The Effects of Two Different Burn Dressings on Serum Oxidative Stress Indicators in Children with Partial Burn

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In this study, we evaluated and compared the effect of treatment with a hydrofiber dressing with silver (HFAg) and a polylactic membrane (PLM) on systemic oxidative stress in systemic inflammatory reaction in thermal burn injuries in children. A prospective randomized and matched pairing study of 20 to 50% of TBSA was performed from children equal to both sexes affected by thermal injuries. The control group was included in normal children of both sexes. Serum malondialdehyde (MDA), total antioxidant capacity (TAC), total oxidant capacity (TOC), and glutathione (GSH) levels were analyzed and the results were analyzed statistically. In this study, it was found that PLM treatment increased TAC and GSH levels in burn patients significantly more than the other group. With the use of PLM, TOC decreased to normal level from day 3. In the HFAg group, TAC and GSH levels began to increase on the seventh day. On the first day of the burn, the TOC level started to increase. This increase continued on days 7 and 14. The TOC level began to fall on the 21st day. The increase in TAC was higher in the PLM group. In the PLM group, TOC fell faster. As a result, we think that different burn dressings can have different systemic effects. We can speculate that PLM has an antioxidant effect in the burn tissue due to high lactate content. Therefore, PLM may have decreased serum oxidative stress indicators more effectively than HFAg.

Burns with fires are now the fifth leading cause of deaths in homes in the United States.[1] They are the third cause of deaths related to unintentional injury among children between the ages of 5 and 14.[1] Pediatric burns from scalding are the most common cause of hospitalization for patients under the age of less than 5 years, whereas fire and flame injuries in older children are more common.[2,3] Burn is a trauma that has systemic effects that cause physiological damage to the patient in many ways.[4] These injuries lead to the deterioration of the comprehensive skin barrier, which creates new areas for bacterial colonization and contributes to the immunosuppressive situation, rendering the burning defenseless to infectious complications.[5,6]

In a severe burn, the main event that provides the emergence of systemic inflammation in the body is tissue damage.[7] This tissue damage occurs both due to thermal trauma and subsequently due to ischemia-reperfusion.[7] Recent research has shown that the event that initiates inflammation is the activation of Toll-like receptors (TLR) and NOD-like receptors (NLRs).[7] TLR is expressed by leukocytes and some paranchymal cells and provides the production and release of endogenous cellular factors that initiate the response of inflammation.[7] TLR is activated by fragmented intracellular particles because of thermal damage.[7] NLRs are found in the cytoplasm of leukocytes.[7] The destroyed cell membrane particles activate the NLRs. NLRs and TLR are two sensing systems that play a role at the onset of inflammation.[7] When both TLR and NLRs are activated, it ensures that the IL-1 and the IL-18 are activated. They then initiate the IL-1 and IL-18 inflammation process.[7]

Recent studies has revealed that a second inflammatory process has occurred after burns in severe burns.[7,8] They explained this process with two-hit hypotheses.[7,8] In this hypothesis, TNF-α has been suggested to play a key role.[7,8]

Some studies have suggested that taking control of the inflammatory process can reduce the mortality in burns.[7,9–11] In these studies, hemofiltration, antimicrobial treatment, proper fluid resuscitation, nutritional support, necrotic tissue excision, hemodynamic support, and appropriate wound dressing have been suggested to be effective in controlling inflammation.[7,9–11]

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In severe burns, free radicals arise due to regional and systemic inflammation.[12] Adequate occurrence of free radicals affects wound healing positively with antimicrobial activity occurring in the wound area.[12] However, excessive amount of free radical formation means increased oxidative stress.[12] Increased oxidative stress products are controlled by the body's antioxidant capacity.[12] In the case of free radical formation above the antioxidant capacity, pathophysiological events may develop in the burn.[12] Therefore, the balance between oxidant capacity and antioxidant capacity to minimize the damage caused by the burns is very important.[12] If the storage of antioxidant capacity is high, the danger does not occur.[12] However, if the antioxidant capacity is limited tissue destruction, organ failure may develop.[7,12] In the treatment of burns, it is important to provide antioxidant support as required by diet, as well as strategies for reducing the formation of oxidative stress products in the wound area.[7,12]

In this study, we evaluated the efficacy of two different wound dressings in reducing oxidative stress in thermal burn injuries in children. We measured serum total antioxidant capacity (TAC), total oxidant capacity (TOC), glutathione (GSH), and malondialdehyde (MDH) levels in blood samples taken from the patients.

MATERIAL AND METHODS

Study Protocol

Ethics committee approval was obtained from the ethics committee of Inonu University Clinical Trials for this study (No: 2017/129). This prospective study included 60 thermally injured children of both sexes, aged 1 to 16 years, who were admitted to the Burn Unit of the Pediatric Surgery Department, Inonu, Faculty of Medicine, Turgut Ozal Medical Center, from January 2018 to June 2018. All children suffered acute burns affecting 20 to 50% of TBSA, including deep burns to 5 to 10% of their TBSA. The control group consisted of normal children who brothers and sisters of patient with equal sexes, also aged 1 to 16 years.

Patients admitted after the first 24 hours following their injury, those with previous or current gastrointestinal diseases, or with chronic diseases, such as diabetes, as well as those with burns of the upper respiratory tract or an inhalation injury, were excluded from the study. Patients who did not develop sepsis or renal insufficiency during the treatment were included in the study.

After the informed consent of the patient's parent was completed, all patients underwent a thorough clinical evaluation and appropriate fluid replacement for the burn area and weight.[13] All patients were given the same standard burn treatment during the treatment period with fluid treatment.[13] All patients were started to be fed early and nutrition was performed in calories and diets in accordance with standard burn patients.[13] The burn wounds of all patients were thoroughly and gently cleansed by wiping with serum saline-treated sterile gauze. The wounds were debrided with a surgical sponge, rinsed, and dried.
Dressings checked were:

- A dressing with silver (HFAg)(Aquacel® Ag; Convatec, Princeton, NJ) is a moisture-retaining dressing comprising sodium carboxymethylcellulose fibers forming a gel in contact with the wound fluid.[14]
- A polylactic membrane (PLM) is a synthetic copolymer of Suprathel® (PMI Polymedics, Denkendorf, Germany), DL-lactide (>70%), and ε-caprolactone.[15]

The PLM group (n = 20) was dressed with a synthetic copolymer composed of DL-lactide (>70%) and ϵ-caprolactone first 7 days. After that, the dressing was changed every 3 days. The HFAg group (n = 20) was dressed with a hydrofiber dressing containing silver first 7 days. After that, the dressing was changed every 3 days. The wounds in the HFAg and PLM groups were re-evaluated before each dressing, respectively, and those with early signs of healing, including macroscopic re-epithelialization, advancing margins, decreased bleeding and exudates, and less pain continued to be dressed.

At the end of the treatment period, burn wounds of all patients completed the re-epithelialization. During 21-day dressing period for each dressing group, clinically wound healing was evaluated. The dressings in the PLM group and HFAg group were changed on days 7, 10, 14, 17, and 21. Wound healing was evaluated as the completion of epithelialization. Two different pediatric burns physicians (with years 26 and 11 of experience, respectively) evaluated wound healing.

All patients participating in the study were subjected to routine examinations. Every week, full blood count, blood culture, plain chest x-ray, liver function, arterial blood gases, and coagulation profile were performed.

Blood Samples

Blood samples were collected from all burn groups on days 0, 3, 7, 14, and 21 after the burn and once in the control group. Blood was collected into tubes. The samples were stored at −80°C until analysis.

The blood samples were warmed to 23°C on the day of the analysis, and centrifuged immediately at 4000 rpm for 7 minutes; the serum was collected for analyses of MDA, TAC, TOC, and GSH levels.

Malondialdehyde Levels

The MDA levels were measured with the method of Uchiyama et al[16] Determination of the lipid peroxide product of sample is performed by means of MDA with TBA (Thiobarbituric acid) at 95°C. The MDA content extracted with n-butanol and the pink-colored product determined at 532 nm wavelength.

Glutathione Levels

The GSH levels were determined with the method developed by Elman.[17] It was placed in a tube and DTNB (5,5′-dithiobis 2-nitrobenzoic acid) reacted to samples. The yellow-greenish color product obtained and concentrations were determined by spectrophotometrically at 410 nm wavelength.

Total Oxidant Capacity

TOC levels were determined in the serum using a method developed by Erel.[18] Oxidants content in serum oxidize the ferrous ion–chelator complex to ferric ion. Ferric ions generate colored solution with chromogen in an acidic media. This color changing

was determined at 660 nm wavelength. Test is calibrated with H_2O_2 standard. The results are given as µmol H_2O_2 equivalent/L.

Total Antioxidant Capacity

TAC levels were determined in the serum using a method developed by Erel.[18] Antioxidants are measured by the reduction of ABTS radical which is dark green colored. In the test, the reduced colorless ABTS form was measured. The ABTS is decolorized by antioxidants depending on their contents. This color changing was determined at 660 nm wavelength. Test is calibrated with Trolox standard. The results are given as mmol Trolox equivalent/L.

Statistical Analysis

Normality of data was evaluated by Shapiro-Wilk test and summarized by median, minimum and maximum values. Group comparisons in each time point were performed by Kruskal–Wallis test. The differences among time points in each group were analyzed by Friedman test. After both omnibus test statistics, pairwise comparisons were made by Conover method. For two group comparisons, Mann–Whitney *U* test was used. In all analysis, significant level was considered as 0.05.

RESULTS

There was no significant difference between the demographic data of all groups (Table 1).

The statistical analysis of the burn wound healing evaluation of both groups was shown in Table 2. The wound healing of the PLM group was found to be earlier than that of the HFAg group (PLM group: 13,[9–21] HFAg group: 21[12–21]).

The statistical analysis of changes on serum oxidative stress indicators in control group and treatment groups is shown in Table 3.

In PLM Group:

Serum TAC levels began to rise on day 3, continued to rise on day 7. The group in which the TAC value started to rise at the earliest and remained the longest was the PLM group. Serum TOC levels were highest on day 0, began to fall on day 3. Then, it remained low until end of study. Serum GSH level started to increase on the 3rd day, then reached the highest level on day 7, and then started to decrease on day 14 and finally to be normal at 21st day. Serum MDA levels were the highest at day 0, it started to decrease on the 3rd day, and it was at the same level as the control group at 7th, 14th, and 21st days (Table 3).

In HFAg group:

Serum TAC levels were low on day 0 and started to increase from day 3 and elevation continued on end of the study. However, this elevation is weaker than the PLM group. Serum TOC levels started to increase on day 0, reached the highest level on day 3, still high on days 7 and 14, and started to fall on day 21. However, on the 21st day, it was higher than the control group and the PLM group. Serum TOC level was only equal in the HFAg group to the PLM group on day 0. However, on days 3, 7, 14, and 21, it was higher than PLM group. Serum GSH levels were similar in the control group on day 0 and day 3. On days 7 and 14, they were higher than the control group. On day 21, there was no difference from the control group. Although the HFAg group had the highest levels of GSH on days 7 and 14, even at those days, it was lower than the level of the PLM group on same days. Serum MDA levels were the highest at day 0, it started to decrease on the third day, and it was at the same level as the control group on days 7, 14, and 21. Serum MDA levels of the HFAg group were similar from the PLM group during the study period (Table 3).

Serum TAC, TOC, GSH, and MDA levels were 0.67 (0.29–1.41), 4.55 (3.55–5.34), 96.15 (56–118.48), and 4.12 (3.2–6.02), subsequently, in the control group.

Statistical evaluation of the results was performed between the control group and two different groups treated for burns. Graphics show the course of oxidative stress indicators throughout the treatment process of the groups (Figures 1 and 2).

In this study, it was found that PLM dressing increased TAC and GSH levels in burn patients significantly more than the other groups. The TAC levels show the body's ability to resist oxidative stress. On the other hand, GSH is a component of TAC. This increase in TAC and GSH levels became apparent from the third day of treatment. The indicators of oxidative stress such as TOC and MDA, that damage the body, were significantly elevated on the first day of the burn. With the use of PLM, TOC decreased to normal level from day 3, and MDA decreased to normal level after 7 days.

In the HFAg group, TAC and GSH levels began to increase on the seventh day. From the first day of the burn, the TOC level started to increase. This increase continued on days 7 and 14. The TOC level began to fall on the 21st day. On the first day of the burn, MDA level increased and on day 7, it decreased to the same level as control group.

The PLM dressing produced a systemic response to oxidative stress in burn patients faster and stronger than HFAg treatment.

DISCUSSION

Burns are a trauma with very strong systemic effects.[4] In this study, our aim is to investigate whether this systemic effect changes different burn dressings.

After a major burn, it has been shown that severe systemic inflammatory conditions occur.[19]

The most important event that initiates local and systemic inflammation after serious burns is tissue damage.[7] Tissue destruction occurring after burns is caused by both thermal trauma and the conditions of existing oxidative stress in the environment.[12] Oxidative stress occurs when the homeostatic process fails and the formation of free radicals, which leads to cellular and tissue damage, is far beyond the defense capacity of the body.[20] This damage resulting in lipid peroxidation of cellular membranes, and may comprise DNA and protein content of cell lysis.[21] The destroyed cell particles ensure that TLR and NLRs in leukocytes are activated.[7] The activated TLR stimulates the transcription of the genes that initiate the response of inflammation, while the activated NLRS activates inflammatory capase such as caspase-1.[7] Activation of both

Table 1. Patients demographics

Variable	PLM Group (n = 20)	HFAg Group (n = 20)	Control Group (n = 20)	P
Age (year)	4.9 ± 3.80	4.85 ± 3.96)	4.90 ± 3.25	**NS**
Gender (M:F)	10/10	10/10	10/10	**NS**
Type of burn (n)	20	20		**NS**
Scald	12	13		
Flame	7	7		
Contact	1	0		
TBSA burn (%)	31.95 ± 4.43	32,15 ± 4.52		**NS**
Deep burn (%)	6.6 ± 1.27	6.5 ± 1.19		**NS**
Length of intensive care unit stay (day)	2.45 ± 0.68	2.35 ± 0.58		**NS**
Length of hospital stay (day)	23.4 ± 1.5	23.3 ± 1.8		**NS**

HFAg, hydrofiber dressing with silver; *PLM*, polylactic membrane; *NS*, non-significant.

Table 2. Statistical analysis of clinical wound healing evaluation of HFAg and PLM groups.

	Aquacel Ag (n = 20) Median (Min-Max)	Suprathel (n = 20) Median (Min-Max)	P
The time of completion of the wound epithelization (day)	21 (12–21)	13.5 (9–21)	**<.001**

HFAg, hydrofiber dressing with silver; *PLM*, polylactic membrane.

mechanisms ensures that cytokines such as TNF-α, IL-1, and IL-6 are activated.[7]

TNF-α is the most important cytokin in the inflammatory event that initiates systemic response.[22] TNF-α is a cytokin that causes the onset of metabolic and systemic inflammatory phenomena that are produced by macrophages and released into the bloodstream after minutes of tissue destruction.[22] TNF-α is a predictive value indicator in determining the development of septic complications in a burn patient.[23]

IL-1 is produced by monocytes and macrophages and allows stimulation of T cells. The stimulated T cells activate inflammation.[24]

IL-6 is produced by T cells and plays an important role in early inflammation.[22]

IL-8 is a cytokines produced by macrophages after injury and provides chemotaxis of other immune cells.[22] IL-8 level is associated with mortality in burn patients and cutting off value is 234 pg/ml.[25] The values above this value are associated with high mortality.[25]

At the beginning of the inflammation process, cytokines such as IL-10, transforming growth factor-beta (TGF-β) play an active role in the restoration of an activated inflammatory response.[26] The IL-10 and TGF-β are produced by regulatory T cells, platelets, macrophages, and lymphocytes, respectively.[26] If this restoration fails, then organ failure and death or chronic inflammation may develop.[7]

During the restoration of inflammation, protective and antioxidant mechanisms fight against oxidative stress.[27] Oxidative stress is an imbalance in which oxidants are produced at high rates in pathophysiological conditions.[27] Systemic TOC levels are significantly increased in burn patients and systemic TAC decreases significantly if not treated properly.[4]

The tripeptide GSH is composed of three amino acids: L-glutamic acid, L-cysteine (Cys), and glycine and acts as an important cellular antioxidant.[28] An increase in serum GSH levels indicates an increase in the oxidative stress response of cells.[28]

MDA, which is a highly reactive metabolite of free radical-induced lipid peroxides, is a commonly used lipid peroxide index.[29] Oxidative stress leading to peroxidation of membrane lipids produces MDA as the end product of this process.[30] An increase in MDA indicates that oxidative stress is very serious.[30] Studies have shown that MDA increases in significant amounts and that GSH decreases significantly in thermal burn patients.[4]

Various studies have been carried out to prevent the damage caused by oxidative stress resulting from severe burns.[31–34]

Pielesze et al did a local antioxidant application to prevent the negative effects of free radicals on wound healing from the burn wound. They applied ascorbic acid in the healing phase of the wound area and determined that this treatment was positively impacted by wound healing.[31]

In addition, it has been suggested that the application of β-glucan and cerium nitrate in the burn wound locally affects the healing of wounds positively by showing antioxidant effect.[32,33]

Moldanado et al suggested that systemic oxidative damage caused by the burns could be treated with systemic melatonin giving.[34]

In addition, to prevent the systemic damage caused by the burn process, the removal of free radicals from the blood circulation by hemofiltration method was found to be effective in preventing damage.[9]

Many studies have examined the effects of oral and intravenous treatments given to thermal burn patients for systemic and burn wounds.[4,5] However, no study has been conducted on the systemic effects of burn wound dressings. The burn wound is the first area where free radicals occur in the burning event.[7] Therefore, we thought that local treatments for burn wounds could have both wound healing and different systemic effects.

Table 3. Statistical analysis of changes in oxidative stress indicators for control group and treatment groups over time

	Groups	Day 0	Day 3	Day 7	Day 14	Day 21	*P* (for days)
TAC (mmol/L)	Control	0.67 (0.29–1.41)	0.67 (0.29–1.41)[a]	0.67 (0.29–1.41)[a]	0.67 (0.29–1.41)[a]	0.67 (0.29–1.41)[a]	
	Aquacel Ag	0.77 (0.16–1.92)	**1.28 (0.52–5.99)[b]**	**4.34 (1.4–14.29)[b]**	**4.52 (1.65–7.57)b**	**4.57 (1.78–5.96)[b]**	**<.001**
	Suprathel	0.74 (0.07–1.98)	**5.34 (1.21–11.62)[c]**	**14.47 (6.55–21.23)[c]**	**11.11 (4.21–45.69)c**	**11.06 (8.61–21.36)[c]**	**<.001**
P (for groups)		.828	**<.001**	**<.001**	**<.001**	**<.001**	
TOC (µmol/L)	Control	4.55 (3.55–5.34)[a]	4.55 (3.55–5.34)[a]	4.55 (3.55–5.34)[a]	4.55 (3.55–5.34)[a]	4.55 (3.55–5.34)[a]	
	Aquacel Ag	**14.35 (9.53–34.47)[b]**	**27.54 (12.83–55.86)[b]**	**16.78 (12.2–28.01)[b]**	**15.1 (4.61–25.6)b**	**6.99 (4.87–26.28)[b]**	**<.001**
	Suprathel	**12.49 (10.05–27.54)[b]**	4.35 (3.09–5.24)[a]	4.91 (3.18–13.98)[a]	4.9 (1.1–6.45)[a]	4.05 (0.01–5.93)[a]	**<.001**
P (for groups)		**<.001**	**<.001**	**<.001**	**<.001**	**<.001**	
MDA (µmol/L)	Control	4.12 (3.2–6.02)[a]	4.12 (3.2–6.02)[a]	4.12 (3.2–6.02)	4.12 (3.2–6.02)	4.12 (3.2–6.02)	
	Aquacel Ag	**8.05 (6.05–10.13)[b]**	**5.43 (4.05–7.24)[b]**	4.31 (3.04–5.1)	4.27 (3.42–6.8)	4.12 (3.48–5)	**<.001**
	Suprathel	**8.15 (6.05–10.24)[b]**	**5.08 (3.91–6.98)[b]**	4.43 (3.13–5.74)	4.02 (3.26–4.56)	3.83 (3.26–4.56)	**<.001**
P (for groups)		**<.001**	**<.001**	.642	.209	.140	
GSH (µmol/L)	Control	96.15 (56–118.48)	96.15 (56–118.48)[a]	96.15 (56–118.48)[a]	96.15 (56–118.48)[a]	96.15 (56–118.48)	
	Aquacel Ag	92.6 (8.18–118.47)	98.48 (87.89–142.01)[a]	**105.54 (85.54–199.65)[b]**	**109.65 (87.89–167.3)b**	102.01 (87.89–120.83)	**.003**
	Suprathel	94.77 (89.6–198.47)	**167.01 (87.89–292.6)[b]**	**228.77 (179.65–299.65)[c]**	**124.07 (87.89–297.3)b**	101.42 (87.89–134.95)	**<.001**
P (for groups)		.585	**<.001**	**<.001**	**<.001**	.198	

GSH, glutathione; *MDA*, malondialdehyde; *TAC*, total antioxidant capacity; *TOC*, total oxidant capacity.
Superscript letters in table represents the comparisons of groups in each time point. The difference between the groups with different superscript letters found to be statistically significant. Also, the group measurements significantly different from control group were bolded.
Data were represented as median (minimum-maximum) values.

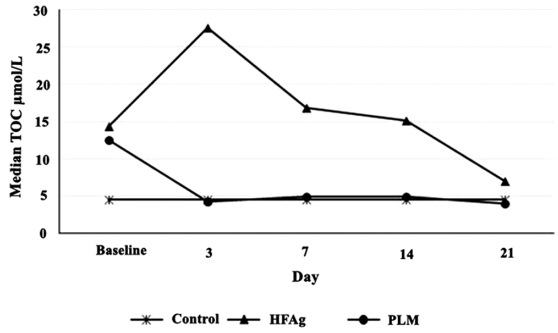

Figure 1. This picture shows the change in serum mean total oxidant capacity levels of the three groups over the course of treatment.

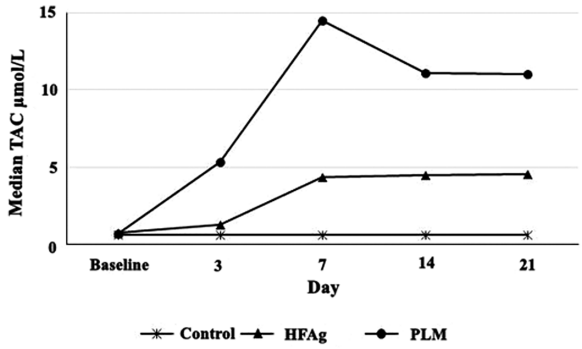

Figure 2. This picture shows the change in serum mean total antioxidant capacity levels of the three groups over the course of treatment.

Good results in wound healing have been reported by PLM treatment in thermal burns.[35] In a recent clinical study, it has been reported that PLM can be used effectively in deep burns.[35] In this study, it was claimed that PLM produces more scar tissue than autograft application and the healing process is slightly longer.[35] In the same study, it was claimed that PLM can be used effectively in large surface area burns. In another study, PLM was suggested to relieve pain in burned patients.[15] In the same study, it was found that PLM necessitated simple and useful sparse dressing.[15]

Lactate is the structural component of PLM.[15] There are important studies about the antioxidant effect of lactate in the literature.[36–38] These experimental and in vitro studies have shown that lactate has an antioxidant effect against free oxygen radicals.[36–38] They mentioned free radical scavenging effect of lactate.[36] The mechanisms underlying the antioxidant effect of lactate has not yet been clear how this effect is formed in burned tissue. It is likely that the PLM dressing has an antioxidant effect on burn tissue. We think that this effect might be created by lactate, which is contained in its content. By this antioxidant effect, we think it helps to form a strong body struggle against systemic oxidative stress. While this study could not prove the exact mechanism of this effect, PLM reduced the oxidative stress indicators and increased the body's ability to fight against oxidative stress.

The HFAg is a sterile wound dressings containing silver. Silver has been widely used in wound care for many years.[14] Silver wound dressing has a broad antimicrobial activity resulting in the inhibition of matrix metalloproteinases and proinflammatory cytokines, and a beneficial effect in the wound bed.[39] Burd et al discovered that silver wound dressings increase apoptosis and change the inflammatory process in burn wounds.[40] Silver wound dressings have been evaluated in multiple clinical studies and have been suggested to have positive effects on antimicrobial activity and wound healing.[41–43] However, these effects are all related to the wound site. No study has investigated the effects of silver wound dressings on systemic oxidative stress.

CONCLUSION

For ethical reasons, the comparison to untreated burn patients could not be done within this study. Therefore, this study was performed between burn patients who received different dressings and an unburned group.

As a result of present study, we think that different burn dressings can have some systemic effects due to the different features they have on burn tissue. We speculate that this systemic effect may be due to changes in blood levels of cytokines released from burn tissue. Although we have not proved in this study, we speculate that PLM has an antioxidant effect in the burn tissue by its lactate content. We believe that this effect increases the body's ability to fight against oxidative stress caused by burns. Therefore, PLM may have decreased serum oxidative stress indicators more effectively than HFAg. In addition, in this study, it was found that PLM wound dressing performed earlier wound healing in clinically burn wound.

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