Diabetic Gait Dysfunction: A Literature Review of Underlying Mechanisms and Changes Seen Involving the Diabetic Gait Cycle

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Abstract: The diabetic gait cycle and gait efficiency are negatively affected and at a seemingly early stage of the disease process and worsens as the disease progresses. The aim of this paper is to explore some of the differences in the diabetic gait, gait function, and the likely causes. The effect that diabetes has on the gait cycle is multifactorial and involves a significant cascade of events leading to acceleration of a pathologic process in glycosylation. Glycosylation causes widespread problems in the diabetic individual from neuropathic involvement, microvascular destruction, and dysfunctional and weakened soft tissue structures. The net effect on the diabetic a slower, more rigid gait with loss of muscle strength and changes in expected ROM through the gait cycle.

Introduction:

Diabetes mellitus includes a large scope of complications on microvascular, macrovascular, and neurological. While the different types of diabetes are physiologically different rather based on insulin resistance in type 2 or lack of endogenous insulin secretion in type 1, the common theme of battling to control hyperglycemia is shared [1,2]. In the US, the CDC estimates that more than 30million people in the US have diabetes with an alarmingly increasing rate. The increase is blamed on the concurrent raise in obesity and the prevalence of early age and childhood obesity. [1]

Many diabetics are aware of the complications that their disease can have on their heart, kidneys, eyes, and nerves. [2,3] However, the effects diabetes can have directly on the patient's soft tissue structures and their biomechanics may not be as well-

known leaving patient's unsure as to what symptoms may present.

Some of the effects to soft tissue and skin are a function of aging such as atrophy of skin, muscle and fat pads, reduced healing rates of wounds, and longer recovery from minor sprains and strains, to name a few [4]. In diabetics one of the normal ageing processes, glycosylation, is increased. The worse the patient controls their hyperglycemia the faster this process acts. Glycosylation can affect many proteins in the body and lead to a laundry list of the complications seen in diabetes.

Tendons and ligaments that are affected in the diabetic patient are less elastic, more prone to injury, have increased healing times, and are overall biomechanically dysfunctional compared to healthy tendon [3,4,5]. The gait of a diabetic can therefore be significantly altered compared to a hypothetical normal gait. Glycosylation, neuropathy, and decreased blood flow all play a role in decreasing the effectiveness of the diabetic foot and ankle and increasing patient's risk of morbidity due to acute injury or increased plantar pressures causing wounds.[6] The purpose of this paper look at the literature for a better understanding of how diabetes negatively effects our patient's through their gait and soft tissue structures of the foot and ankle.

The importance of the change in diabetic gait and biomechanics of tendons is significant due to their prevalence in increase rates of falls and ulcerations. As the gait in a diabetic becomes less reliable we start to see an increase in fall rates. Likewise, as the gait and biomechanics become less efficient we see a subsequent increase in plantar pressures leading to increase in damage to plantar skin

and ultimately an increase in ulceration risk [3,6].

Gait Cycle

The gait cycle in a healthy individual with ideal biomechanics is seen to be compromised of 60% of the cycle in stance phase and 40% as swing phase [7]. Breaking that down farther we see that each is composed to smaller motions. Stance phase of gait is composed of initial contact/heel strike, loading response/foot flat, mid-stance, and terminal stance/heel off. Swing phase of gait is composed of pre swing/toe off, initial swing, mid swing, and late swing [8].
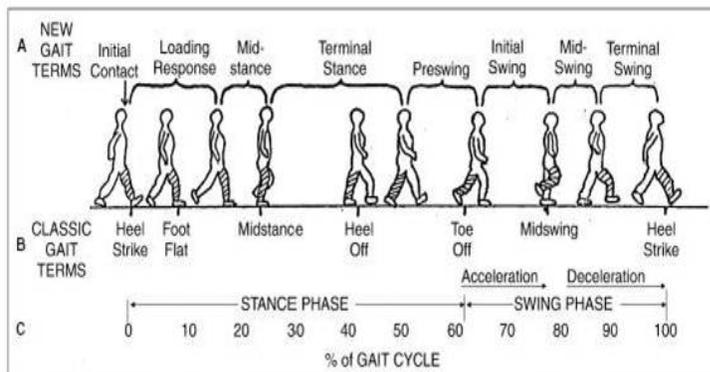

In the ideal gait, we see that during stance phase of gait we go from the initial heel touch after finishing a swing phase, roll through the mid foot from central lateral to central medial. The forefoot is the last to be loaded with continued increase in vertical ground reactive forces below the metatarsals until the end of stance phase and subsequent toe off to start another cycle of swing phase. The Swing phase of gait is characterized by starting immediately after toe offloading to anterior swing of the leg in relation to the motion of the body and continues until toe-off of the contralateral foot terminating in ipsilateral heel contact[7,8].

While the above may hold true for the healthy individual, multiple studies have looked at the differences noted in diabetic gait cycles. Wei-chun et al in 2015 found that diabetic patients had a noticeable decrease in knee flexion and hip adduction. They noticed that diabetics routinely have decreased strength and range of motion in the plantar flexor motion moment of the ankle [9]. The decrease in PF leads to an increased compensation demand at the knee and thigh musculature to make sure the toe clears the ground. The increase in loading time and strain across the knee reduces the knee ROM, increases total amount of time the foot is in contact with the floor, and indirectly decreases the ROM of the hip [6,9] These findings were consistent to what Swung-uk Ko and colleagues found in an article published in 2010 [13]. They noted that diabetics had increased time in knee flexion leading to a decrease in total stride length and increased stride duration compared to healthy individuals. Kinematically, they showed that diabetics had a decreased amount of plantar flexor power on average (238j/kg in diabetics v 277j/kg in healthy cohort). Also found was that the knee subsequently had to absorb more impact to make up for the global loss of plantar flexor strength at the ankle as the loss of PF decreased the ankle shock absorption. The knee on average in the diabetic absorbed 332j/kg with each step compared to the 283j/kg absorbed in another-wise healthy individua[10,13].

Further studies have broken down the motions of the gait cycle to the joints involved to further show the dysfunction seen in a diabetic gait. Deschamps et al. used 3D segmental foot mobility tests on a cohort of 26 diabetic patients compared to 13 healthy individuals. They showed that diabetic individuals had decreased ROM at the ankle, STJ, and 1st metatarsal compared to the healthy cohort leading to significantly less efficient global foot loading. A longer foot on floor period was observed due to this lack of ROM and loss of power in the posterior leg compartment. It has been shown in their study that the loss of ROM seen in the ankle and STJ in DM is also correlated to a significant dysfunction in the windlass mechanism activation and deactivation through the gait cycle [5,8]. A major significance of this is the foot stays in a more rigid construct through stance phase leading to markedly increased plantar pressures under the heel and forefoot. This increase in pressure combined with the loss of ROM in the transverse planes of the midfoot and transverse planes leads to increased shear strain on the plantar soft tissue structures increasing the risk of breakdown and ulceration [2,3,5,8]
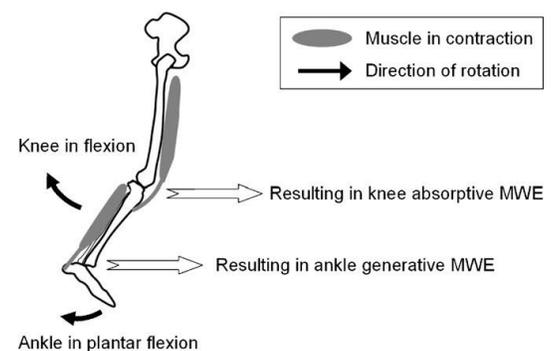
Gait Dysfunction

Diabetics have been shown to have decreased efficiency in the overall gait cycle compared to non-diabetic persons independent of age, body weight, height and sex [5,8,10]. Overall it has been shown that diabetics routinely have a shorter stride length, decreased velocity, increased variability in get speed, and increased variability in tandem positions (the repeated relationship of

heel to toe) during gait, and decreased postural stability [11,12]. The decrease in stride length and velocity is a function of much of the loss of strength and ROM of motion in the foot and ankle and the compensation measures. The variability in speed and tandem positions is much more multifactorial due to multiple deficits diabetics can have. Motor neuropathy leads to increased variability due to the inability to maintain significant motor tone throughout multiple cycles of gait as well as considerable impairment of intrinsic muscles of the foot[13]. Sensory neuropathy decreases the ability of the diabetic patient to adequately and reliably sense the ground and react with a consistent reflex. Loss of soft tissue elasticity and joint ROM increases amount of muscle force that must be recruited leading to irregular muscle loading that varies based on center of gravity. Added to that the diabetic population has a decreased postural stability due to considerable lower extremity variability and having to compensate for a constantly changing center of gravity [2,9,12].

Glycosylation

Glycosylation is a process that is heavily implicated in the literature for the onset of symptoms and dysfunction in the diabetic individual [5,14]. It's a process that takes glucose and cross links it to nitrogen groups found on proteins and alters their function and ultimately leads to the cell with the affected proteins demise. It is noted to be a factor in causing retinopathy, neuropathy, and tendinopathy as well as many other diabetic related complications[2,3,14].

In hyperglycemic states, we see at the cell level there is much more glucose available than is needed to keep the cell functioning and with an increased concentration outside

of the cell the glucose sequestered in the cell [3]. The polyol pathway becomes activated in this situation upregulating the local production of aldolase reductase. The polyol pathway is a storage pathway that turns glucose into a longer, less metabolic sugar alcohol called sorbitol. As sorbitol increases the concentration gradient greatly increases with it . sorbitol cannot escape the cell membrane and therefore acts as a chronic source of increased oxidative stress. If the hyperglycemic state subsides the cell can quickly reduce the amount of sorbitol back to glucose and transport the glucose out getting the cell back to equilibrium[2]. Additionally, as the hyperglycemia persists the cell utilizes resources necessary for normal cell function such as NADPH to produce sorbitol. The cell gets locked in to a cycle of increased oxidative and osmotic stress. The cell cannot perform its normal functions and is faced with a growing influx of water following the concentration gradient. In time either the cell will stop being able to support its normal function and die or the osmotic pressure will cause the cell to undergo hemolysis [3,14]

Insulin independent cells such as retina, urogenital/kidney, nervous tissue especially Schwann cells, and cells with very slow turnover are the most affected [2,3]. Most famously noted for glycosylation is hemoglobin proteins of red blood cells. The process that damages the eyes, kidneys, nerves, tendons, ligaments, and other tissues of the body is also the process that gives us the HbA1c test. HbA1c is formed in the presence of hyperglycemia leading to binding of glucose covalently to the N-terminal of hemoglobin proteins. Due to the lifespan of the average blood cell being roughly 90-120 days the HbA1c is the average percentage of hemoglobin bound to glucose circulating in

the blood that is a linear relationship with the average blood glucose of the past 90-120 days[2].

Hyperglycemia leading to increased glycosylation is the driving force in the dysfunction seen in the gait efficiency and change in gait mechanics seen previously. Glycosylation affects the tendons, ligaments, skin, nerves, and microvasculature of the foot and ankle leading to the significant dysfunction and loss of efficiency seen in DM gait[4,5,15].

Effect on Soft Tissues

While the effect of Diabetes on the individual's gait is largely multifactorial; one of the most profound and possibly underestimated aspects is the loss of tendon, ligament and other soft tissue function due to advanced glycolytic end products[2,5]. Any soft tissues in the body can be negatively affected by glycosylation but in the foot and ankle we see the most noticeable deficits occurring at the Achilles tendon, plantar fasciitis, and plantar skin.

The Achilles tendon is very well studied in both healthy and diabetic individuals. We know that on an average adult Achilles tendon is about 15cm in length from musculo-tendinous junction to insertion and a thickness of 4.8 to 10mm. The collagen in normal, healthy tendon consists of very well organized collagen with a parallel arrangement throughout its entirety. In the diabetic individual, the overall length and thickness remain the same, but a much higher proportion have evidence of disorganized collagen fibers appreciable on ultrasound[16]. Many human and animal studies have looked at the effect of hyperglycemia and glycosylation on the degradation and dysfunction of the Achilles tendon, most of

which can be extrapolated to other soft tissue structures. One of the assumed pathways is caused by the advanced glycation end products integrated and deposited in the soft tissues as a byproduct of glycosylation[16,17]. This pathologic process leads to cross-links in collagen leading to the more disorganized appearance. Also, hypothesized, the increase in AGEs and hyperglycemia leads to an increase in matrix metalloproteinases which are known to down regulate collagen formation. It has been shown that tendons, especially the Achilles, are prone to decrease in elasticity, reduced tensile strength, and increase in thickness in the presence of diabetes and even more so in advance, uncontrolled disease. The structural dysfunction that the tendon incurs is translated to the mechanical aspect showing a decrease in tendon contraction and contraction strength which can cause a domino effect on the diabetic gait[9,13,16].

D'ambrogi et al showed that as the diabetic disease progresses there is a significant effect on the Achilles tendon and plantar fascia that correlates to a significant change in the gait pattern and efficiency[5]. As the Achilles starts to have its collagen arrangement disrupted it becomes functionally more rigid. The plantar flexor strength of the gastro-soleus complex is reduced due to the loss of contractibility of the Achilles tendon decreasing the amount of plantar flexion ROM obtained at the ankle joint. A net loss of plantar flexion requires the muscles of the leg to have to compensate leading to a decrease in gait velocity and increase in gait variance[11,13,18]. Similarly, the plantar fasciitis can become thickened and functionally rigid just as the Achilles does. The loss of the elasticity of the plantar fascia leads to a decrease in the ability of the foot to adapt to

changes in terrain as well as causing dysfunction in the ability to activate and deactivate the windlass mechanism. Individuals with decreased Achilles function and decreased plantar fascia elasticity have a compensatory early onset windlass mechanism [5]. The windlass mechanism is then maintained throughout most of the stance cycle causing the increase in forefoot pressures with a significant increase seen going in to the toe off motion[5,13,19].

As the plantar pressures increase due to the gait dysfunction and muscle imbalances caused by neuropathy and tendon glycosylation we see a significant increase in risk to ulcerating and falls in the diabetic patient [6,11,13,19]. Plantar pressures increased at heel strike and toe off subject the plantar skin to increased friction and shear forces. The skin is effected like the tendinous structures- loss of elasticity and tensile strength due to AGEs and cross linking as well as the impairment of collagen synthesis[3,13]. The diabetic foot is put in a position where it is undergoing significant stress with a decreased ability to repair micro damage prior to frank ulceration. Couple the significant effect diabetes has on gait function and mechanics with other complications of diabetes and it becomes easier to see how the dysfunctional gait increases risk for ulceration, falls, and in extreme cases Charcot neuroarthropathy.
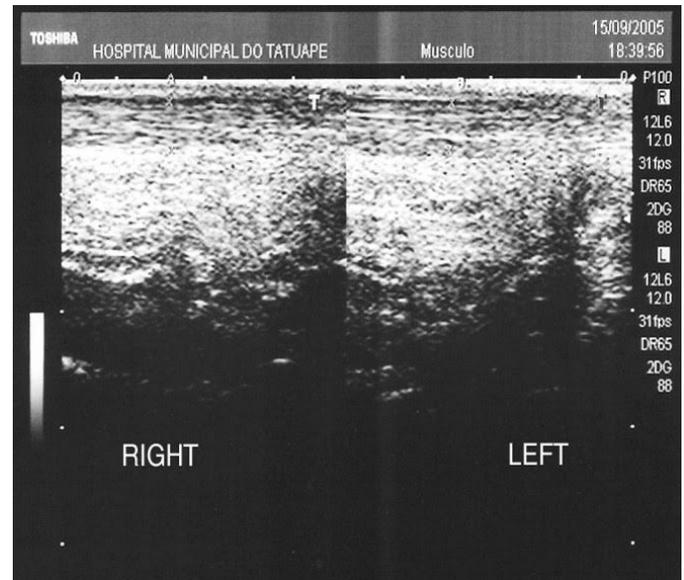
Ultrasound of Achilles tendon of healthy individual. Demonstrates parallel, organized nature of tendon[16]
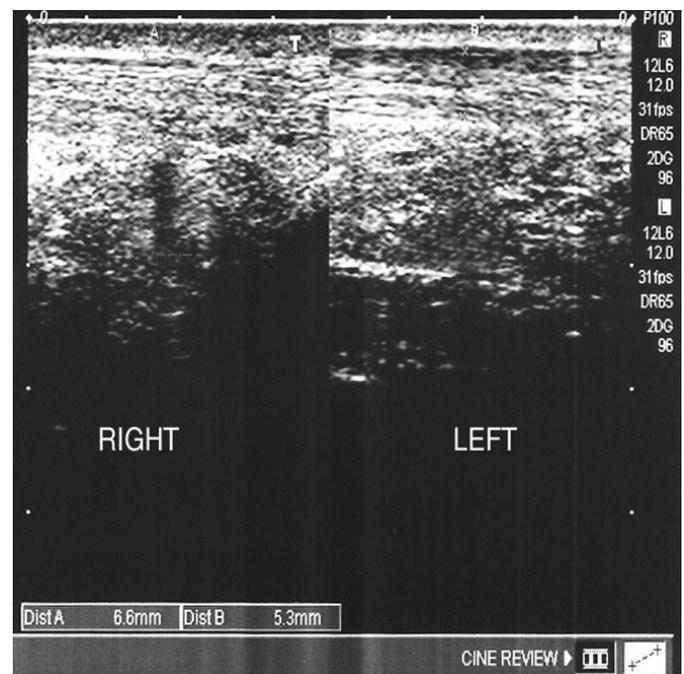
Ultrasound of Achilles tendon in Diabetic. Demonstrates increase disorganization and loss of parallel nature[16]

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