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COAGULATION FACTORS CHANGES IN BURN PATIENTS

THESIS

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Ву

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INTRODUCTION AND AIM OF WORK

INTRODUCTION & AIM OF THE WORK

Many of the haematologic changes that occur following burn injury are the characteristic responses seen in patients following surgery or trauma (Eurenius 1979).

Damage to the vascular wall, decreased blood flow and changes in platelets adhesiveness, blood coagulation, and fibrinolysis are factors which are thought to predispose to thrombosis. These factors interact in a complex manner and thrombus formation may be caused by changes in one or several of them. The mechanism of this interaction is as yet unknown.

Thrombosis is an invariable pathophysiologic reaction to severe burn, and thromboembolism is a common later complication. Full thickness burns result in immediate confluent thrombosis of the dermal vasculature. In the peripheral, less severely injured tissues, progressive thrombosis occurs for the first 12 to 24 hours and may result in lesion extension (Robb, 1967).

Later, especially in patients with extensive injuries, sepsis and / or circulatory failure may trigger a haemorrhagic diathesis due to consumption of platelets and other intrinsic coagulation factors (Gehrke, et al., 1971).

REVIEW OF LITERATURE

Many investigations have been carried out to determine if any typical changes in one or several of these factors are present in conditions which are associated with high risk of thrombosis (Johan Jgge, 1970).

haemorrahgic complications may develop in the severely burned patients as a result of defects in haemostasis. Blood coagulation after burn injury has been studied only to a limited extent, and data obtained in experiments often have been inconsistent.

Campbell and his co-workers in 1950 made the observation that there are very interesting changes in several individual clotting factors but no marked defect in coagulation (Gehrk, 1971).

Lebeaupin and his associated in 1965 noted severe disturbances in coagulation which provided prognostic information.

In this thesis we shall investigate fifteen major burn patients to study the changes in the individual coagulation factor in the first ten post burn days to show:

- If there is typical course of these factors or not.
- Clinical application of these changes.

PATHOLOGY OF BURN

Thermal injury always constituted as one of the chief medical problems. Burns not only alteration of the outer surface of the body, but there is also local and general physiological changes that affect survival of the patients.

The magnitude of the burn is directly proportional to the causative factor

Causes of thermal injury:

Thermal injury occurs as a result of an energy transfer from a heat source to the body. This can occur by direct conduction or by the effect of electromagnetic radiation.

Conduction of the thermal energy occurs along the gradient from the higher to the lower temperature. Many factors can affect the response of the body to these energy transfer.

Conductivity through the different local tissue has a major effect upon the rate of loss or gain of thermal energy. Studies of tissue conductivity indicate that nerves and blood vessels conduct heat with the greatest ease, where bone is the most resistant, and other tissues are intermediate. (Ponder, 1962).

The peripheral circulation will also be a major factor in determining the rate of absorption or dissipation of heat throughout body tissue (Moritz, 1947).

Also surface pigmentation and the presence or absence of different pigment densities, presence or absence of other insulating material such as hair, natural skin oil, cornified layers of surface epithelium and total water content of the tissue will be other factors that can affect conduction of thermal energy.

Eletromagnetic radiation also can produce major thermal injury. This electromagnetic spectrum includes a wide variety of radient energy as , X-ray, microwaves (radar), infrared, ultraviolt, visible light and gamma rays. The capacity of these radiations to penetrate living tissues depends on their frequency and wavelength. Only the shortest rays in the electromagnetic spectrum (X rays and gamma rays) are capable of significantly penetrating and ionizing molecules within living cells . The shorter the wavelength, the greater the energy and penetrating power of the rays. The precise mechanism of injury following radiation is not clear, but there are several theories, the formation of ion pairs within a cell may inactivate or damage enzyme systems; breakdown products as a consequence of ionization diffuse toxic throughout the cell; and genetic or metabolic patterns may be injured by the ionization of key molecules within the molecular anatomy of the cell. Recent studies demonstrate sensitivity of deoxymibonucleic acid (DNA) to ionizing radiation. Both structural and functional damage can be assessed in vitro. Particulate radiation, e.g. alpha and beta particles are another type of radiation injury.

These particles, traveling at various speeds and with variable energy, are capable of causing radiation injury due to the ionization of molecules within the cells of living tissue by the displacement of electrons from the molecular structure (Hartwell, 1979)

Effect of heat on local tissues:

Below 44 °C local cellular damage dose not occur to a significant degree unless the exposure is for protracted periods of time. Around this temperature, the rate of local tissue damage and recovery is in such delicate balance, that although equilibrium can be maintained for a period of approximately six hours, beyond this time irreversible damage as deep as the basal cells of the epidermis occurs.

In temperature ranges between 44 and 51°C at the surface, the rate of cellular destruction doubles with each degree rise. And even limited exposure to this temperatures is necessary for tissue destruction. At temperatures greater than 51°C, the exposure time required to destroy the epidermis is so brief and the reparative processes so overwhelmed that rate of destruction is very rapid. Above 70°C cellular destruction is so rapid that only extremely brief periods of exposure are necessary for total tissue destruction (Moncrief 1979).

Evaluation of burn injuries:

Because the effect of the causative agent on the local tissue varies according to its temperature and to the time of exposure; the burn can be classified according to the depth of the destructed tissue. In addition the magnitude of the injury is also depends on the extent of the surface area burned.

a- <u>Depth</u> :

In the past, the burn wounds have been classified into first, second and third degree according to the tissue involved which indicates an increase in depth of tissue destruction.

Today, thermal injury can be classified as either partial or full thickness. Partial thickness wounds also divided into superficial and deep wounds.

The superficial partial thickness wound involves usually only the epidermis and extends in general to the upper portion of dermal papillae.

The deep partial thickness wound destroys the entire thickness of the epidermis, including the dermal papillae, but leaves intact sweat glands and hair follicles from which marginal spread of epithelial elements gradually covers the wound.

The full thickness burn wound must include destruction of not only the epidermis but also all of the dermal appendages and other epithelial elements, so spontaneous restoration of skin coverage is not possible.

Clinically, the superficial partial thickness wound shows that the surface may be covered with blisters or bullae of varying sizes and when these are removed the epidermis beneath it is weeping, glistening, bright pink or red and sensitive to temperature changes, exposure to air, and light touch. The deep partial thickness wounds are waxy, white but still soft and elastic, and although they are sensitive to pressure, they are insensitive to light touch or soft pin prick. The full thickness wound are dry and hard, ten or fawn coloured and with exposure to air in a matter of a few hours becomes dessicated, parchment like and translucent. The thrombosed dermal vessels beneath the surface can be seen. This type of thermal injury results in a very inelastic eschar which leads to compression of deeper tissues when edema forms beneath it (Moncrief 1979).

b- Extent : ·

Estimation of the surface area of the burn can be done roughly by application of the well known rule of nine as advised by Wallace 1951 (Fig. 1). This does not give an accurate estimation of the involved area as other formulae; but it is easy enough for practical purposes and easy to remember, especially in acute stages where early starting of the fluid replacement is desirable, (Muir & Barclay 1974) this rule is less accurate in children because of the different relative proportions of the head, trunk and extremities as compared to the fully grown individual (Salisbury, 1983).

Rule of Nine (9)

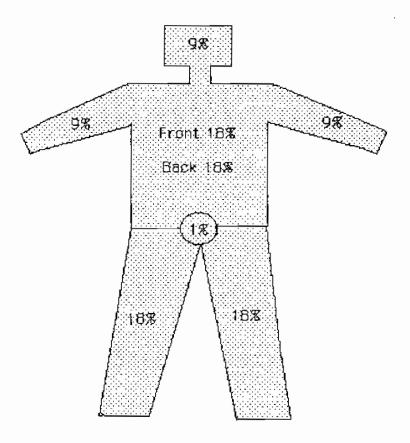


Fig. (1): Estimation of surface area (After Muir & Barclay, 1974)

So, a more accurate assessment of the area involved can be obtained by employing a more inclusive chart as, the Lund Browder chart which is a modification of Berkow's work in 1924. (Curtis & Dabney 1977) This chart not only takes into consideration variations in body proportions with age but also provides a permanent medical record of the initial injury. (Fig. 2) (Salisbury, 1983) .

In a small lesions, a good guide is that the area covered by the patient's hand and fingers is 1% of the body surface (Muir & Barclay 1974).

Accordingly the burn injury can be classified into major, moderate and minor injury.

The major burns includes partial thickness skin injuries involving more than 30 % of the body surface, partial and full thickness injuries of the face, hands, genitalia or feet, and deep burns involving more than 10% of the body surface. Also electrical burns or burns complicated by respiratory tract damage, fractures and extensive soft tissue injuries or burn with pre-existing disease.

Moderate burns includes superficial partial thickness burn of more than 15% of the body surface, deep partial thickness burn of 15–30% and full thickness burn less than 10% excluding the hands, face and feet.