Subcutaneous Compartment Syndrome in Burns

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EDITORIAL

Subcutaneous compartment syndrome [1] occurs due to raised subcutaneous tissue pressure above a critical level. Subcutaneous compartment pressure if raised above 30-40 mm Hg will compress capillaries in overlying dermis, subcutaneous tissue and underlying fascia [2] and thereby as per oxygen nutrient cut off theory [3] due to failure of energy dependent cellular mechanisms cell death will occur.

Subcutaneous compartment syndrome for better understanding of clinical picture may be divided in 3 stages:

Stage 1: In subcutaneous compartment syndrome, initially there will be spasm of capillary sphincters and sluggish circulation will be noted. At this stage [stage 1] clinically tense shiny skin, delayed capillary filling and blanching [brawny indurations] becomes evident. This is followed by increased hypoxia [characterized by discomfort and pain with increased permeability]. Blister with clear fluid may appear.

Stage 2: If condition deteriorates further cessation of venous out flow due to compression of venous end of capillaries will occur. At this stage [stage 2] if condition progresses slowly and persists for long, there will be increased hypoxia and capillary oozing that will manifest clinically in form of skin blisters and after rupture of blisters dark lived red/purple/bluish colored wet floor will be visible.

Stage 3: If this stage continues for long, there will be dark discoloration of overlying skin and skin necrosis [stage 3] indicating permanent damage cellular death due to failure of vital cellular mechanisms.

If condition progresses very fast, blistering [stage 2] may not occur and the conditions will rapid progress from stage 1 to stage 3 without formation of blisters. If condition is due to infective in origin, thrombosis of capillaries further hastens the progress. If blisters rupture, infection may further aggravate the condition due to increased capillary permeability following inflammation.

Understanding subcutaneous compartment syndrome may help a burn surgeon to control the conversion of superficial burn to deep burn due to subcutaneous compartment syndrome [4]. Basic pathophysiological change in burn patient is increased capillary permeability which in absence of sepsis, gradually returns to normal on successful resuscitation. Hence, loss of circulating fluid in subcutaneous tissue may increase the subcutaneous compartment pressure during fluid resuscitation [a large amount of positive fluid balance is frequently present during initial burn resuscitation due to accumulation of fluid in subcutaneous tissue and 3rd spaces]. If intermittent compression is applied on the involved area the conversion of superficial wound to deep and damage to epithelial cells of sweat/sebaceous glands in dermis may be controlled provided it is due to compartment syndrome. In my experience of limited access dressing [LAD] which follows definite intermittent negative pressure schedule, wound healing is faster than conventional semi-occlusive dressing.

REFERENCES