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Acute Lower Extremity Nerve Compression Syndromes caused by Retroperitoneal Hæmatoma; Medical, Surgical & Rehabilitation Perspectives

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Background

Acute lower extremity nerve compression syndromes due to retroperitoneal haematomas within the psoas major and iliacus muscle compartments are increasingly common clinical entities. Historically these were rare clinical presentations associated with haemophilia & other bleeding diathesis [1]; however, the increased utilisation of anticoagulation and antiplatelet medications in clinical practice has contributed to a rising incidence of haemorrhagic complications [2]. Associated with this is an apparent increase in the incidence of retroperitoneal haematomas and consequent nerve injuries.

There is limited awareness of the neurological sequelae of retroperitoneal haematomas amongst clinicians, leading to delays in diagnosis and management observed within clinical practice [3, 4] which may contribute to adverse clinical outcomes. This is further compounded by the lack of clear guidelines for diagnostic investigations, management and rehabilitation protocols [5]. Available literature is largely restricted to case reports and case series/reviews that are limited by both their heterogeneity and the use of non-uniform language.

This article aims to reflect the current state of knowledge around this clinical issue by reviewing the available scientific publications, alongside the authors' clinical insights and reasoning in order to improve awareness amongst clinicians.

Anatomy: The Iliacus muscle arises from the iliac fossa of the innominate bone. Psoas major originates from the 12th thoracic to 5th lumbar vertebrae. Both are contained within tight osteo-fascial compartments. The origin of psoas major is arranged in two components; the anterior portion from the vertebral bodies and the posterior portion from the transverse processes. The lumbar plexus lies in a plane between these components [6]. Iliacus and psoas major join and pass deep to the inguinal ligament, eventually inserting onto the lesser trochanter of the femur. Iliacus and psoas major muscles contribute to flexion and external rotation at the hip joint [7]. The fascial sheaths of these muscles

merge in distally in a dense funnel, where the fascial compartments are continuous with each other [1].

Ventral rami of the first four lumbar segmental nerves enter the psoas major muscle as they exit from the intervertebral foramina and form the lumbar plexus within the substance of this muscle. The lumbar plexus lies close to the coronal plane [8] and is a deep structure with limited susceptibility to direct trauma [9]. Nerves arising from the lumbar plexus are classified according to their exit in relation to the psoas major muscle margins. The genitofemoral nerve exits anteriorly. The iliohypogastric, ilioinguinal, femoral and lateral cutaneous nerves exit lateral to psoas major [7, 10]. The obturator and nervus furcalis exit medial to psoas major [11].

Although the lumbar and sacral plexuses are commonly considered together, they are anatomically distinct and non-contiguous, commonly affected by differing pathological insults. The nervus furcalis (meaning forked nerve) arising from the medial psoas margin, is the only anatomical connection between these plexuses. It arises from L4 nerve root and contributes branches to femoral and obturator nerves in addition to the lumbosacral trunk [11], thereby contributing to the nerve supply of sacral plexus innervated muscles including gluteus minimus, gluteus medius, tensor fasica latae and tibialis anterior.

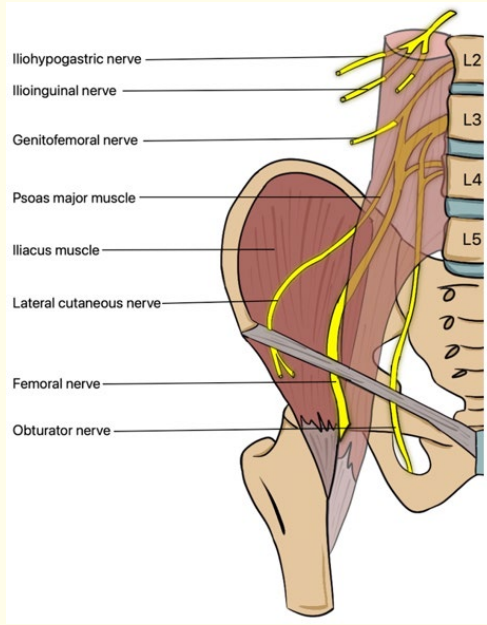

Figure 1: Demonstrating the anatomy of the lumbar plexus within psoas major.

The femoral nerve arises from the lumbar plexus, principally innervating quadriceps. Additionally, it innervates iliacus, sartorius & part of pectineus within the femoral triangle. Its anterior division extends sensory branches to supply the skin of most of the anterior thigh & knee. The posterior division contributes motor branches to quadriceps and continues as the saphenous nerve supplying sensation to the medial lower leg, ankle & foot [7]. Following exit from the lateral margin of psoas, the femoral nerve extends to run in a groove between psoas major & iliacus and enters the thigh deep to the inguinal ligament [2]. The femoral nerve is vulnerable within the iliopsoas gutter for several reasons; firstly, the transversalis fascia overlying the iliacus muscle and femoral nerve is not distensible in the presence of haematoma [12]. Haematoma may progress down the iliac muscle into the femoral canal where the femoral nerve may be compressed against the inguinal ligament. Finally, the nerve is also vulnerable to ischemia due to its limited vascular supply [13].

The obturator nerve innervates most of the adductor compartment of the thigh; adductor longus, adductor brevis, gracilis & obturator externus muscles alongside segments of pectineus & adductor magnus. Sensation is also provided to the hip joint and skin of the medial thigh. The lateral femoral cutaneous nerve supplies skin sensation to much of the lateral thigh. The Ilio-hypogastric, ilio-inguinal & genito-femoral nerves supply cutaneous sensation in the lower abdomen, groin and pubic part of external genitalia. The latter also supplies the cremaster muscle [8].

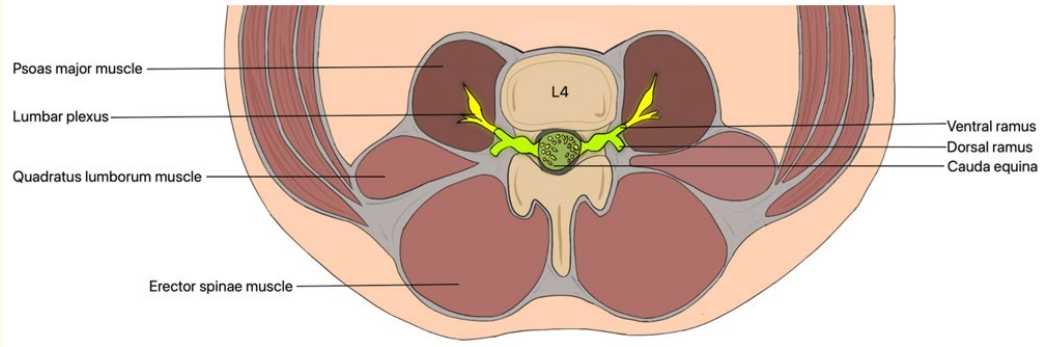

Figure 2: Demonstrating the axial anatomy and orientation of the lumbar plexus within the psoas major muscle.

The Psoas minor muscle arises from the 12th thoracic & first lumbar vertebral bodies, runs alongside psoas major and inserts to the ilio-pectineal eminence [6]. In a minority, this muscle is absent either unilaterally or bilaterally. Psoas tertius and psoas quartus have been described in dissection, with suggestions of possible nerve entrapment due to these anomalies [14]. Like brachial plexus, lumbar plexus may also exhibit 'pre-fixed' and post-fixed' variations along with changes in vertebral structure (such as sacralisation of the 5th lumbar vertebra) and psoas muscle attachments. Variations in the course of femoral nerve in relation to iliacus & iliac fascia have been identified and may predispose to nerve compression syndromes [15].

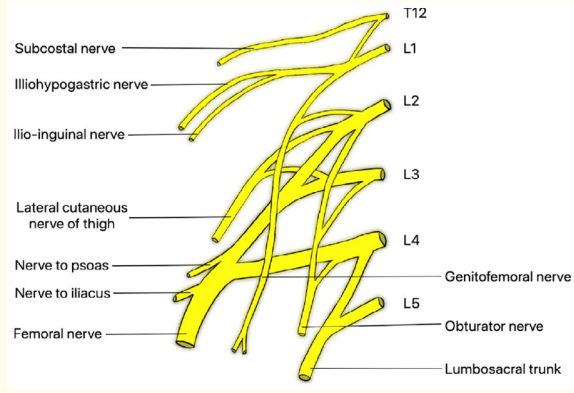

Figure 3: The nerves of the lumbar plexus.

Aetiology: There is a number of potential causes of lumbar plexopathy; however the scope of this article is limited to acute haematomas within the osteo-fascial compartments of iliacus and psoas major muscles. Early case reports identified haemophilia as the underlying aetiology of iliopsoas haematoma formation [1]. More recent publications cite broader aetiologies of haematoma formation including spontaneous [16], von Willebrand's disease [17], traumatic injury [3, 18], vertebral compression fracture [19], osteopathic manipulation [20], post spinal surgery [21, 22], following femoral artery/vein catheterisation [5], as a consequence of severe hypertension [23], and following hip hemiarthroplasty [24]. Coagulopathy is commonly identified in case reports, with the majority of case reports receiving anticoagulation. Cases have cited thrombolysis [25], liver disease [26], and antiplatelets medications [27] as contributory. Cases have been reported in association with ruptured abdominal aortic aneurysm [5]. Multiple aetiologies may be apparent.

There is no identified literature that highlights the role of pre-existing neuropathies that may induce susceptibility to acute nerve injury and adverse outcomes. Similarly, the role of drugs or patients' nutritional status creating susceptibility to lumbar plexus lesions following haematoma formation has not been explored.

It has been observed in clinical practice that patients affected by haematomas whilst using anticoagulants & antiplatelet agents may experience multiple haematomas into musculature both simultaneously and sequentially, without other organ systems being affected by bleeding (such as the gastrointestinal or urinary tracts). The reasons for this is unclear but may relate to the intensity of anticoagulation [2].

Incidence: There is limited data regarding incidence of haematoma formation with nerve compression. Prior estimates of iliopsoas haematoma incidence being between 0.003% and 0.6% of patients with predisposing factors [5], ranging to estimates of the incidence of retroperitoneal haematoma being between 0.6% and 6.6% of patients receiving therapeutic anticoagulation [2]. Within clinical practice, the incidence appears to have increased, with the authors typically encountering 2-3 cases per year referred for rehabilitation in a catchment of 0.5 million patients. As these cases likely reflect more severe neurological sequelae this may be only a fraction of the true incidence.

The authors' experience suggests a male predominance (which may reflect a male predominance of cardiovascular disease requiring anticoagulation) and also a left sided predominance in unilateral presentations. Additionally, there are some muscles more predisposed to haematoma formation such as iliacus, psoas major, and quadriceps. The reasons for this are not established. The observed higher incidence in clinical practice of left sided unilateral haematoma (if it is real), is hypothesised to be related to the pressure of the abdominal aorta upon the left lumbar veins and the pressure of the right common iliac artery on the left common iliac vein, resulting in elevated venous and capillary pressures in left psoas & iliacus muscles. In the majority of cases, it remains uncertain as to which vessel is the source of bleeding, and whether it is arterial, venous or mixed. The 3rd and 4th lumbar arteries have been highlighted as culprit vessels in case reports [19, 28-30]. It is also observed in patients with iliopsoas haematoma occurring post stroke, the haematoma forms more commonly on the hemiplegic side. This is for uncertain reasons but may reflect a consequence of sensory inattention, spasticity, or muscle imbalance as a result of stroke.

Pathophysiology: Unfortunately, neurological injury as a sequalae of haematoma is an often delayed diagnosis [31] and symptoms occurrence may be delayed following an insult [19, 30]. Arterial bleeding may be identified on imaging, and due to elevated pressures of accumulation is more likely to cause compartment syndrome-like physiology in iliopsoas [32]. Venous bleeding often progresses more slowly, with more delayed occurrence of symptoms.

Haematoma occurring more proximally in either iliacus or psoas major is may result in compartment syndrome of the individual muscle, with associated neuropathy, with psoas major haematoma causing whole or partial lumbar plexus palsy [19, 33, 34], and iliacus haematomas causing isolated femoral nerve palsy [1]. The lateral femoral cutaneous nerve may be affected on its course passing psoas major before entering a tunnel between the inguinal ligament and the anterior superior iliac spine [35]. The aetiology of nerve injury may be related to sustained pressure, stretching, ischemic injury, breakdown of blood products and inflammation, or by combined mechanisms.

Compartment syndrome is caused by a sustained rise in fascial compartment pressures resulting in necrosis of the contained muscle and pressure palsies of the nerves traversing the compartment. At compartment pressures over 30 mmHg, capillaries are compressed, and muscle ischemia ensues, causing a well-recognised clinical syndrome of disproportionate pain that is unresponsive to analgesia, with worsening pain observed on stretching the affected muscle. There is little data regarding how much pressure, and sustained over what period will result in neurological injury. Large diameter A-β fibres are most vulnerable to compression and ischemia [36] with associated sensory loss.

Many secondary events and complications can occur following retroperitoneal haematoma formation including shock, anaemia, rhabdomyolysis [5], abdominal compartment syndrome [37], and death. Complications of nerve palsies include lower motor neurone (LMN) syndrome with wasting, contractures, osteopenia, complex regional pain syndrome (CRPS), falls, and secondary injuries over insensate skin areas [9].

Nerve injury can be graded into 3 (Seddon) or 5 (Sunderland) classes.

Seddon's classification

Neuropraxia is associated with no structural damage to the axon; there is segmental demyelination and a potentially reversible conduction block. Prognosis for recovery is good, with recovery typically being complete occurring over days to weeks. This occurs in a random pattern without motor march.

Axonotmesis is caused by axon damage with preservation of the endoneurium tube. The nerve degenerates distally from the site of the injury (Wallerian/antegrade degeneration) followed by slow (approximately 1 mm/day) and progressive recovery occurring over several months. Eventually nerve fibres reach their original targets as the endoneurium tube is intact across the injury site. If recovery is delayed beyond 18 months, the affected muscle may not recover, despite the nerve recovery reaching it, due to motor end plate death.

Neurotmesis is the most severe injury and involves anatomic severance of the nerve, which may be bluntly or sharply transected. Neurotmesis is associated with poor recovery. Neuroma formation and cross-connection among sensory, motor & sympathetic nerve fibres can occur, resulting in adverse clinical recovery and unwanted secondary phenomena such as CRPS [38].

It may be observed that differing grades of nerve injury can be identified within the plexus or nerve. Therefore, it is challenging to establish the grade of nerve injury resulting from retroperitoneal haematomas. Serial examination over several weeks may be informative [38]. If neurological deficits persist beyond 6-8 weeks, then it is likely to be a more severe injury than neuropraxia. If serial clinical & electromyography (EMG) examinations show proximal to distal marching recovery of the muscles affected, that would suggest axonotmesis. If no recovery occurs over many months, neurotmesis is likely. The optimal timing of electrodiagnostic studies following nerve injury is uncertain, though grading of injury severity usually requires delays of at least 1-2 weeks, and 3-4 weeks for the maximum diagnostic information to become apparent [38]. Even a transected nerve will take 3-5 days for Wallerian degeneration to result in observable changes to occur on EMG [39].

Presentation, clinical features: Pain is a prominent feature in the majority of cases [1] and is often disproportionate to any preceding injury [34] and may have limited response to analgesia. Stretch pain is common. Patients tend to sit or lie with a flexed hip joint, to relieve the pressure in the swollen compartments. In post-stroke patient with bleeding on the hemiplegic – hemi-anaesthetic side, pain may be less prominent.

In the weeks following the initial bleeding event, this pain gradually improves. However, new symptoms of neuropathic pain & paraesthesia may develop in the lower limb if there is neurological involvement. Features of CRPS may appear in the months following the initial insult and nerve palsy.

Bruising over the lower abdomen, flank or groin may appear, suggesting breaching of the compartment. Where this occurs, it cannot be assumed that the affected nerves are adequately decompressed. Swelling of the groin and thigh is often seen. A tender mass may be felt above and below the inguinal ligament [1, 20]. Psoas enlargement may be palpated as a cylindrical mass in the iliac fossa on examination. On the left side, this can be mistaken for descending colon distended from constipation, particularly in the hemi-anaesthetic patient who may not demonstrates tenderness. In those with upper motor neuron lesions (such as stroke), a hyper-reflexic knee jerk reflex may become hypo-reflexic, because of the new lower motor neuron (LMN) pathology.

Patients may present as shocked with anaemia, tachycardia and hypotension developing over hours-days [5]. The priority at this time is resuscitation, and patients may progress to a fatal outcome [40]. Features of underlying conditions like cirrhosis with hypersplenism may be present [26]. Acute kidney injury may develop due to shock, rhabdomyolysis or distortion of the ureter [41]. Comorbidities such as cirrhosis and cardiovascular conditions are correlated to mortality, as is late recognition of haematoma formation [26].

Occasionally, neurological deficits may be the presenting feature, typically developing over hours to days. These may develop in a progressive manner with early sensory involvement; commonly paraesthesia in the saphenous nerve (a branch of the femoral nerve) distribution, progressing to anaesthesia and possibly weakness [42]. The presentation may be due to groin pain and increasing difficulty with mobilising [34]. Serial examination in the early phase, noting progression of neurological deficits may be valuable to identify suitable patients to undergo decompression.

Clinical examination may reveal a lower motor neuron pattern of quadriceps weakness, paraesthesia or anaesthesia of anterior, medial & lateral thigh and the medial lower leg. A complete lumbar plexus injury features additional weakness of the adductor muscles due to involvement of the obturator nerve. As adductor magnus is partly innervated by the sciatic nerve, paralysis may be incomplete. Caution must be paid to the interpretation of isolated marginal improvements in adductor strength over coming weeks as there may be some intramuscular collateral sprouting from the sciatic nerve innervation, and this does not reflect true recovery [43].

Recovery from neuropraxia is typically complete within 1-2 months, whereas axonotmesis grade injuries may take several months and progress from proximal to distal muscles. The order of expected motor recovery in isolated femoral nerve palsy is: iliacus, sartorius, rectus femoris, vastus lateralis & vastus medialis. As sensory recovery will occur in a concentric fashion through collateral sprouting to an extent, even in case of complete injuries, it is difficult to assess recovery through sensory testing [43]. However, if gradual downward progression of Tinel's sign is observed from proximal to distal thigh over months, nerve recovery is likely. In neurotmesis, no motor recovery is likely [38]; however, the insensate territory would be expected to shrink concentrically to a significant extent, owing to collateral sprouting by the adjacent sensory nerves.

Identifying a new lower motor neuron lesion overlying a pre-existing stroke related deficit is often challenging, particularly if the stroke is recent. Clues to a new lesion may include worsening of transfers & gait, increased tendency to fall, higher dependence on walking aids, ipsilateral thigh wasting and loss of knee jerk with preserved ankle reflex [44].

Diagnostic delays are common, often due to limited awareness amongst clinicians of this clinical entity [31]. Complicating this, the development of neurological signs/symptoms can be remote from the inciting event [19, 30]. Therefore, many late features & complications may be noted at the time of diagnosis and these may be irreversible. These include established contractures, wasting, features of CRPS, neuropathic ulcers, secondary injuries from falls, activity avoidance, and distress related to lack of correct diagnosis & counselling. Cases may be incorrectly labelled as deconditioning or functional neurological deficit. Similarly, evidence of partial recovery may cloud the clinical picture.

Differential diagnosis: Unfortunately, the early impression of shock, anaemia, and pain within the abdominal, groin & thigh regions presents a broad clinical differential and should prompt urgent assessment and resuscitation. The differential diagnoses includes large occult bleeds such as gastrointestinal and ruptured abdominal aortic aneurysm. If abdominal or groin symptoms are present assessment for appendicitis, incarcerated hernia and female pelvic organ pathology should be considered. If neurological features predominate, then stroke, spinal cord and cauda equina injuries should be considered. Lumbar spine radiculopathy and many other

causes of lower motor neuron disease should be considered, including metabolic causes such as diabetes and toxic exposures such as alcohol. Neuro-inflammatory conditions are uncommon differentials to consider. These lower motor neuron pathologies may affect the plexus or peripheral nerves. It is uncommon for psoas abscess to causes significant nerve plexus pathology, most likely related to early presentation with infective features prompting surgical decompression.

Investigations: Laboratory testing may initially unremarkable, with the exception of potential coagulopathy or the effect of anticoagulant medication [40]. Following a significant haemorrhage, haemoglobin and haematocrit are reduced. An acute kidney injury may be observed alongside a rise in creatine kinase and the presence of myoglobinuria heralding rhabdomyolysis. A significant haematoma may cause a rise in inflammatory markers; this should still prompt the clinician to assess for infection.

Abdominal X ray may identify psoas haematoma in some instances; however, it lacks sensitivity and provides little quantitative information. CT scan offers a rapid diagnosis of haematoma, with quantification and localisation of affected muscles. Contrast leakage may be observed during the arterial phase (the spot sign), demonstrating ongoing bleeding, which may prompt consideration of vessel embolization. The limitations of CT scan includes the limited ability to differentiate between solid and fluid components of haematoma, and increased difficulty in differentiating from abscess & neoplasm [45].

MRI scan has improved sensitivity for haematoma diagnosis and is considered gold standard in the diagnosis of iliopsoas haematoma [46]. Furthermore as the lumbar plexus lies close to the coronal plane, appropriate protocols allow visualisation of the lumbar plexus [10]. T2 weighted imaging may demonstrate the mosaic sign of varying signal intensities', reflecting repeated haemorrhage and absorption over time suggestive of chronic expanding haematoma [45, 47]. MRI scanning unfortunately has greater costs associated, is more time consuming and is less appropriate in shocked patients with emergent pathology [46].

The utility of ultrasound scan in diagnosing iliopsoas haematoma has not been fully determined, however there is evidence of its use in providing rapid bedside diagnosis [27, 46]. Sonography can also provide additional information on the expected fluid density and viscosity, and is useful in guiding percutaneous drainage [48].

Compartment syndrome is a potential consequence of iliopsoas haematoma [30] and likely contributes to nerve injury. Measurement of compartment pressure presents diagnostic information and may support a decision to proceed to early decompression. However, measurement of compartment pressure is impractical due to the intra-abdominal nature of the affected muscles, coagulopathy and potentially unstable condition of the patient.

Due to their proximal location, muscles innervated by the lumbar plexus can be challenging to conduct motor nerve conduction studies. Assessment of iliohypogastric, genitofemoral and ilioinguinal nerves is technically difficult. Needle EMG has the greatest utility in evaluating lumbar plexus lesions, and typically will assess muscles innervated by the femoral and obturator nerves, psoas major and the high lumbar paraspinal muscles [49]. The optimal timing of electrophysiological studies following iliopsoas haematoma is not established and depends somewhat on the clinical question; with localisation being possible from the first week. The majority of diagnostic information can be obtained by 4 weeks following injury. Reinnervation can be detected as early as 3 months post injury [38]. Serial EMG may offer some prognostic benefit and identify suitable candidates for salvage surgery options such as neurotisation. If there is absent nerve conduction in the lateral femoral cutaneous nerve and denervation features in adductor longus, this may indicate involvement of the entire lumbar plexus. Gluteus medius, tensor fascia latae and tibialis anterior muscles may appear clinically unremarkable, but show feature of partial denervation on EMG at an early stage, due to involvement of the 4th lumbar ventral ramus (nervus furcalis) within psoas major. Delayed EMG of these muscles may demonstrate polyphasic potentials or high amplitudes indicative of intramuscular collateral sprouting of nerve fibres derived from the L5 ventral ramus within these muscles, appropriating the adjacent paralysed muscle fibres previously supplied by L4. It is useful to consult with a reconstructive surgeon before ordering delayed electrophysiological studies, as donor muscles and nerves may require analysis.

Acute management: If the patient is shocked, the initial priority is resuscitative care, with prompt transfusion and reversal of coagulopathy as indicated clinically. Where there is ongoing haemorrhage, vessel embolization has emerged as a treatment strategy, with the 4th lumbar artery being the culprit vessel in case reports [28, 29]. This will not offer any reversal of an existing haematoma. Following diagnosis of iliopsoas haematoma available treatment strategies include; conservative, surgical decompression or percutaneous drainage. Unfortunately, there is no high-level evidence or guidelines for preferred interventions and patient selection. Patients with pre-existing neurological impairment (such as stroke) that are otherwise suitable candidates for surgery deserve equal consideration, as a new LMN palsy may be profoundly disabling and render a mobile patient immobile.

In the case of recent complete palsy, large haematoma, and progressive neurological impairment despite optimisation of coagulopathy, open decompression of the femoral nerve or lumbar plexus should be considered [5]. This may present the best opportunity to prevent a low grade nerve injury progressing to an irreversible state. Haematoma may cause compression of nerves external to the fascia, such as the femoral nerve in the groove between iliacus and psoas major, or beneath the inguinal ligament. Where compartment syndrome is identified early fasciotomy (within 6 hours) is recommended [50]. Patients should be counselled that there is no method of offering an early validated prognosis of injury to the lumbar plexus, and that many will recover spontaneously over weeks (in the case of neuropraxia) to months (in the case of axonotmesis), and that recovery may be incomplete [38]. As the palsy may develop gradually, there may be value in late decompression in selected patients [21]. However there is little data regarding the use of late fasciotomy in preventing or reversing neurological deficits.

Percutaneous drains of haematoma have emerged as a treatment strategy [51] and has gained popularity to being a less invasive option that remains viable in higher risk patients. There are technical limitations of percutaneous drainage, as once a clot has formed it is not possible to achieve decompression with this strategy [27]. This has proved to be safe in the short term with the main risks being secondary infections, excessive bleeding and worsening neurovascular compromise [48]. There are concerns regarding further injuring the lumbar plexus, with the potential to convert a reversible injury (neuropraxia or axonotmesis) to an irreversible injury (neurotmesis). This could be clinically silent to both clinician and patient; it may occur during insertion as well as removal of drain, as the nerve / plexus elements can get adherent to the suction drain due to its vacuum effect. There is limited exploration of this issue in available literature. A recent systematic review of case reports highlighted worse neurological outcomes from percutaneous drainage, as compared to medical and surgical treatments [5]. Within this review low case numbers, and the lack of meta-analysis preclude drawing clear conclusions regarding intervention efficacy and safety.

Rehabilitation: Patients with lumbar plexus or femoral nerve palsy acquire a significant degree of disability that may include loss of active knee extension, an inability to lock the knee & weight bear, and loss of active hip flexion. Therefore, rehabilitation services should be engaged early. There is a lack of clear rehabilitation protocols. Key elements of rehabilitation include optimisation of patient positioning, adequate analgesia, patient & therapy team education, avoidance & early recognition of secondary complication and consideration of a knee brace to maintain knee extension during stance & walking.

If there is unilateral lumbar plexus palsy with significant weakness of hip flexion, a consideration can be given to encouraging the development of hip flexion contracture of approximately 30° with the aim of facilitating swing phase propulsion of the affected leg. This is similar to the aims of hip arthrodesis in a slight flexion attitude, and requires normal strength and mobility in the contralateral hip joint and lumbar spine. Late rehabilitation stages should consider repeat neurophysiological studies, with referral for consideration of salvage surgery if appropriate [52].

Late salvage: If neurotmesis has become apparent at 6-8 months post injury to the lumbar plexus or femoral nerve with no recovery apparent on repeat EMG, there is little to be gained from further observation. Selected patients with favourable risk profiles should be considered for neurotization procedures. As nerve recovery post-surgery may take several months to reach the intended target, and motor end plates may die by 18 months following denervation, there is likely an optimal window for surgical intervention following neurotmesis grades of injury. There is a variety of potential donor options depending on the degree of injury. In an isolated femoral nerve injury, the anterior branch of obturator nerve may be used. In complete lumbar plexus palsy, superior gluteal nerve, lower ab-

dominal nerves or nerves supplying the hamstring muscles may be potential donors. Late salvage may have powerful clinical effects. In cases of bilateral quadriceps palsy, patients may recover from being wheelchair bound to become ambulatory with walking aides, even with unilateral neurotization procedures [52].

Prognosis: At time of injury, it is difficult to draw clear prognostic conclusions as a significant interval is required for the underlying degree of neurological injury to become apparent. Complicating this is the potential for a slowly progressive lesion. There is a risk of mortality [40], that is likely correlated to the extent of patient comorbidities apparent and extent of haemorrhage. In a systematic review of 174 cases of femoral nerve lesions following iliopsoas haematoma the highest rate of motor and sensory recovery was observed in patients undergoing surgical decompression, though 34% had persistent motor weakness. Worse outcomes were observed in those with percutaneous drains, with 83% experiencing ongoing motor deficit [5].

Even in complete nerve injury, sensory recovery may be observed, usually occurring in a concentric fashion due to collateral sprouting from adjacent intact sensory nerves. Therefore, sensory testing alone is not sufficient in establishing whether recovery of the injured plexus has occurred. Such sprouting does not occur in motor recovery due to fascial barriers and shear forces between adjacent muscles. However, where a muscle is partially paralysed due to multiple innervations arising from differing nerve roots, intramuscular collateral sprouting occurs leading to larger motor units and recovery of the paralysed portion of the muscle [43]. In the case of lumbar plexus palsy, this mechanism may be observed affecting muscles that are supplied by both the lumbar and sacral plexuses; these include: adductor magnus, gluteus medius, gluteus minimus, tensor fascia latae, and tibialis anterior. This may lead to clinical and neurophysiological features of recovery, but is not evidence of true recovery of the injured plexus.

Future Directions: The knowledge base of lumbar plexus lesions associated with iliopsoas haematoma has many unanswered questions around incidence, aetiology, and optimal treatment strategies. The incidence in clinical practice appears to be increasing and is associated with significant mortality and morbidity. Delayed diagnosis is thought to contribute to disability; incorporating appropriate cues in radiology reports regarding neurological effects when a retroperitoneal haematoma is observed may ameliorate this.

There is a need for robust guidelines on diagnosis and management, particularly the questions around optimal invasive strategies and patient selection for procedures. However, due to the relatively infrequent and heterogenous aetiologies and presentations, little reliable data exists. In the absence of prospective data, guidelines would need to be drawn from consensus of clinical expertise.

Conditions that occur with relative infrequency do not lend themselves to prospective trials, and the heterogenous aetiologies converging to clinical presentation of lumbar plexus palsy secondary to iliopsoas haematoma likely means case-control studies would be of limited use. It is suggested that the optimal data capture to track diagnosis, interventions and outcomes would be a disease registry, that over time could yield data for analysis by future investigators.

Imprecise terminology is encountered in clinical practice related to the described clinical entities, such as ilio-psoas, lumbosacral and neuro-radiculo-plexopathy. It is possible these terms contribute to conflation of clinical correlates and blur diagnostic clarity.

Limitations: There is limited scientific literature available to support management in clinical practice beyond case reports and review of these. As such many assumptions, generalisations and extrapolations have been employed in this article outlining authors' perspectives about this complex subject. The authors have drawn on clinical experience and reasoning from first principle of anatomy, physiology and pathology. Clear and definitive guidance on management is beyond the scope of this article. It is hoped future clinical knowledge will enlighten clinicians as to optimal management strategies, case selection for invasive strategies and how early prognostic information can be gathered.

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