CHAPTER **11**

ACUTE EXERTIONAL COMPARTMENT SYNDROME

David C. Alder, DPM
Robert B. Weinstein, DPM

Exertional compartment syndrome, especially the acute form is a serious injury that needs to be recognized and treated early to prevent permanent disability.

With proper history taking, physical exam and testing, the physician will be able to differentiate this injury from common muscle strains, stress fractures, cellulitis or other joint injuries and treat it in an effective and appropriate manor.

ETIOLOGY

Compartment syndrome arises when the pressure within a muscle fascial compartment increases to the point that it decreases the capillary perfusion. When the pressure increases enough, the perfusion level decreases below the level needed to keep the muscle tissue viable.[1] This leads to tissue necrosis and eventually can include join contracture, neuromuscular dysfunction, drop foot and chronic pain.[2]

The pressure needed for compartment syndrome can come from trauma such as burns, fractures, or crush injuries. However compartment syndrome can also come from over use of a muscle compartment that goes beyond strain and elevates the intra-compartmental pressure to the point of tissue necrosis.

Although the end result can be the same, the main difference between trauma-related compartment syndrome and exertional compartment syndrome is the time it takes to occur. Compartment syndrome can occur rather quickly after a serious burn or a fracture, where as exertional compartment syndrome symptoms can be delayed. In chronic exertional compartment syndrome the symptoms will appear after exercise and will usually resolve after adequate rest. In acute exertional compartment syndrome the symptoms can be severe and they do not decrease with rest. This by no means belittles the importance or seriousness of the consequences that can occur if the disease process is left untreated. Acute exertional compartment syndrome is a medical emergency and needs to be treated immediately. Muscle compartment pressures are about 0-8 mmHg at rest.[3] Pathophysiologic changes start to occur when the pressure in the compartment exceeds 30 to 40 mmHg.[1]

DIAGNOSIS

Acute exertional compartment syndrome has been described in all of the muscle compartments of the lower leg.[3] The compartment syndrome can be delayed several hours after the onset of pain.[3] The activities are usually repetetative in nature such as running or weight lifting. The initial symptom is pain. The pain will often be described as a pressure type of pain that is out of proportion to the activity just performed or the examination just given. The traditional symptoms of compartment syndrome are the 6 P's which include pain, pressure, paresthesia, paresis, pain with passive stretch and pulses present.[2] In the early stages the skin can feel supple but will often feel taught as the process advances. The skin will also have a shiny appearance. Sensory deficit may be present depending on the severity and the particular compartment that is involved.

Pulses will most likely be palpable since the pressures needed for initial muscle necrosis is less then that needed to stop arterial flow.

Once the suspicion of compartment syndrome is made and other diagnoses ruled out, based on the history and the physical exam, the compartment pressures should be checked. The pressures should be checked with one of several types of needle catheter measuring devices.[3] One effective method to measure compartment pressures is to use the compartment pressure monitor described by Whiteside. The advantage to this device is that it uses materials easily found in a hospital setting and does not rely on knowing where the "device" has gone and no one can find it syndrome when you need it.

Below are the instructions on how to construct and use the Whiteside device.[4]

CONSTRUCTION AND USE OF WHITESIDE'S COMPARTMENT SYNDROME MONITOR

The materials needed are listed in Table 1. The basic concept behind the apparatus is to assess an intra-compartmental pressure. Pressure is detected in the compartment by way of a stick catheter (spinal needle) attached to a pressure delivery source (20 ml syringe filled with air) and pressure monitor (modified Sphygmomanometer). These 3 devices are all connected to a 3-way stopcock which is adjusted to direct pressure (negative or positive) according to need. The actual intra-compartmental pressure is measured as the point at which the pressure can be overcome externally.

The indicator in this apparatus is a saline column trailed by an identifiable meniscus. The column sits inside the IV line with the catheter at the end. The catheter is inserted into the compartment and the saline column sits against the pressurized compartment. While air pressure from depression of the syringe plunger builds behind the saline column, the pressure monitor sits at the other end of the apparatus detecting the increase in column pressure. The saline column (meniscus) is static so long as the intra-compartmental pressure is higher than the pressure behind the column. Once the compartment pressure is overcome, the meniscus will move in the direction of the catheter as the saline is pushed into the compartment. This point is the intra-compartmental pressure, and the pressure is read on the monitor.

The apparatus is assembled as follows. (Figure 1) An empty sterile 20 ml syringe is attached to the 3-way stopcock in the center position. The IV line is Luer-locked to one end of the stopcock and a 25g spinal needle is attached to the end of this line. The rubber tubing input on the sphygmomanometer is attached to the standard bore side of an Interlink® T-connector. The Luer-lock end of the connector is then connected to the stopcock. If the sphygmomanometer has a Luer-lock connector on the pressure input then the Interlink® T-connector is not necessary, and Luer-lock tubing is attached directly between the pressure monitor and the 3-way stopcock.

A sealed 10ml bottle of 0.9% bacteriostatic normal saline is punctured with an 18g needle. The needle is left in the rubber seal and serves to normalize the pressure within the bottle. The spinal needle is inserted into the saline bottle alongside the 18g needle all the way to the bottom. While holding the bottle upright saline will be withdrawn up into the IV tubing. First, the 3 way stopcock is set so the line to the sphygmomanometer is OFF while the syringe and catheter side are ON. This will prevent negative pressure from building on the pressure monitor side of the assembly. The plunger is then pulled to begin drawing up the saline column. If this is done correctly, a meniscus will appear with no air bubbles in the catheter side of the line. False readings can occur when there are air bubbles in the line due to the compressibility of saline and presence of multiple menisci.

Once the saline column is established, the spinal needle is removed from the saline bottle and the needle is inserted into the myofascial compartment. The 3-way stopcock is then switched so that all three lines (syringe, catheter, and sphygmomanometer) are open. Gentle pressure is then applied to the plunger and the monitor will show pressure increasing in the column. The meniscus will initially be static. Once the meniscus moves, the pressure reading is taken and this is the intra-compartmental pressure.

If additional measurements are to be taken, the line is partially evacuated of saline while the spinal needle is again submerged in the bottle. This will prevent air bubbles from entering the line. Additional saline is once again withdrawn and measurements taken.

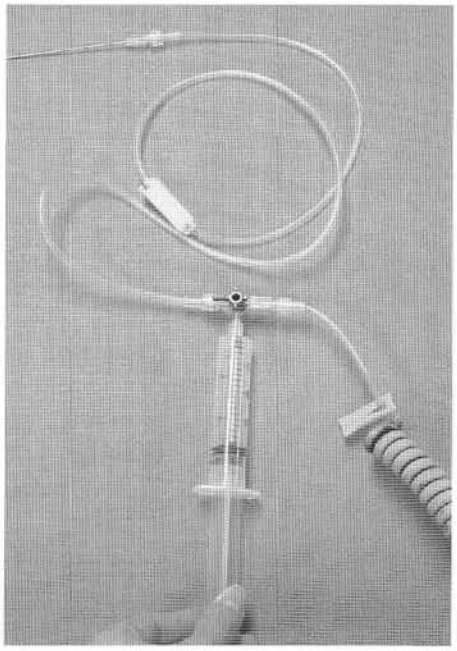

Figure 1. Whiteside's compartment pressure monitor.

TREATMENT

The treatment for acute compartment syndrome is open fasciotomy. The sooner this done the better the better the outcome will be. No precise standard exists to determine the pressure at which surgery should be performed. The decision needs to be based on an elevated compartmental pressure usually above 30 to 40mmHg, signs and symptoms and clinical judgment.

In the lower leg all 4 compartments should be checked and opened if needed. The deep fascia should be opened the entire length of the compartment to avoid residual pressure in the closed area. Care should be taken to preserve neurovascular structures during and after the surgical procedure. The anterior and lateral compartment can be easily approached from the anterior later side of the leg. Both compartments can be opened with a single skin incision. When the posterior compartments are involved a medial approach allows access with a single skin incision.

Necrotic muscle tissue should be debrided but it may be best to minimize this during the fasciotomy. After the procedure wet dressings need to be applied to prevent desiccation of the muscle tissue. Steri strips can also be carefully applied during later dressing changes to keep the skin covering the neurovascular structures, however the wounds need to be open enough to relieve the pressure. The careful and gradual use of steri strips on the wound edges will also help to prevent contracture of the skin so that closure will be easier. The postoperative dressing should be placed on so that no external pressures are being applied. Antibiotics should be given to prevent infection and cultures should be taken at the time of surgery especially if significant amounts of necrotic tissue are present.

While waiting for closure dressing changes need to be done frequently enough to prevent the tissues from drying out. Frequent dressing changes also allow for visual inspection to determine tissue viability and if further debridement will need to be done prior to closure.

Kidney function should be monitored and a renal consult may be needed. Serum creatinine kinase should be checked and this should stabilize prior to closure of the wound.

The wound is usually ready to close about 3 to 7 days after fasciotomy. When the skin and subcutaneous tissue can be mobilized delayed primary closure can be performed. If the tissue is too contracted skin grafting may need to be done for closure.

Regardless of the type of closure the deep fascia is left open only the skin subcutaneous tissue are closed.

After closure the patient needs to be monitored closely to make sure that there is no more muscle necrosis or dehiscence of the wound. As the patient heals the activity level can be increased. Should the muscle compartments become painful this means that the activity level is being increased too quickly and needs to slow down. If the patient has residual contracture or stiffness physical therapy may be needed.

CASE STUDY

The patient in this case history is a 30-year-old female who presented to the emergency department with a complaint of severe pain in her left leg and moderate pain in her right leg. She related that 3 days before she started working out at a local gym. She was training herself and had done extensive workouts on her legs such as using a step machine, standing leg raise and leg presses. She did this 3 days in a row and each day her legs felt worse. On this day the pain had escalated and she noticed an odd feeling in her left leg besides the pain. She also found it difficult to walk.

The patient tried to rest and this did not relieve the pain on the third day as it had on the other days. The patient decided to come to the emergency department to be checked.

In the ER the patient was examined and labs were done. The patient had taut anterior and lateral compartments of the left lower leg and they were extremely painful to touch. On the right leg the compartments were supple and only moderately painful to touch.

The patient had a serum creatinine kinase of 84,300. The anterior and lateral compartments were checked on the left leg using the Whiteside device and note to be 140mmHg on the anterior and 80mmHg on the lateral. Since a fasciotomy needed to be done it was decided to check the posterior compartments in the operating room after the administration of anesthesia. The posterior compartments were high but after the anterior and lateral releases the posterior compartments measured well below 30mmHg, so they were not opened. The lateral approach was used and the deep fascias of both compartments were completely opened. Mild muscle necrosis was observed. The wound was then dressed and the patient was taken to the recovery room.

Within an hour of the procedure the patient was asked how she felt and she said that her right leg was now

very painful and that her left leg felt much better. The right leg was examined and now was very painful on palpation. The anterior and lateral compartments were checked and they were 60mmHg and 40mmHg respectively. Because of the measured pressures and the sudden increase in pain the decision was made to open up the compartments on the tight leg. The posterior compartments were checked and were found to have normal pressures in the OR. Minimal necrosis was found in the right leg.

After surgery the patient was admitted to the ICU because of fear of renal failure due to tubular toxicity from the rhabdomyelisis. The patients CK dropped but did spike the next day and this may have been due to the delay in symptoms in the right leg compared with the left. After serial dressing changes and slow steri stripping of the wounds a delayed primary closure was then done 5 days later. The next day the patient went home and slowly increased activities and 6 months later had a full recovery with a professional trainer to help her with her training.

CONCLUSION

Acute exertional compartment syndrome is a medical emergency that if diagnosed and treated in a timely fashion can lead to a complete recovery. Part of the diagnosis process is to use a device that will measure the compartment pressure and a complete description of Whitside's device has been included.

Table 1

MATERIALS LIST FOR CONSTRUCTION OF WHITESIDE'S COMPARTMENT SYNDROME MONITOR

20ml syringe
Manual Sphygmomanometer (with Luer-lock attachment)
3-way stop cock
18g needle (1 1/2 inch)
25g spinal needle
10 ml sealed bottle 0.9% bacteriostatic saline
IV line with Luer-lock attachments
Interlink® T-connector Luer-lock connector*

*only needed if sphygmomanometer has no Luer-lock attachment

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