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PRESSURE SORE: A BRIEF REVIEW

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ABSTRACT

Pressure sore is one of the most common complications of long-term bedridden patients. In many cases, it is avoidable with the help of proper identification of patients at risk and education of the patient. The caregiver and nursing staff of the hospital have a very important role in the prevention and treatment of pressure sores. Understanding the pathophysiology of pressure sores and breaking the chain is important for their management. Much research work has been carried out regarding this topic, and as a conclusion, many pressure-relieving cushions, mattresses, and other devices like time logger devices and wheelchair patient monitors are used for treatment. This article provides a brief review of pressure sores.

Keywords: Pressure sore, Bedsore, Decubitus ulcer, Pressure ulcer

INTRODUCTION

A pressure sore is a localised injury to the skin and underlying tissue caused by continuous unrelieved pressure, generally over a bony prominence[1]. It is also known as a decubitus ulcer or a trophic ulcer. Decubitus means the position assumed when lying down[2]. A trophic ulcer is defined as a pressure ulcer caused by external trauma to a part of the body that is in poor condition because of disease, vascular insufficiency, or loss of afferent nerve fibers[3]. It is also referred to as a "bedsore" because it is commonly

seen in bedridden people. Unrelieved pressure and other risk factors cause ulceration of the skin and deeper tissue, which results in a pressure sore.

Risk factors:

Sir James Paget first described the aetiology of pressure sores as "the sloughing and mortification or death of a part produced by pressure, usually over bony prominences." The severity varies depending on the cause and site of the injury. Common risk factors are friction force, shear force, impaired sensory perception, impaired physical mobility, altered level of consciousness, faecal and urinary incontinence, malnutrition, dehydration, excessive body heat, old age, chronic medical conditions (diabetes, cardiovascular diseases), anticoagulation medication, low serum albumin level, low total lymphocyte count, fractures, social state of the person, less ability to respond to thermal stimulus, smoking, etc [4,5].

Pathophysiology:

This area of research is still full of many unanswered questions and needs more research work. Generally, a pressure sore occurs when pressure exceeds the mean capillary pressure (32 mmHg) of the affected part, which results in microcirculatory occlusion, which results in tissue ischemia, necrosis, and ulcer formation[6]. According to the hypothesis of Witkowski and Parish, due to constant pressure endothelial cells of capillaries and venules swell, separate and dilatation occurs. It will result in platelet aggregation, red blood cell engorgement, and perivascular bleeding. Oedema and perivascular round-cell infiltration also occur in affected sites. Due to these early vascular changes, necrosis also starts in subcutaneous fat. They claim that until the disease progressed very far, the epidermis didn't show any signs of necrosis[7]. Krouskop discussed another theory regarding tissue necrosis. He described the role of collagen synthesis, lymphatic drainage, and interstitial fluid in the process of tissue necrosis. He thinks thatwater-soluble collagen is generated due to hypoxia, and when this water-soluble collagen washes out, the mechanical force of that particular region is distributed to other elements, reducing the nutritional supply. The level of interstitial fluid also drops, resulting in cell-to-cell contact and rupture. As a result, the contents of the cell are released into the intercellular space. When external pressure is removed, capillaries burst, releasing tiny proteins in damaged tissue. This type of tissue poisoning occurs due to reduced lymphatic flow, which occurs due to hypoxia or drugs like heparin or glucocorticoids. He also explains how glucocorticoids are produced in response to stress and inhibit the movement of smooth muscle in the lymphatic system. So water-soluble collagen is produced. In old age, the formation of elastic fibres decreases, which increases the risk of pressure damage. As a result, two-thirds of pressure ulcers develop in old age[7]. According to the theory of Seiler and Stahelin, fibrinolytic activity is diminished in traumatised tissue, which leads to fibrin deposits in the capillaries and intercellular space. This may result in vascular blockage and tissue necrosis[7].All these theories focus on ischemia and hypoxia, which lead to a decreased supply of oxygen to the tissue. It is the main factor in tissue necrosis.

Common sites [8,9]:

Pressure sore formation depends on the thickness of soft tissue covering prominent bony surfaces and the position of the patient. Pressure sores are more common in human body sites with the less soft tissue on bony prominences.

Table No. 1 – Common sites for pressure sore

Supine position	Lateral position	Prone position	Wheelchair position
Back of the Head	Ear	Elbow	Shoulder blades
Shoulder	Shoulder	Rib cage	Buttocks
Elbow	Elbow	Thigh	Heel
Buttocks	Hip	Knee	Ball of foot
Heel	Thigh	Toes	
	Leg		
	Heel		

Along with these common sites, ischial tuberosity and sacrum are the commonest sites for a pressure sore.

Role of bacteria in pressure sore [9]:

Some researchers believed that bacteria were not the cause of the formation of pressure sores but were responsible for delayed healing. Hypoxia and anoxia are the main causes of impairment of lymphatic flow and waste products of microorganisms that can't be removed from the affected site, which eventually leads to bacteraemia and tissue breakdown. The type of bacteria is also responsible for delayed healing. Generally, slow-healing wounds had no anaerobic bacteria and very few colonies of aerobic bacteria. On the other side, a generally non-healing wound had a high number of both anaerobic and aerobic bacteria.

Diagnosis:

Generally, no equipment is required for the diagnosis of pressure sores. A physical examination and a medical history were used to make the diagnosis. Some researchers believed that physicians should examine the deeper tissue first for signs of ulcer formation in the affected site.

Classification:

According to the Stirling pressure sore severity scale, it is classified into 4 stages [10].

Table no. 2 – Stages of Pressure sore

Stages	Characteristic
Stage 0	No clinical evidence of pressure sore
Stage 1	Discolouration of intact skin (light finger pressure applied to the site does not alter the discolouration)
Stage 2	Partial thickness skin loss or damage involving the epidermis and/or dermis
Stage 3	Full-thickness skin loss involving damage or necrosis of subcutaneous tissue but not extending to the underlying bone, tendon, or joint capsule
Stage 4	Full-thickness skin loss with extensive destruction and tissue necrosis extending to the underlying bone, tendon, or joint capsule

Devices used to measure pressure[7]:

Pressure-sensing devices are used to measure pressure in the form of a cushion or mattress-skin interface while the patient is in contact with it. Air cell transducers are the earliest developed and most commonly used. Now it is replaced by electrical transducers, which work based on differences in resistance levels but monitor only a small area of the skin's surface and interrupt the contact between cushions and skin. Its measures are not given the actual value of pressure. So, it is only used for comparative purposes. Some specially designed evaluator pads are also used for measuring pressure between skin and cushions. The ultrasonic dimension gauging technique is able to collect data without interfering with the skin-cushion contact, but it only collects dimensional contour data. A pressure Scanner is an advanced tool for measuring pressure data. It collects information via 64 transducers and produces data outputs using a minicomputer, which creates a three-dimensional plot diagram. It gives information about the centre point of pressure and where the pressure is distributed. Another useful tool for measuring pressure is the isobarograph. It also provides information on the centre point of pressure as well as pressure patterns with different colours.

Scales for the study of pressure sore

Different types of scales were specially designed for the study of pressure sores.

Waterlow scale[11]: TheWaterlow scale was developed in 1985 by clinical nurse teacher Judy Waterlow. It gives an estimated risk for the development of a pressure sore in a given patient. It has eleven factors for assessment. They are as follows: body type, skin type, mobility, sex, age, appetite or nutrition, neurological deficit, surgery, trauma, and medications. Each factor is assigned a specific score for assessing patients. The minimum score is one, scores of 10 to 14 are considered "risk," scores of 15 to 19 are

considered "high risk," and scores of 20 or higher are considered "very high risk."

Jackson/Cubbin score[12]: It is an upgraded version of the Cubbin/Jackson scale and is specially targeted for ICU patients. It has 15 factors for assessment. They are age, weight, past medical history, mobility, hemodynamic status, respiration, oxygen requirements, nutrition, incontinence, and hygiene. These twelve factors are rated from 1 to 4. The remaining 3 factors are blood products, surgery in the last 24 hours, and hypothermia, which is scored if present and results in the subtraction of one point from the total. A lower score indicates the highest risk for pressure sores. The minimum possible score is 9, while the maximum possible score is 48. Scores less than 30 indicate patients at "high risk."

Braden scale[13]: The Braden scale was developed by Barbara Braden and Nancy Bergstrom in 1987. It has six criteria for assessing patients. They are sensory perception, moisture, activity, mobility, nutrition, friction, and shear. Each factor is rated from 1 to 4, except friction and shear, which are rated from 1 to 3. On this scale, a higher score indicates a lower risk of developing a pressure sore and vice versa. A total score of less than or equal to 9 is considered "severe risk," a total score of 10 to 12 is considered "high risk," a total score of 13 to 14 is considered "moderate risk," and a total score of 15 to 18 are considered "mild risk."

Norton scale[14]: The Norton scale was developed by Doreen Norton in 1962. It uses five criteria to evaluate patients. They are physical condition, state of mind, exercise, mobility, and incontinence. Each factor has a severity scale of 1 to 4. The total score will be between 5 and 20. Total scores of 5 to 9 are considered "very high risk," total scores of 10 to 14 are considered "high risk," total scores of 15 to 18 are considered "medium risk," and total scores of 18 or higher are considered "low risk."

Complications[15]:

Cellulitis, Abscess formation, Osteomyelitis, and Bursitis are the common complications associated with Pressure sores.

Treatment:

Treatment of a pressure sore depends on the depth of the ulcer and the severity of the disease. Its treatment includes surgical and/or non-surgical management. Surgical management includes reconstructive surgery, while non-surgical management includes negative pressure wound therapy, hyperbaric oxygen therapy, etc. Although the first line of treatment is to treat the cause, possible removable risk factors should be removed. Generally, the wound should be cleaned with a normal saline solution when each dressing is changed. If non-viable tissue is present, then debridement should be performed. It may be of the autolytic, chemical, mechanical, or surgical type. A small amount of dead tissue clears by itself with the help of the body's defence system and enzymes in a moist environment; it's called autolytic debridement[16]. Different types of hydrogels, hydrocolloid dressings, hydrofibers, and some transparent films are used for it. Chemical debridement is also known as enzymatic debridement. It uses ointments or gels that contain enzymes that soften and remove dead or unhealthy tissue by digesting or dissolving it[17]. Generally, mechanical debridement is a nonselective type of debridement because it can harm viable tissue along with devitalized tissue and debris. It is carried out with mechanical force like wet-to-dry dressing, pulsatile lavage, and wound irrigation. Surgical debridement is the removal of dead tissue with the use of surgical instruments like a scalpel, scissors, or forceps[18].Wound dressing is an important part of pressure sore healing. It may be absorbent or non-absorbent in type. Absorbent dressings are commonly used to absorb pressure-sore exudates. They mostly provide non-sticky surface attachment, so they are non-adherent by nature[19].Non-absorbent dressings are typically used in wounds with a small amount of exudate. Negative pressure wound therapy is becoming more popular day by day for pressure sore management. It is also known as "vacuum-assisted wound closure." It works by creating a vacuum inside a specific sealed area that covers the wound[20]. Hyperbaric oxygen therapy is also helpful in the management of pressure sores. It delivers more oxygen-rich blood to

the affected pressure ulcer site, causing neovascularization and promoting rapid healing[21].Reconstructive surgery is essential in complex grade III and Grade IV pressure sores. It is performed using skin grafts and skin flaps. A skin graft is the surgical removal of skin from the donor area to the diseased area, whereas skin flap placement is the surgical removal of local tissue around the wound that has an intact blood supply. They may be local or regional. Local flaps are applied using transposition, rotation, the Limberg flap, the V-Y advancement flap, etc. Regional flaps may be musculocutaneous, fasciocutaneous, perforator flaps, etc[22].

Prevention:

Prevention is better than cure for all diseases. For the prevention of pressure sores in certain diseases and the postoperative period, knowledge of risk factors is very important for hospital staff and patients. They should teach the patient to change their body posture at a specific time. Providing an appropriate support surface is necessary to prevent friction and shear forces for elderly and bedridden patients. Incontinence should be managed by timely cleansing, and skin should be protected by applying a moisture barrier ointment. Many seat cushions have been created in order to distribute pressure in the sitting area. Generally, it has four categories based on the material used inside it, i.e., foam, viscoelastic foam, gel, and fluid flotation.

Many researchers also study the above four materials combined or separately in the formation of sitting cushions. Bowker and Davidson used a thixotropic substance in cushions, which is a gel in the stressed state and a solid in the unstressed state. It offers the benefits of flexibility and support at the same time[23].Rosenberg and Lach worked on the design of a pressure sore prevention device. In their device, a receptor chamber located under the person's buttock gathered data and analysed it. It is connected to a small effector balloon. When an area of the buttock has been under pressure for a long time, the device produces an uncomfortable sensation on the healthy skin around the pressure point. This will lead the person to relieve the pressure and change positions[24].The Time Logger Communicator (TLC) system is also a great device for measuring pressure data. It contains a portable computer that collects data and records information about how long the patient has been sitting in the wheelchair, when during the day, and how frequently pressure is lifted. A computer analyses the data and provides different types of plot diagrams. It has an alarm that sounds when the time or pressure level reaches the present level. It is also easily clamped to a wheelchair frame[7].The wheelchair Patient Monitor (WPM) system is another system that works with seat sensors. Its information is gathered by computer, and after analysis, graphs and histograms can be constructed. It also has a time-based alarm that is set off when weight shift behaviour has not been performed for a set period of time[7].

CONCLUSION

There has been a lot of research done on pressure sores that are more closely aligned with laboratory findings but have less practical application. It is necessary to research the histology of affected tissue because deeper tissue is more affected than superficial tissue. Pressure sore development is a complex process that is not dependent on any single factor. So, surgeons try to avoid most of the causative factors in suspected patients. Awareness regarding the causative factor for the patient and caregiver is essential for the prevention of bedsores. After the formation of bedsores, adequate treatment and proper dressing, along with a change of posture and local hygiene care are essential for the management of bedsores.

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