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# Effects of high-voltage electrical burns and other burns on levels of serum oxidative stress and telomerase in children

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ABSTRACT

*Introduction:* Electrical burns cause significant morbidity and mortality worldwide. Here we measured changes in levels of serum oxidative stress and telomerase in children suffering from high-voltage electrical burn (HVEB) injuries and other burns and the significance of these parameters in terms of amputation.

*Materials and methods:* After obtaining approval from our ethics committee for this prospective study, we formed three groups: a group of 18 children with HVEBs, a group of 18 children with thermal burns, and a control group. All children were 1–16 years of age. The HVEB group was divided into HVEB-WA (without amputation) and HVEB-A (with amputation) subgroups. Serum malondialdehyde (MDA) level, total antioxidant capacity (TAC), total oxidant capacity (TOC), glutathione (GSH) level, and telomerase level were measured and compared among the groups.

*Results:* The patients differed in terms of demographics. The healing time of the HVEB group was longer than that of the thermal burn group, and the oxidative stress indicators of the HVEB group remained higher for longer. The mean oxidative stress indices in the HVEB-A group were higher than those in the HVEB-WA group and remained elevated for longer.

*Conclusion:* HVEBs are more destructive than thermal burns; damage may progress over time, and healing takes longer. Healing can be followed biochemically by measuring levels of oxidative stress indicators. Indications for amputation, if not initially obvious, can be predicted by evaluating these indicators, affording therapeutic advantages.



## 1. Introduction

Electrical burns cause significant morbidity and mortality worldwide [1]. Such injuries are less common than other burns (OBs) but are more destructive [2]. Most HVEBs occur in working adults, teenagers, and (usually older) children [1,4]. Electrical burns account for 9% of all burns [5]. An average of 1500 patients in the United States dies each year from electrical injuries [6]. Low-voltage injuries are those inflicted at 60–1000 V, usually 220 or 360 V [3]. High-voltage injuries are inflicted at >1000 V [2,3].

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The most common cause of death in patients with such injuries is cardiac arrest secondary to cardiac arrhythmia [7]. Injuries are evident at the contact area, the forearm, the joints, and the current travel route and exit area. Prevention is essential [2,8]. Here we measured changes in levels of serum oxidative stress and telomerase in patients with HVEBs and OBs and explored whether these parameters predict future amputation.

## 2. Materials and methods

### 2.1. *Study protocol*

We prospectively enrolled 18 children with thermal burns (OB) and 18 children with HVEBs who were referred to the Pediatric Burns Unit of İnönü University School of Medicine from January 2015 to January 2018. The two groups contained equal numbers of boys and girls, and all children were ages 1–16 years (Fig. 1). We subdivided the HVEB group into HVEB-A (with amputation) and HVEB-WA (without amputation) subgroups. Demographic data are listed in Table 1. All children suffered acute burns affecting 20–50% of the total body surface area (TBSA), including deep burns to 5–10% of the TBSA. All high voltage electrical burns were caused by high voltage wire contact. In all of these patients, the burned areas were in multiple areas of their bodies. The control group consisted of normal children (brothers and sisters of patients) of an equal sex ratio also ages 1–16 years. Patients admitted later than 24 h after injury, those with a history of or current gastrointestinal disease, those with chronic diseases such as diabetes, and those with burns of the upper respiratory tract or inhalation injuries were excluded from the study. Patients who did not develop sepsis or renal insufficiency during treatment were included.

After approval was obtained from the İnönü University Clinical Research Ethics Committee, informed consent forms were completed by parents. The respiratory and circulatory systems were checked and necessary measures taken. The affected proportion of the TBSA was calculated and fluid treatment was commenced. A Foley catheter was placed in the bladder and fluid therapy was controlled to a urine output of 1–2 mL/h. HVEB patients received fluid therapy and standard treatments; cardiac markers were also measured. Cardiology consultations were requested when indicated and the suggested interventions performed. The levels of fluid given to patients exhibiting myoglobinuria were increased appropriately. Electrocardiography was performed. Limb circulation was closely evaluated. Doppler ultrasonography, angiography, and escharotomy were performed as indicated.

All patients were fed as directed by a nutritionist who considered age, weight, and the burnt area. All patients received 1 mg/kg/day each of paracetamol (Parol 500 mg intravenous (IV), Flakon; Atabay, Istanbul, Turkey) and omeprazole (Losec 20 mg IV, Flakon; AstraZeneca, Istanbul, Turkey) for the first 48 h. Early enteral feeding featured an oral caloric intake of 35 kcal/kg/day, of which 20% was protein. Then 24 h after admission, patients exhibiting normal urine output were given albumin if indicated. The food contained no fibers or fermented foodstuffs. All burns were thoroughly and gently cleaned by wiping with serum/saline-treated sterile gauze. The wounds were debrided with a surgical sponge, rinsed, and dried. We followed the basic principles of wound healing. Early debridement was performed to remove necrotic tissue. Some HVEB patients underwent amputations under general anesthesia (Fig. 2,Table 2).

All wounds were dressed every 3 days with antiseptic gauze containing silver sulfadiazine (PansAG; Velfina, Campulung, Romania). Patients were prepared for excision and autografting 2 weeks after admission. All procedures were performed by the same surgical/anesthetic team. A thin slice (0.005–0.010 in) of autologous healthy skin was harvested after debridement of the burn scar tissue. The size of the donor skin depended on the area to be grafted. We used a Humeca electrical dermatome (Humeca Skin Transplantation Technology, Borne, The Netherlands) to this end. When necessary, the graft was expanded with an external skin expander (Brennen Skin Graft Mesher; Molnycke H.C., Norcross, GA, USA) before placement.

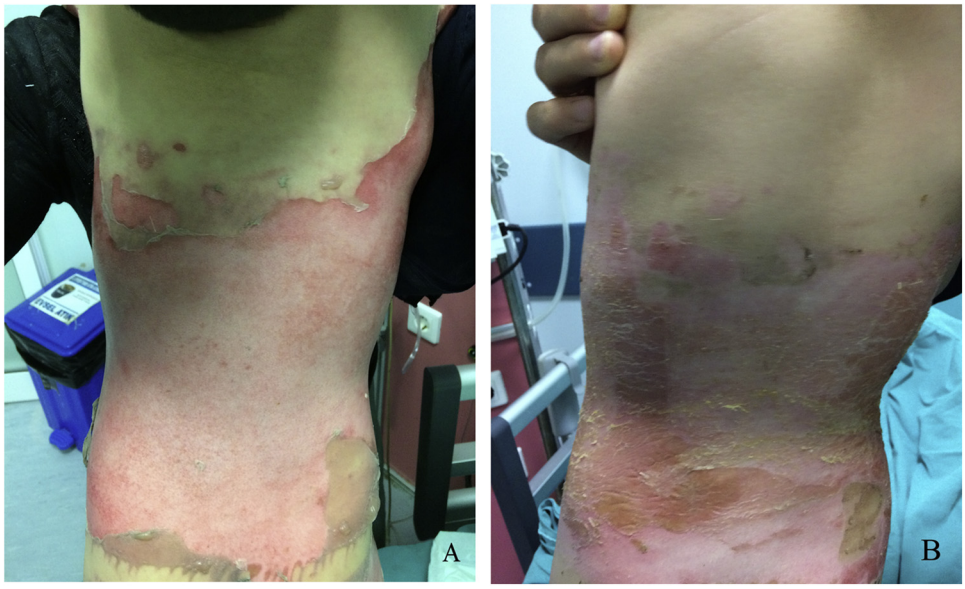


**Fig. 1 – (A) Day 1 image of a 3-year-old boy with a 35% scald burn (an OB). (B) Day 18 image after standard burn treatment.**

**Table 1 – Patient demographics.**

| Variable | OB group (n=18) | HVEB group (n=18) | HVEBA group (n=8) | Control group (n=18) |
|---|---|---|---|---|
| Age (years) | 13±2.9 | 12.1±3.7 | 13.7±1.8 | 13.2±2.4 |
| Gender (M:F) | 9:9 | 14:4 | 7:1 | 9:9 |
| Type of burn (n.%) | | | | |
| Scald | 9 | 0 | 0 | |
| Flame | 8 | 0 | 0 | |
| Contact | 1 | 0 | 0 | |
| Electrical | | 18 | 8 | |
| TBSA burn (%) | 32.5±4.28 | 32,2±2,55 | 33,1±2,58 | |
| Deep burn (%) | 10.5±2.6 | 11.05±3.01 | 13.75±2.31 | |
| Length of intensive care unit stay (day) | 3.72±0.75 | 5.27±0.66 | 5.3±0.74 | |
| Length of hospital stay (day) | 25.27±2.58 | 60.33±20.10 | 78.37±7.15 | |

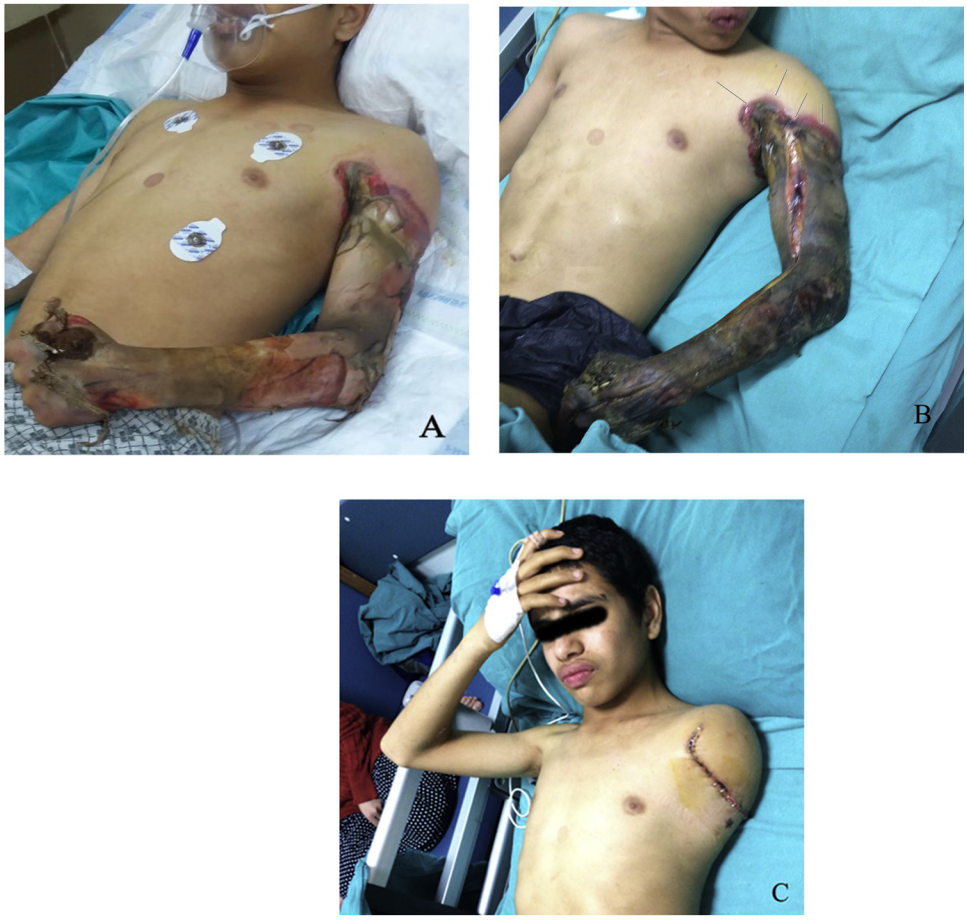


**Fig. 2 – (A) Day 1 image of a 16-year-old male with an HVEB. No demarcation line is visible yet. (B) On day 21, a demarcation line appears (arrows). (C) An image after amputation (day 24).**

**Table 2 – Summary of amputations.**

| Patient no. | Voltage | Extremity involved | Amputated structure | Amputation time (day) |
|---|---|---|---|---|
| 1 | High | Left lower limb | Below the ankle | 45 |
| 2 | High | Right lower limb | Toe | 59 |
| 4 | High | Right upper limb | Below the ankle | 23 |
| 5 | High | Left lower limb | Below the ankle | 22 |
| 8 | High | Right upper limb | Right forearm | 24 |
| 12 | High | Right upper limb | 1st, 2nd, 3rd, 4th, toes | 30 |
| 15 | High | Left upper limb | Left forearm | 77 |
| 18 | High | Left upper limb | Left forearm | 22 |

All patients were subjected to routine weekly examinations (full blood count, blood culture, plain chest X-ray, liver function test, arterial blood gas evaluation, and coagulation profile).

### 2.2. Blood samples

Blood samples were taken from both burn groups on days 0, 3, 7, 14, 21, and 28 after admission and once in the control group. On the days on which blood samples were taken, burn patients did not receive albumin. For patients hospitalized >28 days, blood was taken weekly and on the day of discharge. Tubes containing blood were stored at −80°C prior to analysis, warmed to 23°C on the day of analysis, and immediately centrifuged at 4000 rpm for 7 min. Sera were collected for analyses of malondialdehyde (MDA) level, glutathione (GSH) level, telomerase level, total antioxidant capacity (TAC), and total oxidant capacity (TOC).

### 2.3. Serum telomerase levels

We analyzed human serum telomerase levels by ELISA using a commercial kit (Cat. No: E-EL-H0164 96T, Elabscience Biotechnology). We measured absorbance was measured a BioTek Synergy H1 microplate reader (BioTek Instruments).

### 2.4. Malondialdehyde levels

We measured MDA using Uchiyama et al.'s [9] method. MDA is a byproduct of lipid peroxidation and reacts with thiobarbituric acid at 95°C. MDA was extracted into *n*-butanol and the pink product quantified by absorbance at 532 nm.

### 2.5. Glutathione levels

We measured GSH using Elman's [10] method (via reaction with 5,5′-dithiobis-2-nitrobenzoic acid). The yellow-greenish product was quantified by absorbance at 410 nm.

### 2.6. Total oxidant capacity

We measured serum TOC using Erel's [11] method. Oxidants in serum oxidize a ferrous ion–chelator complex to ferric ion, which then reacts under acidic conditions to generate a chromogen. This chromogen was quantified by absorbance at 660 nm. The test was calibrated using $H_2O_2$, and the results are given as μmol $H_2O_2$ equivalents/L.

### 2.7. Total antioxidant capacity

We measured serum TAC using Erel's [11] method. Antioxidants reduce the dark-green ABTS radical to a colorless form. Color change was measured at 660 nm. Trolox served as the calibrating standard, and the results are given as mmol Trolox equivalent/L.

### 2.8. Statistical analyses

We evaluated the normality of the distribution using the Shapiro–Wilk test. If the data were normally distributed, we calculated means and standard deviations; otherwise, we calculated medians with interquartile ranges. The homogeneity of variance among groups was explored using Levene's test. If the variance was homogeneous, we used one-way ANOVA and Tukey's HSD *post hoc* test for analyses; otherwise, we used the Welch test and the Tamhane T2 *post hoc* test. The Kruskal–Wallis test and the Conover *post hoc* test was used to evaluate nonnormally distributed data. The Mann–Whitney U test was used to compare test and control groups at all time points. We compared mean values before and after amputation using the Wilcoxon signed rank test. Categorical data are presented as frequencies with percentages. Pearson's exact test or Fisher's exact test was used to compare these data. $p<0.05$ was considered to reflect statistical significance.

## 3. Results

### 3.1. Demographics

Age did not differ significantly among the groups ($p>0.05$). The mean length of stay in the intensive care unit was longer in HVEB-WA and HVEB-A patients (5 and 5.5 days, respectively) and was 4 days in the OB group [4]. The average length of stay in the hospital differed among the groups, being 25 days in the OB group, 45 days in the HVEB-WA group, and 78.5 days in the HVEB-A group. The mean burnt TBSA (%) did not differ significantly among the groups ($p>0.05$). In terms of mean burn depth, the OB group (10 mm) and HVEB-WA group (9 mm) did not differ ($p>0.05$), but the burn depth of the HVEB-A group [15] was greater ($p<0.05$). The demographic data are compared in Table 3.

### 3.2. Serum components

Mean serum telomerase, GSH, and MDA levels and the TOC and TAC of all groups were compared (Table 4). Mean serum

**Table 3 – Statistical analyses of demographic data.**

| | Control (n=18) | | OB (n=18) | | HVEBA (n=8) | | HVEB (n=10) | | p |
|---|---|---|---|---|---|---|---|---|---|
| | Median | IQR | Median | IQR | Median | IQR | Median | IQR | |
| Age (years) | 14 | 3,5 | 14 | 6,25 | 14 | 3,5 | 11 | 8,75 | 0,481 |
| Length of intensive care unit stay (day) | | | 4[a] | 1 | 5,5[b] | 1 | 5[b] | 1 | **<0,001** |
| Length of hospital stay (day) | | | 25[a] | 3,25 | 78,5[b] | 13 | 45[c] | 26 | **<0,001** |
| TBSA burn (%) | | | 30 | 5 | 35 | 5 | 30 | 5 | 0,457 |
| Deep burn (%) | | | 10[a] | 3,25 | 15[b] | 3,75 | 9[a] | 2 | **0,002** |

The difference between the groups with different superscript letter is found to be statistically significant.

**Table 4 – Comparisons among the three groups.**

| | Group | N | Mean | Std. deviation | p |
|---|---|---|---|---|---|
| Telomerase (ng/mL) | Control | 18 | 2,76[a] | 0,94 | <**0,001** |
| | OB | 18 | 10,79[b] | 2,37 | |
| | HVEB | 18 | 18,96[c] | 2,80 | |
| TAC (mmol/L) | Control | 18 | 0,75[a] | 0,30 | <**0,001** |
| | OB | 18 | 4,67[b] | 1,03 | |
| | HVEB | 18 | 7,44[c] | 1,13 | |
| TOC (μmol/L) | Control | 18 | 2,70[a] | 0,55 | <**0,001** |
| | OB | 18 | 9,10[b] | 1,63 | |
| | HVEB | 18 | 9,69[b] | 1,19 | |
| MDA (μmol/L) | Control | 18 | 1,37[a] | 0,74 | <**0,001** |
| | OB | 18 | 4,24[b] | 0,37 | |
| | HVEB | 18 | 5,26[c] | 0,60 | |
| GSH (μmol/L) | Control | 18 | 92,85[a] | 14,06 | <**0,001** |
| | OB | 18 | 116,18[b] | 12,30 | |
| | HVEB | 18 | 120,66[b] | 6,60 | |

The difference between the groups with different superscript letters is found to be statistically significant.

telomerase levels (ng/mL) were 2.76 (IQR=0.94) in the control group, 10.79 (IQR=2.37) in the OB group, and 18.96 (IQR=2.80) in the HVEB group. The differences were statistically significant. Mean serum TAC (mmol/L) was 0.75 (IQR=0.75) in the control group, 4.67 (IQR=1.03) in the OB group, and 7.44 (IQR=1.13) in the HVEB group. The differences were statistically significant. Mean serum TOC (μmol/L) was 2.70 (IQR=0.55) in the control group, 9.10 (IQR=1.63) in the OB group, and 9.69 (IQR=1.19) in the HVEB group. The values of the OB and HVEB groups were both significantly higher than that of the control group, but the two former groups did not differ. Mean serum MDA levels (μmol/L) were 1.37 (IQR=0.74) in the control group, 4.24 (IQR=0.37) in the OB group, and 5.26 (IQR=0.60) in the HVEB group. The differences were statistically significant. Mean serum GSH levels (μmol/L) were 92.85 (IQR=14.06) in the control group, 116.18 (IQR=12.30) in the OB group, and 120.66 (±6.60) in the HVEB group. The values of the OB and HVEB groups were both significantly higher than that of the control group, but the two former groups did not differ.

### 3.3. *Times to normalization of serum markers*

The times to normalization of serum markers are shown in Tables 5 and 6. In the HVEB-WA group, the serum TOC and the GSH level normalized at day 21, the serum telomerase level normalized at day 28, the serum MDA level normalized at day 49, and the serum TAC normalized at day 77. In the OB group, the serum telomerase, MDA, and GSH levels normalized at day 21; the serum TOC normalized at day 28; but the serum TAC remained higher than that of the control group at day 28.

### 3.4. *Comparison of the HVEB-WA and HVEB-A groups*

The HVEB group (18 patients) was divided into HVEB-WA (n=10) and HVEB-A (n=8) subgroups. The serum parameters over the first 28 days were compared (Table 7). Mean serum telomerase levels (ng/mL) over the first 28 days were 20.60 (±2.97) in the HVEB-WA group and 34.51 (IQR=5.45) in the HVEB-A group; the difference was significant. Mean serum TAC over the first 28 days was 7.29 (±0.70) in the HVEB-WA group and 13.04 (±3.25) in the HVEB-A group; the difference was significant. Mean serum TOC over the first 28 days was 9.60 (±1.82) in the HVEB-WA group and 16.45 (±2.99) in the HVEB-A group; the difference was significant. Mean serum MDA levels (μmol/L) over the first 28 days were 5.66 (±0.35) in the HVEB-WA group and 7.35 (±0.80) in the HVEB-A group; the difference was significant. Mean serum GSH levels (μmol/L) over the first 28 days were 122.36 (±10.54) in the HVEB-WA group and 145.19 (±9.97) in the HVEB-A group; the difference was significant (Fig. 3).

## 4. Discussion

OBs are more common than HVEBs in children [7]. Approximately 9% of all burns are electrical burns, which differ from other burns [5]. Although electrical burns initially seem to burn only small areas of skin, textural destruction becomes obvious over time. The destroyed area is much larger and deeper than the initial appearance suggests [7]. The extent of tissue damage depends on the current delivered, tissue resistance, and exposure time [12]. When tissue resistance is high, high voltage raises the temperature of the tissue [7]. The TAC increases significantly over the first 24 h in patients with severe burns [13], which is associated with oxidative stress [13]. Both physical protection and antioxidative mechanisms can be used to counteract these harmful effects. Oxidative stress reflects high-level, pathophysiological production of oxidants [14]. Telomerase prevents senescence by stabilizing chromosomal telomeres [15,16]. An increase in serum telomerase levels is an indication of cellular damage and oxidative stress [17]. However, no study has yet investigated serum telomerase activity in patients with thermal or electrical

**Table 5 – Days on which HVEB group parameters became normalized.**

| | Control | | | HVEB | | | p |
|---|---|---|---|---|---|---|---|
| | n | Median | IQR | n | Median | IQR | |
| TOC (μmol/L) day 21 | 18 | 2,86 | 0,82 | 18 | 4,18 | 13,66 | 0,126 |
| GSH (μmol/L) day 21 | 18 | 93,48 | 14,96 | 18 | 108,77 | 42,00 | 0,055 |
| Telomerase (ng/mL) day 28 | 18 | 2,77 | 1,48 | 18 | 2,37 | 10,60 | 0,839 |
| MDA (μmol/L) day 49 | 18 | 1,47 | 0,94 | 8 | 2,77 | 3,48 | 0,102 |
| TAC (mmol/L) day 77 | 18 | 0,75 | 0,45 | 4 | 1,20 | 4,93 | 0,066 |

**Table 6 – Days on which OB group parameters became normalized.**

| | Control | | | OB | | | p |
|---|---|---|---|---|---|---|---|
| | n | Median | IQR | n | Median | IQR | |
| Telomerase (ng/mL) day 21 | 18 | 2,77 | 1,48 | 18 | 3,19 | 2,99 | 0,584 |
| MDA (μmol/L) day 21 | 18 | 1,47 | 0,94 | 18 | 1,31 | 1,50 | 0,988 |
| GSH (μmol/L) day 21 | 18 | 93,48 | 14,96 | 18 | 102,01 | 10,59 | 0,051 |
| TOC (μmol/L) day 28 | 18 | 2,86 | 0,82 | 18 | 2,22 | 1,30 | 0,004 |
| TAC (mmol/L) day 28 | 18 | 0,75 | 0,45 | 18 | 1,26 | 1,57 | 0,014 |

**Table 7 – The 28-day averages of relevant parameters in the HVEB-WA and HVEB-A groups.**

| | HVEB (n=10) | | HVEBA (n=8) | | p |
|---|---|---|---|---|---|
| | Median | IQR | Median | IQR | |
| Telomerase (ng/mL) | 20,60 | 2,97 | 34,51 | 5,45 | **<0,001** |
| TAC (mmol/L) | 7,29 | 0,70 | 13,04 | 3,25 | **<0,001** |
| TOC (μmol/L) | 9,60 | 1,82 | 16,45 | 2,99 | **<0,001** |
| MDA (μmol/L) | 5,66 | 0,35 | 7,35 | 0,80 | **<0,001** |
| GSH (μmol/L) | 122,36 | 10,54 | 145,19 | 9,97 | **<0,001** |

burns. Systemic total oxidant capacity (TOC) increases significantly in burn patients, whereas systemic total antioxidant capacity (TAC) decreases significantly [18]. The TOC reflects the extent of attack during oxidative stress, and the TAC the capacity to resist such attack [18].

An increase in the level of MDA, which is created when free radicals peroxidize lipids [19], indicates that the level of oxidative stress is very high [20]. MDA levels increase significantly and GSH levels decrease significantly in patients with thermal burns [18]. The tripeptide GSH is an important cellular antioxidant [21]. An increase in the serum GSH level indicates an increase in the oxidative stress response [22].

Electric burns are divided into high-voltage (>1000 V) and low-voltage (<1000 V) burns [23]. All of our patients had high-voltage burns. The reported amputation rate associated with all electrical burns is 29% [24] but was higher (44%) in our study because all patients had HVEBs. Initially the burns appeared to be small [6], but further damage developed after days or even months despite early treatment and debridement (Fig. 4). We measured oxidative stress weekly. Biochemical healing was complete in 28 days in OB patients but not until day 49 in HVEB patients. Early amputation was sometimes necessary, and levels of serum

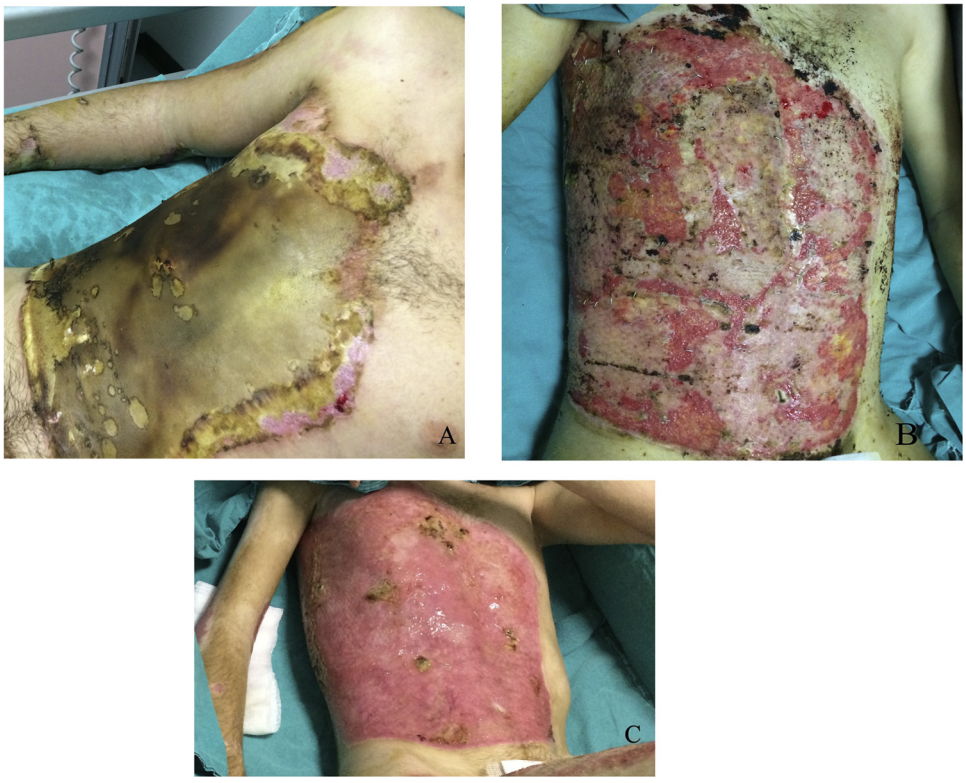


**Fig. 3 – (A) Day 1 image of a 16-year-old male with a 35% HVEB. (B) Day 22 image taken after autografting. (C) Recovery is evident on day 45.**


**Fig. 4 – (A) Day 68 image of the left hand of a 12-year-old who suffered a 30% HVEB. (B) Day 77 image before amputation. (C) Image after amputation. (D) On day 73, the left radial artery is open but no flow is detected because of distal extravasation (arrows).**

oxidative stress (markers of biochemical healing) then recovered rapidly in most patients (Fig. 5). In one patient, however, biochemical healing continued to day 77; levels of serum oxidative stress remained high throughout this time. Although arterial flow was evident on Doppler ultrasonography performed a few days after injury, arterial extremity angiography performed on day 77 revealed no flow although the artery was open (Fig. 4D). This patient's level of oxidative stress returned to that of the controls after amputation.

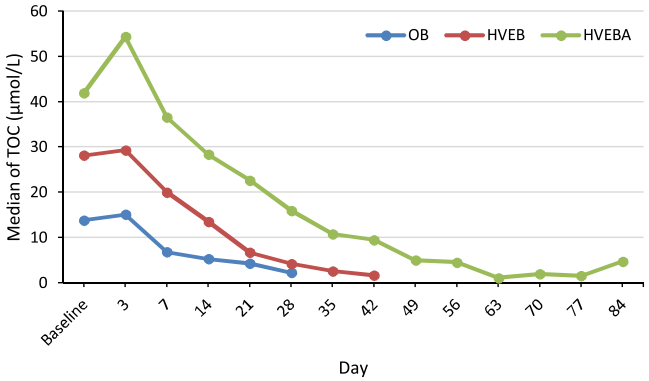


**Fig. 5 – Change in mean serum TOC (μmol/L) over time in the OB, HVEB-WA, and HVEB-A groups from day 1 to the day of discharge.**

Researchers have studied patients with HVEBs with regard to the extent of amputation, the distribution of burns, and affected areas [24]; biochemical parameters have not been evaluated. Here we found that parameters of oxidative stress were higher in those with HVEBs than OBs both at baseline and later and that higher values predicted indications for amputation.

## 5. Conclusion

HVEBs are more destructive than OBs and take longer to heal. We believe that the monitoring of oxidative stress indications in high voltage electrical burns every week since the first application of the patient may give more realistic information in terms of amputation and clinical course in the follow-up of these patients and provide a therapeutic advantage. Other laboratory parameters may not reflect this, even if the burning process continues for weeks afterwards. This can be followed biochemically by measuring the oxidative stress indicators.

## Conflict of interest

None of the authors has any commercial associations that might pose or create a conflict of interest with information presented in this article. No intramural or extramural funding supported any aspect of this work.

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