EXPERIMENTAL STUDY OF A NEW POLYMER COVERING FOR BURNS TREATMENT

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Measures creating the most favorable conditions for rapid epithelization of superficial burns and for skin autografting for deep burns, which are reliable methods of preventing the numerous possible complications, are of the utmost importance in the local treatment of burns. Early removal of necrotic tissue from deep burns and the earliest possible skin autografting promote the rapid healing of burn wounds and prevent metabolic disturbances and the development of an invasive infection [2, 4, 6, 9, 18].

The formation of a necrotic scab on a burn wound begins at the moment of trauma and can proceed with the development of wet or dry necrosis, the former being characterized by intensive suppuration. Because of this marked degree of suppuration, wet necrotic tissues are rejected earlier, but in the initial stages there is no demarcation barrier, and the suppurative process spreads to underlying healthy tissues, and this is accompanied by marked toxic manifestations and frequent development of generalized infectious complications [7, 13]. If a dry scab is present the quantity of toxic products passing through the wound surface into the body is sharply reduced, and the mummified burn scab prevents the abundant loss of fluid and protein with the wound exudate and protects the body against bacterial invasion [7, 8, 13]. The presence of dry necrosis must therefore be regarded as more favorable, and for that reason local treatment must be aimed at the formation of dry coagulation necrosis [6]. Epithelization of superficial burns takes place under the scab, and the presence of a coagulation scab on a burn wound in the case of deep thermal injuries allows early debridement and skin autografting. Among the various types of medication promoting the formation of coagulation necrosis on a burned surface is tannin (tannic acid). Treatment of burns with tannin, which was introduced into clinical practice by D. P. Nikol'skii (1888) and improved by Davidson, soon gained worldwide recognition [9, 15]. However, a number of important disadvantages, such as the instability of the tannin solution, the need to irrigate the burned surface frequently, and the soiling and spoiling of clothing and bed linen proved to be an obstacle to widespread use of the tannic acid treatment.

The all-union "Medpolimer" Research Institute jointly with the A. V. Vishnevskii Institute of Surgery, Academy of Medical Sciences of the USSR, has developed a new polymer covering for the treatment of burns, which has a tanning and absorbent action, and its investigation is described below.

EXPERIMENTAL METHOD

A comparative experimental study was made of coverings for the treatment of burns. The experimental animals were noninbred male albino rats weighing 180-210 g. After preliminary removal of the hair a steam burn was inflicted on the anesthetized animals, on the inferolateral surface of the trunk, through a rectangular window measuring 3.5 × 4.5 cm in asbestos paper, with an exposure of 30 sec; this resulted in a burn of the IIIb-IV degree covering an area of 5-6% of the body surface. The depth of the burn was determined by histological investigation and its area was calculated by the equation

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TABLE 1. Comparison of Experimental Results for Different Groups ($p < 0.05$)

Group	Time of formation of dry scab, days	Time of chemical debridement, days	Microbial contamination of wound, number per gram of tissue
Experimental	1—2	6—7	3,5±0,1
Control:			
1 subgroup	7—8	8—9	4,8±0,04
2 « »	2—3	7—8	5,6±0,01
3 « «	8—9	9—10	6,6±0,1

$$S = K \cdot W^{0.6},$$

where S denotes the area of the body surface in cm^2, K is a coefficient equal to 12.54, and W the animal's weight, in grams [2]. All the animals were divided into two groups: experimental and control, with 30 rats in each group. In the experimental group, local treatment consisted of application of the polymer covering with tanning and absorbent action. The control group was divided into subgroups 1, 2, and 3, each consisting of 10 rats. In subgroup 1 local treatment was by application of algipore [12], in subgroup 2 with a 2-5% solution of tannin, and in subgroup 3 with a 0.02% solution of furacillin (nitrofurazone). The polymer covering was applied once only. The algipore was changed daily. The dressings were irrigated with tannin solution every 6 h during the first 2 days after burning. The dressings with furacillin solution were changed daily. As a dry coagulation scab formed on the wound, and allowing for the times of formation of the demarcation barrier, chemical debridement of the wounds was performed on the burned animals with a 40% salicylic acid ointment; a morphological investigation of the scab was carried out with quantitative and qualitative monitoring of the wound microflora by the method described in [5, 1, 16]. The tissues of the liver and kidneys also were studied morphologically.

EXPERIMENTAL RESULTS

It was found in the course of the experiment that in the animals of the experimental group a dry scab formed on the burn wound after application of the polymer covering on the 1st or 2nd day after burning. Morphological investigation of wound biopsy material showed that scab formation on the wound of the animals of this group proceeded with the development of coagulation necrosis. The scab under these circumstances incorporated coagulated skin, subcutaneous cellular tissue, and muscle. A marked degree of leukocytic infiltration with the formation of a demarcation barrier was observed in the substance of the subcutaneous tissue. Remnants of epithelium in a state of mummification remained on the surface of the scab. Microbial contamination of the wounds of animals of the experimental group amounted to 3.5 ± 0.1 per gram of tissue. During statistical analysis of bacterial contamination of the burn wounds, the logarithmic scale to base 10 was used.

In the animals of subgroup 1 of the control group, scab formation was complete on the 7th-8th day after application of algipore. The morphological investigation showed that the scab on the animals of this subgroup was edematous and densely infiltrated with keukocytes. In the subjacent tissues edema and infiltration by polymorphs were present. The surface of the scab was covered with a layer of microflora. Contamination of the wounds measured 4.8 ± 0.04 per gram of tissue.

Application of tannin solution to the animals of subgroup 2 led to the formation of coagulation necrosis on the wound on the 2nd-3rd day after burning, but morphological investigations revealed a high concentration of microorganisms both on the surface and in the depth of the scab. Microbiological investigation revealed 5.6 ± 0.01 microorganisms per gram of tissue.

In the animals of subgroup 3, morphological investigation 8-9 days after the beginning of application of 0.02% furacillin solution showed that the scab on the wound was loose and edematous, with many microorganisms on its surface. In individual cases, edema and leukocytic infiltration of the proliferating granulation tissue, with the formation of a deep abscess, were observed beneath an ill-defined demarcation barrier. Microbial contamination in this subgroup was the highest of all, namely 6.6 ± 0.1 per gram of tissue. The experimental results for the different groups are compared in Table 1.

Morphological changes in the liver and kidneys of the animals of the experimental and control groups were similar in type and consisted of congestion of the hepatic and renal capillaries, which varied in degree. In some cases degenerative changes were observed in the hepatocytes and the epithelium of the convoluted tubules of the kidneys. No necrosis of hepatic or renal tissue was observed.

The use of the new polymer covering, possessing tannin and absorbent action, for the treatment of burns thus leads to the formation of a dry coagulation scab on the wound in the course of 1 or 2 days, and allowing for the time of formation of the demarcation barrier, this means that early debridement can be carried out on the 6th-7th day after burning. When the polymer covering was used to treat experimental burns, no toxic effects were found on the liver or kidneys.

LITERATURE CITED

1. P. O. Barr, G. Birke, and S. O. Liljedahl, Éksp. Khir., No. 2, 39 (1968).
2. L. B. Berlin, Morphology of the Skin after Burns and Free Grafting [in Russian], Leningrad (1966).
3. N. I. Kochetygov, Burns [in Russian], Leningrad (1973).
4. M. I. Kuzin, Vestn. Akad. Med. Nauk SSSR, No. 3, 56 (1979).
5. M. I. Kuzin, I. I. Kolker, V. K. Sologub, et al., Klin. Med., No. 3, 93 (1981).
6. M. I. Kuzin, V. K. Sologub, I. Chervenkov, et al., Khirurgiya, No. 4, 10 (1982).
7. M. I. Kuzin, V. K. Sologub, N. A. Olyunina, et al., Khirurgiya, No. 1, 127 (1984).
8. V. A. Lavrov, N. A. Olyunina, Yu. E. Babskaya, et al., 3rd All-Union Conference on Modern Methods of First Aid and Treatment of Burns [in Russian], Moscow (1986), pp. 52-54.
9. R. I. Murazyan and N. R. Pachenkov, Emergency Aid in Burns [in Russian], Moscow (1983).
10. D. P. Nikol'skii, Vrach, **9**, 166 (1888).
11. V. K. Sologub, V. G. Borisov, and V. V. Yudenich, Sov. Med., No. 6, 89 (1983).
12. V. K. Sologub, I. Chervenkov, E. I. Vyglenova, et al., Khirurgiya, No. 3, 110 (1983).
13. M. B. Shenker, V. S. Yakubovich, and L. P. Raskina, New Therapeutic Preparations (Express Information, All-Union Research Institute of Medical Information) [in Russian], No. 2 (1985), pp. 2-7.
14. C. R. Baxter, P. W. Curreri, and J. D. Marvin, Surg. Clin. N. Am., **53**, 1509 (1973).
15. E. Davidson, Surg. Gynec. Obstet., **41**, 202 (1925).
16. C. Hendley, K. C. Robbins, B. Deetinger, et al., Arch. Int. Pharmacodyn., **106**, 164 (1956).
17. E. C. Loeble, J. A. Marvin, E. L. Heck, et al., Am. J. Clin. Path., **61**, 20 (1974).
18. W. W. Monafo and B. Frudman, Surg. Clin. N. Am., **67**, 133 (1987).
19. L. D. Stern, Scand. J. Plast. Reconstr., **13**, 105 (1979).