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REHABILITATION OF MUSCLE DYSFUNCTION IN HEMOPHILIA

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Summary

Musculoskeletal dysfunction is a common manifestation of haemophilia, and may be associated with imbalances between muscle groups. Evidence emerging from the literature suggests that the rehabilitation of this dysfunction is very relevant for the patient with musculoskeletal problems. Treatment of muscle imbalances may be linked with a reduction in recurrence of symptoms. Further research is needed to establish the relevance of this area in patients with haemophilia but the clinical experience supports the developing work in this field.

Introduction

Haemarthroses and haematomas are common manifestations of haemophilia particularly in the severely affected patient where bleeding can occur spontaneously or follow minimal stresses. Bleeding into the muscles is said to account for between 10% to 30% of all musculoskeletal bleeding episodes (1, 2).

This paper will focus primarily on two areas. Firstly the initial physiotherapy management following common acute musculoskeletal haematomas will be outlined. The second section of the paper will focus on the assessment and principles of treatment for the correction of muscle imbalances which may follow either muscle bleeds or other muscle or joint problems which may occur in haemophilia. The rehabilitation process will be illustrated by two case studies.

Acute Muscle Bleeds

Bleeding can occur in both skeletal or smooth muscles (3-5). This paper will consider only those bleeds which affect the musculoskeletal system. Symptoms can include bruising, pain, swelling and reduced range of movement. Stretching the affected muscle can provoke severe pain (6). For accurate diagnosis, ultrasound is an inexpensive alternative to MRI and CT scans that yields good information about the size and distribution of the haematoma. It also shows whether the haematoma is solid or liquid (2). Common sites of bleeds include iliopsoas, gastrocnemius and the forearm flexor muscles.

The reduction in bleeds due to effective prophylactic programmes (7-9) has enabled many younger patients to be more active and play sport. This has led to an increase in sports-related injuries (10), including muscle haematomas. Mild and moderately affected patients have a reduced risk of spontaneous bleeding and are more likely to take part in more vigorous pursuits (10). Patients need to be aware of the potential for direct blows to cause muscle haematomas, because if left untreated they may be associated with increased morbidity and even pseudotumour (11).

Prior to physiotherapy intervention it is essential that a full assessment is undertaken in order to establish that treatment is indicated, that there are no contraindications or precautions and to determine baseline measurements to monitor the effectiveness of treatment (12). Outcome specific measurements should be used to measure the effectiveness of the interventions (13).

The principal management of acute muscle bleeds is rest until haemostasis has been achieved through adequate dosage of factor replacement. It is essential that replacement therapy is continued until the bleed has resolved. If treatment is discontinued too quickly it may lead to rebleeding and further complications (1,11).

The aims of physiotherapy following an acute muscle bleed include relief of pain, return to maximal function with full range of movement of adjacent joints and maximal strength and normal length of the muscle and prevention of recurrence. Treatment includes supervision of appropriate active movements within pain-free limits and electrotherapy including pulsed shortwave (14) and ultrasound to hasten the resolution of the haematoma. As the bleed resolves active mobilisation including hydrotherapy can be commenced progressing to gentle stretching of tight muscles as pain-free mobility permits. Slow progression is recommended rather than vigorous treatment programmes in order to prevent rebleeding into muscles (15). Insufficient management can lead to long-term disability (16-18).

Iliopsoas Bleeds

Iliopsoas bleeds are relatively common and recovery can be slow. Signs and symptoms include flexion contracture of the hip with possible compensatory lordosis of the lumbar spine and pain in the groin. Pain is usually less severe than haemarthroses due to the greater volumes tolerated in the muscle before compression of painful structures (1). The pain may radiate into the iliac fossa and upper thigh and there may be tenderness in the anterior aspect of the hip. Differential diagnosis is essential to determine whether there are other causes of pain in this region (18-20).

Complications of Iliopsoas bleeds

Complications include femoral nerve palsy which has been reported as occurring in 37% of iliopsoas bleeds (1) with loss of sensation in the anterior thigh, paralysis of the quadriceps muscle and loss of patella reflex. Symptoms and signs can take six months or more to resolve and there may be long term disability due to the results of compression of the femoral nerve. It is therefore important to avoid rebleeding and progress with rehabilitation slowly. Complications of repeated bleeds into the same muscle include pseudotumours, muscle ischaemia and contractures and neurological involvement (1). Recovery from nerve lesions may be delayed in patients with inhibitors and residual sensory loss is common (1, 21).

Rehabilitation of Iliopsoas bleeds

Treatment of iliopsoas bleeds includes adequate factor replacement and bedrest until the flexion contracture starts to resolve. It is important to maintain general mobility and strength of the upper limbs and the unaffected lower limb while on bedrest. Inner range quadriceps exercises of the affected side may also be practised. Active mobilisation, including hydrotherapy and gentle stretching in the pool, avoiding recurrence of pain may begin when the flexion deformity improves to 20-30°. Partial weight bearing with crutches is permitted and patients are progressed to active exercises emphasising extension of hip and knee, i.e., glutei and quadriceps muscles (17) and full weight bearing. All rehabilitation should be carried out under the cover of factor replacement to minimise the risks of rebleeding.

A backslab may be required to stabilise the knee if the quadriceps are weak secondary to femoral nerve compression, a sequelae of the bleed or due to prolonged bedrest or associated knee pathology.

Rehabilitation under factor replacement cover should be continued until full extension of the hip has been achieved and there is good power in quadriceps and glutei muscles.

Gastrocnemius Bleeds

Calf bleeds are relatively common (20). Adequate factor replacement and rehabilitation are essential to prevent long-term disability due to a permanent equinus deformity (18). Management principles are similar to those described earlier. Full rehabilitation of the gastrocnemius muscle to ensure return to normal length is recommended as 10° of dorsiflexion is required for a normal gait pattern. Associated problems of ankle arthropathy may co-exist and it is necessary to establish whether the equinus deformity is due to bony impingement or tight muscles. Anterior osteophytes may provide a bony restriction to range of movement (22).

Other Bleeds

Bleeds into the forearm muscles can cause significant problems as the muscles lie within closed fascial compartments (16) and the increased volume due to bleeding can lead to entrapment neuropathies, vascular insufficiency causing ischaemic necrosis and contractures (18,20,23). Prompt and adequate factor replacement and appropriate rehabilitation are necessary to prevent long-term deformity.

Acute compartment syndrome is considered a surgical emergency requiring immediate decompression (2). Heim et al. (24) published a case report of a child with severe haemophilia who presented with stunted foot growth and a calcaneo-equino varus deformity following the development of a compartment syndrome in the calf which was inadequately treated with replacement therapy.

Muscle Balance Rehabilitation in Haemophilia

Physiotherapy management of neuro-musculoskeletal dysfunction involves the assessment and treatment of the articular system, neural system and muscle system. Each of these systems can be affected in haemophilia, but this section will consider the muscle system only. General principles will be discussed and then related to more specific muscle problems common in haemophilia.

In a patient with haemophilia, muscle imbalance may occur as a direct result of bleeds. Habitual poor posture, predisposition to overuse injury and insufficient flexibility may also result from haemarthroses and haematomas and these may also lead to muscle imbalance. Insufficient attention to this aspect of rehabilitation may lead to recurrence of symptoms (25).

Muscles have three important functions. They have a role in the static control of posture and alignment of joints, a role in the dynamic control and production of movement and they also provide important proprioceptive input into the central nervous system (26). The concept of muscle balance is that muscles provide stability and movement but it is the balance between different muscle groups which ensures that correct joint loading and correct alignment occurs. Incorrect loading of tissue and alignment may lead to the development of tissue pathology (26). Muscles respond to dysfunction in one of two ways, either by becoming overactive and tight or by becoming inhibited and weak. This does not occur in a random fashion but frequently follows common patterns. These patterns were originally described by Janda (27). One example is the shoulder crossed syndrome, in which a patient presents with rounded shoulders and a forward head posture.

Classification of Muscles

Muscles can be classified in a number of different ways. One system is to classify muscles according to their functional characteristics. Considering these characteristics, muscles can be divided into those which have mainly stabilising characteristics and those which have mainly mobilising characteristics (28). Muscles with mainly stabilising characteristics can be subclassified into two groups, local stabilisers and global stabilisers. Muscles with mainly mobilising characteristics may be termed global mobilisers (26). For muscle balance to occur, muscles with primarily stabilising characteristics should demonstrate greater tonic recruitment (i.e., the ability to sustain relatively low force contractions for long periods of time). Muscles with mainly mobilising characteristics should demonstrate greater phasic recruitment (i.e., the ability to generate relatively high forces, albeit briefly).

Local stabilising muscles

Examples of the local stabilising muscles are the deep neck flexors, multifidus and transversus abdominis (TA) which are all concerned with stability of the trunk (29). These muscles tend to be small, deep or part of a larger muscle. Although TA is not small it is the deepest abdominal muscle. The local stabilisers are usually associated with passive joint structures such as joint capsules or ligaments. They do not usually have any significant torque producing function but tend to have a greater endurance capacity with tonic activation during joint movements. Recent research has suggested that these muscles tend to be activated prior to movement occurring (30-32) highlighting that their role is to stabilise the trunk and provide a stable base from which appendicular movement can occur.

The main roles of these deep local muscles are joint protection and support, control of the ideal alignment of the spine and providing an important proprioceptive function about where the trunk and body are in space. If there is pain for whatever reason, these muscles will become inhibited. There will be selective weakness, a decrease in force production and a decrease in tonic holding capacity or endurance capacity of the muscle. It is possible that the lordotic posture resulting from an iliopsoas bleed may lead to TA inhibition as this posture is associated with tight hip flexors and weak abdominals. If untreated this could lead to insufficient stability within the trunk and subsequently other symptoms such as low back pain.

There is increasing evidence that TA and multifidus have key roles as stabilising muscles of the trunk (32-34). TA contracts in all movements of the trunk regardless of the primary direction of movement and is recruited first in sudden movements of the trunk (30,31). It has been demonstrated that TA activation is delayed in low back pain patients compared to normals during arm movements. This delayed onset may indicate a deficit in motor control which results in inefficient stabilisation of the spine (32). There is also evidence that multifidus was segmentally inhibited within 24 hours in patients with first episode acute low back pain (LBP) and that recovery of multifidus was not spontaneous when LBP had resolved (33,34).

The clinical implication of this evidence is that the patient with haemophilia with musculoskeletal pathology affecting the spine or peripheral joints may have poor trunk control. Therefore assessment of stability in the trunk is important and where necessary, treatment must be considered.

In the cervical spine segmental stability is provided by the deep neck flexors (DNF) particularly in mid range positions. The DNF muscles demonstrate predominately tonic activity whereas sternomastoid primarily functions in torque production. Patients with neck pain or headache demonstrate weakness of DNF muscles and a forward head posture (35,36). Dysfunction improves with retraining and is associated with a reduction in symptoms (37). A progressive series of low load cranio-cervical flexion exercises may be used to retrain the DNF muscles using increments of increasing load and time as muscle function improves (38).

Global stabilising muscles

Global stabilisers such as lower and mid-trapezius and gluteal muscles are not only stabilisers but also produce torque. They tend to produce movement in one plane only, have an important role in controlling antigravity positions and are primarily involved in slow controlled eccentric movements and deceleration of joint movements. Dysfunction in these muscles due to pain leads to inhibition of the muscle with delayed activation. These muscles are lengthened and test weak in inner range positions. The activation threshold of these muscles increases so it becomes more difficult to recruit tonic fibres. However more phasic fibres, which fatigue quickly, are recruited.

Changes in muscle function have been identified in the activation patterns in the gluteal muscles following a sprained ankle (39). Fourteen patients with a sprained ankle and five controls performed hip extension in prone lying whilst electromyography surface electrodes were placed on the lumbar spine extensors, the gluteal muscles and the hamstrings. The controls demonstrated a high degree of repeatability and an almost simultaneous pattern of activation of the tested muscles. The group who had a sprained ankle demonstrated that the pattern was variable between subjects and there was more likely to be a delay in the gluteal muscle recruitment. Interestingly this occurred on both the affected and unaffected sides suggesting that there was a delay in motor control (39). It could be hypothesised that a similar abnormal pattern of muscle activity could occur in a patient with haemophilia with ankle arthropathy.

Patients who have experienced bleeds around the shoulder may present with pain and anterior translation of the humeral head, often associated with poor scapular control. Rehabilitation programmes to regain appropriate activation of the rotator cuff (40) and scapular stability (41) are important considerations in these patients.

Global mobilising muscles

Gastrocnemius and hamstrings are examples of global mobilisers. These muscles are main torque producers, they tend to be more superficial and not linked directly to joints. They usually have fusiform, long fibres which are required for their function for increased load and speed, i.e., when greater muscular forces are needed. These muscles may have a tertiary stabilising role. Pain due to dysfunction in the musculoskeletal system may cause these muscles to go into spasm (26). For example, in the presence of a bleed within the muscle, the muscle will be held in a shortened position. Also in the presence of sensitised neural tissue or inappropriate movement patterns, these muscles may become overactive and develop an increased stabilising role not being provided by the stability muscles. The overactive muscles are inappropriately recruited and their low threshold activity predominates so more tonic fibres are recruited which have an increased resistance to fatigue and these muscles assume an antigravity postural function. As the muscles become overactive, short and dominant this can have an effect upon joint alignment with resultant pathology (26).

Assessment and Principles of Treatment of Muscle Imbalance

The difference in functional characteristics of the stabilising and mobilising muscles necessitates different methods of assessment and treatment. The stability muscles are assessed by their ability to activate and hold in inner range (25,42) while muscle length tests (43) are more appropriate for determining dysfunction in mobilising muscles.

A progressive programme of muscle rehabilitation has been described by Comerford and Mottram (26). This includes:

1. Control of the stability muscle in neutral positions
2. Regaining dynamic control in the direction of symptom-producing movements
3. Rehabilitation of the global stabilising muscles through range
4. Lengthening of global mobilisers
5. Integration into normal function

The initial stages of the programme focus on low load, low effort and isolated activation of muscles that can be ideal for patients with marked joint pathology.

When treating the local stabilising muscles it is important to focus on the appropriate activation of the affected muscle in isolation from the mobilising muscles. Facilitation strategies such as tactile facilitation or working the muscle with another stability muscle may be used (44). It is important to activate these muscles in the absence of pain otherwise the muscle will remain inhibited. Once activated, it is necessary to focus on the endurance capacity by increasing the holding time and avoiding substitution from other muscles. It is important not to work to fatigue because once again these muscles will become inhibited. Once the correct activation pattern has been established the exercises should be repeated frequently by being incorporated into functional activities. It may not be appropriate to progress to high load activities when rehabilitating patients who have haemophilia (45). Fast ballistic exercises are not indicated because they can inhibit stability muscles (46).

The classic muscle test positions of inner range holds are used for the global stabilisers again increasing the holding time (43). It is important to avoid fatigue. Once activated, exercises should be incorporated into functional activities and repeated frequently.

When imbalances are present between muscle groups the stability muscles are activated prior to lengthening the overactive short muscles. If the mobilising muscles are just hypertonic they will relax by reciprocal inhibition. If they are adaptively shortened, then it is necessary to add lengthening techniques for the tight muscles. The trunk, cervical spine, scapula and pelvis should provide a stable base from which functional movement can occur. Clinically poor stability of the trunk is often associated with tightness of more peripheral muscles such as hamstrings or gastrocnemius as the body strives to achieve stability.

Neural control of movement

Alterations in proprioception have been identified in low back pain patients and whiplash patients (47-49). Neck muscles contain many proprioceptors and these muscles contribute to balance and postural control (25). Jull (25) suggests that there is likely to be a link between the joint stability role of deep local muscle system and neck kinaesthesia. If these muscles are not functioning optimally, there may be a dysfunction in proprioceptive afferent input. Further research is needed to increase the knowledge in this area and support or refute the clinical findings.

Case History 1

A 46-year-old patient with factor IX deficiency (< 1%) presented with left anterior knee pain and a feeling of weakness of the joint, but no giving way. Symptoms had started 6 months ago for no apparent reason, but were now worsening. Pain was aggravated by getting in or out of a car, standing from sitting and going up and down stairs. He had had no recent bleeds into the left knee but had had occasional bleeds in the past.

Previous medical history included an arthrodesis of the right knee when he was 26. He complained of intermittent bleeds into both ankles.

Examination

On examination the right leg was 2.5 cm shorter than the left and he tended to compensate for this by standing with his left leg abducted. He had a ROM 20-120^{o} of knee flexion. Passive knee flexion movement was associated with patellofemoral crepitus and pain. A small effusion was present. The quadriceps muscle was weak, especially vastus medialis obliquus (VMO), and the iliotibial band was tight. There was weakness of the stabilising muscles of the trunk and pelvis. Assessment of the patellar position (50) identified that it was sitting more laterally than normal and was tilted laterally.

A plain A-P radiograph showed minor degenerative changes of the tibiofemoral joint.

The clinical impression was that this was a left patellofemoral dysfunction secondary to the fixed flexion deformity and tight lateral structures.

Management

All the physiotherapy was undertaken under factor replacement cover.

Management consisted of passive mobilisation of the tibiofemoral joint to increase range of extension. The patella was passively mobilised to lengthen the tight lateral structures. Tape was applied to provide a sustained stretch to the tight lateral structures as well as facilitate contraction of VMO.

The stabilising muscles, VMO and the posterior fibres of gluteus medius were re-educated by focusing on sustained low intensity contractions. Trunk stabilisation work to improve the function of the deep abdominal muscles was included. The normal positions used for these exercises (e.g., four-point kneeling) were modified because of this patient's arthrodesis.

The patient had 12 sessions of physiotherapy and made good progress with significant relief of pain. There was no sensation of weakness although intermittent swelling of the knee did persist following prolonged weight-bearing activities. ROM improved to 10-120^{o} of flexion with a bony end feel on extension.

Summary

The anterior knee pain may have developed due to overuse of the left knee as a result of the longstanding arthrodesis on the right side. The shortness of the right leg was compensated by abducting the left leg and flexing the knee. The patient had adapted to this position and did not wish to consider a heel raise. The stabilising muscles were weak and so were re-educated along with lengthening of the tight structures. The patient was discharged with advice on a home programme.

Case History 2

A 19-year-old male student with factor IX deficiency (2-5%) presented with right hip pain which radiated intermittently to his groin and upper thigh. Pain was aggravated by standing, kicking a football and walking especially on an incline. He also complained of a feeling of 'giving way', a general feeling of leg weakness and intermittent low back pain. Symptoms had been present for over a year but had been worsening over the last three months with an associated loss of function. He had a tendency to overdo activities and found the increase in pain was a frustration. He had experienced no recent bleeds into the right hip.

Previous history included Perthes disease of the right hip that had resulted in surgery. He had had recurrent right hip and iliopsoas bleeds since removal of the metalwork 18 months ago. Other joint bleeds were rare until recently when there had been an increase in the frequency of bleeds in his knees and right elbow.

Examination

On examination the right leg was 2 cm shorter than the left. Right hip movements were limited in all ranges, particularly extension and medial rotation. Weight bearing was reduced on the right side, and the right hip was held in external rotation. Gluteal and quadriceps wasting and weakness were present, and there was tightness in the iliotibial band, hamstrings and iliopsoas. Knee movements were full. The patient had an increased lordosis and scoliosis at the thoracolumbar junction and limited lumbar flexion. There was poor trunk stability and no neurological deficit. X-rays of the right hip demonstrated moderate degenerative changes.

The clinical impression was that there was right hip dysfunction and pain due to the underlying pathology, and this was associated with trunk and muscle imbalances.

Management

Treatment included passive mobilisation to the right hip joint to increase extension and medial rotation. Trunk stability training was undertaken focussing on TA and multifidus. A progressive exercise programme to strengthen gluteal and quadriceps muscles included closed kinetic chain exercises to aid tonic recruitment and improve proprioceptive afferent input. Exercises to lengthen iliopsoas, hamstring and iliotibial band was added and good posture re-educated. The patient was also given a home exercise programme.

The patient received 10 sessions of physiotherapy. The main outcomes were a marked relief of hip pain, cessation of back pain, improved range of movement at the hip and lumbar spine, increase in muscle strength, improvement in posture (as evidenced by reduction in lumbar lordosis and scoliosis) and no sensation of hip weakness or giving way. The patient was able to walk and stand for longer periods and he reported a reduction in bleeds.

Summary

This patient's hip pain was due to underlying hip pathology, exacerbated by associated muscle imbalances in the trunk and hip region. Re-education of trunk stability was undertaken prior to the progressive strengthening and exercises to lengthen affected muscles. This was to ensure that the trunk was sufficiently stable to allow the subsequent exercises to be performed safely and effectively. Accordingly, the patient's symptoms were not exacerbated during the treatment programme.

Conclusion

Musculoskeletal dysfunction is a common manifestation of severe haemophilia, typically resulting from haemarthroses and muscle haematomas. Common sites of spontaneous muscle bleeds include iliopsoas, gastrocnemius and forearm flexors. The advent of prophylactic replacement therapy has enabled even severely affected patients to participate in sport and therefore all patients may sustain sports-related injuries, including direct injuries to muscle as well as spontaneous bleeds. Patients need to be aware of the significance of such injuries and seek prompt treatment.

Habitual poor posture, repetitive activities or insufficient rehabilitation may lead to imbalance between different muscle groups. Appropriate management is essential to prevent long-term disability. The principles of rehabilitation following acute muscle bleeds have been outlined, together with general principles for the management of muscle imbalances. These are important considerations in the management of patients with haemophilia.

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