viswanathan C. Non-parasitic chyluria: a rare cause of acute urinary retention in a young Caucasian male. *J. Clin. Urol.* 2017; **10**: 197–9.
21 Ohyama C, Saita H, Miyasato N. Spontaneous remission of chyluria. *J. Urol.* 1979; **121**: 316–7.
22 Tandon V, Singh H, Dwivedi US, Mahmood M, Singh PB. Filarial chyluria: long-term experience of a university hospital in India. *Int. J. Urol.* 2004; **11**: 193–8.
23 Akisada M, Tani S. Filarial chyluria in Japan- lymphography, etiology and treatment in 30 cases. *Radiology* 1968; **90**: 311–7.
24 Saha M, Ray S, Goswami M *et al.* An occult filarial infection presenting as chyluria with proteinuria: a case report and review of literature. *BMJ Case Rep.* 2012; bcr0120125635.
25 Cheng JT, Mohan S, Nasr SH, D'Agati VD. Chyluria presenting as milky urine and nephrotic-range proteinuria. *Kidney Int.* 2006; **70**: 1518–22.
26 Wijayarathna S, Abeygunasekera A. Reply – non-parasitic chyluria: a rare cause of acute urinary retention in a young Caucasian male. *J. Clin. Urol.* 2016; **9**: 436.
27 Mehta VK, Lohar H, Banerjee GK, Reddy MV, Harinath BC. Surgical filariasis: immunoscreening for filarial IgG antibodies using Wuchereria bancrofti microfilarial excretory-secretory antigen. *J. Commun. Dis.* 1999; **31**: 35–40.

28 Well GJ, Ramzy RM. Diagnostic tools for filariasis elimination programs. *Trends Parasitol.* 2007; **23**: 78–82.
29 Hagiya H, Terasaka T, Kimura K *et al.* Filarial chyluria as a rare cause of urinary retention. *Intern. Med.* 2014; **53**: 2001–5.
30 Franco-Paresdes C, Hidron A, Steinberg J. A woman from British Guyana with recurrent back pain and fever: chyluria associated with infection due to *Wuchereria bancrofti*. *Clin. Infect. Dis.* 2006; **42**: 1297.
31 Nandy PR, Dwivedi US, Vyas N, Prasad M, Dutta B, Singh PB. Providone iodine and dextrose solution combination sclerotherapy in chyluria. *Urology* 2004; **64**: 1107–9.
32 Singh KJ, Srivastava A. Nonsurgical management of chyluria (sclerotherapy). *Indian J. Urol.* 2005; **21**: 55–8.
33 Sabnis RB, Punekar SV, Desai RM, Bradoo AM, Bapat SD. Instillation of silver nitrate in the treatment of chyluria. *Br. J. Urol.* 1992; **70**: 660–2.
34 Dalela D, Rastogi M, Goel A. Silver nitrate sclerotherapy for clinically significant chyluria: a prospective evaluation of duration of therapy. *Urol. Int.* 2004; **72**: 335–40.
35 Mandhani A, Kapoor R, Gupta RK, Rao HS. Can silver nitrate instillation for the treatment of chyluria be fatal? *Br. J. Urol.* 1998; **82**: 926–7.
36 Dash SC, Bhargav Y, Saxena S, Agarwal SK, Tiwari SC, Dinda A. Acute renal failure and renal papillary necrosis following instillation of silver nitrate for treatment of chyluria. *Nephrol. Dial. Transplant.* 1996; **11**: 1841–2.
37 Su CM, lee YC, Wu WJ, Ke H, Chou YH, Huang CH. Acute necrotizing ureteritis with obstructive uropathy following instillation of silver nitrate in chyluria: a case report. *Kaohsiung J. Med. Sci.* 2004; **20**: 512–5.
38 Yagi S, Goto T, Kawamoto K, Miyawaki I. Endoscopic treatment of refractory filarial chyluria: a preliminary report. *J. Urol.* 1998; **159**: 1615–8.
39 Badani KK, Hemal AK, Peabody JO, Menon M. Robotic radical prostatectomy: the Vatikutti Urology Institute training experience. *World J. Urol.* 2006; **24**: 148–51.
40 Ji YZ, Zheng JH, Chen JN, Wu ZD. Microsurgery in the treatment of chyluria and scrotal lymphangial fistula. *Br. J. Urol.* 1993; **72**: 952–4.
41 Cockett AT, Goodwin WE. Chyluria: attempted surgical treatment by lymphatic-venous anastomosis. *J. Urol.* 1962; **88**: 566–8.
42 Zhao WP, Hou LQ, Shen JL. Summary and prospects of fourteen years' experience with treatment of chyluria by microsurgery. *Eur. Urol.* 1988; **15**: 219–22.
43 Hou LQ, Liu QY, Kong QY *et al.* Lymphonodovenous anastomosis in the treatment of chyluria. *Chin. Med. J. Eng.* 1991; **104**: 392–4.
44 Kim RJ, Joudi FN. Chyluria after partial nephrectomy: case report and review of the literature. *ScientificWorldJournal* 2009; **9**: 1–4.
45 Tuck J, Pearce L, Pantlides M. Chyluria after radical nephrectomy treated with N-butyl-2-cyanoacrylate. *J. Urol.* 2000; **164**: 778–9.
46 Campieri C, Raimondi C, Dalmastri V *et al.* Posttraumatic chyluria due to lymphorenal fistula regressed after somatostatin therapy. *Nephron* 1996; **72**: 705–7.
47 Giordano M, Crillo D, Baron I *et al.* Treatment of post-traumatic chyluria with subcutaneous octreotide administration. *Nephrol. Dial. Transplant.* 1996; **11**: 365–7.
48 Horton J, Witt C, Ottsesen LA *et al.* An analysis of the safety of the single dose, two drug regimens used in programmes to eliminate lymphatic filariasis. *Parasitology* 2000; **121** (Suppl): S147–60.
49 Nandha B, Sadanandane C, Jambulingam P, Das P. Delivery strategy of mass annual single dose DEC administration to eliminate lymphatic filariasis in the urban areas of Pondicherry, South India: 5 years of experience. *Filaria J.* 2007; **6**: 7–13.
50 Zeldenryk LM, Gray M, Spare R, Gordon S, Melrose W. The emerging story of disability associated with lymphatic filariasis: a critical review. *PLoS Neg. Trop. Dis.* 2011; **5**: e1366.
51 Molyneux DH, Zagaria N. Lymphatic filariasis elimination: progress in global programme development. *Ann. Trop. Med. Parasitol.* 2002; **96**: 15–40.
52 Rao RU, Nagodavithana SD, Wijegunawardana AD *et al.* A comprehensive assessment of lymphatic filariasis in Sri Lanka six years after cessation of mass drug administration. *PLoS Negl. Trop Dis.* 2014; **8**: e3281.