AGE ESTIMATION FOR SCALD INJURY AND ITS PROBABLY RELATION TO TESTICULAR FUNCTION IMPAIRMENT

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ABSTRACT

Background and Rationale: From the medicolegal point of view, long term systemic consequences of scald injury should be identified for proper compensation and legal action, especially for scald injury involving skin full-thickness in 20% of total body surface area (TBSA) which is known to have long term grave consequences on general health. Aim of the Study: The current experimental study was carried out to: 1- identify the histopathological changes and TNF-α Immunoexpression of different ages of a full thickness scald injury occupying 20% of the total body TBSA; 2- study the probable relation between the scald age and the testicular function impairment. Materials and Methods: A total number of 40 adult male albino rats were used in the study. Histopathological examination for the scald area and testis were performed with hematoxyline & eosin stain and TNF-α Immunoexpression. Epididymal semen analysis and serum testosterone were also performed. These procedures were carried out after 2 days, 7 days, 1 month and 3 months of scald infliction. Results: Progressive histopathological changes were observed in the early scald ages while healing manifestations and improvement in testosterone serum levels, when compared to the other periods, started to appear after 1 and 3 months of scald infliction. Conclusion: It can be concluded that scald injury involving 20% of TBSA can cause long term impairment of testicular function.

Keywords: Scald; TNF-α; histopathology; Testicular function

INTRODUCTION

Worldwide the circumstances surrounding scald assault and burning fall into three main categories: domestic maltreatment, elder abuse, or conflicting business affairs (Peck, 2012), where in most cases the assailant is known to the victim which allows him to be at a near distance from the victim (Dorn et al., 2001).

The incidence of burn injury is not specific to any population or age group, but the forensic medicine is concerned if the occurrence of burns has been attributed to abuse, ill-treatment, neglect or torture (Huang et al., 2008; Pollanen, 2018).

According to Adeteye et al. (2011), severity is related to burn depth and percentage of the total body surface area (TBSA), where 3rd degree is a full thickness burn destroying all the skin layers till below hair follicles, sweat glands and subcutaneous fat tissue, accordingly, it is usually not painful due to destruction of the nerve endings.

As stated by Agay et al. (2008), a scald burn is a type of tissue damage from any hot liquid or fluids as oils, steam and molten rubber, and however, scalding by water is a common domestic accident, especially to children and old people, who are vulnerable to many types of accidents.
Scalding burns are of three types: immersion in a hot liquid whether accidental or deliberate, splash (or spill) burns which is usually accidental, and steam burns i.e. exposure to superheated steam. Hot water is found to be the most common cause of the immersion, spill, and splash burns (Jewo et al., 2010).

It was proven that burn injury caused changes in the endogenous production of cytokines, adrenal and gonadal steroids, where previous studies have reported sex-related differences in the outcome following burn injury (Bergquist et al., 2016). Cytokines are important mediators in post-burn pathophysiological process (Agay et al., 2008). This enhanced production of inflammatory cytokines, such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), interleukin 1beta (IL-1β), and prostaglandins may cause a failure of different organ systems, at least in part, due to increased apoptotic cell death and (Gatson et al., 2009), where tumor necrosis factor-alpha (TNF-α), as one of the proinflammatory and immunoregulatory cytokines, exhibits a surge particularly in the early stage of the wound healing process (Bai et al., 2008).

The current experimental study was carried out to: 1- identify the histopathological changes and TNF-α Immunoeexpression of different ages of a full thickness scald injury occupying 20% of the total body TBSA; 2- study the probable relation between the scald age and the testicular function impairment.

MATERIALS AND METHODS

MATERIALS

1. Animals:

- Experimental Design

Forty adult albino rats were used in the study. Eight albino rats formed the control group which received only regular diet and tap water to measure the basic parameters. The remaining 32 rats were subjected to a model for scald injury to be sacrificed by cervical dislocation (Tomita et al., 2004) and studied at 4 different scald ages: after 2 days, 7 days, 1 months and 3 months of injury infliction (8 albino rats/scald age).

This study was done after taking acceptance of Institution Review Board (IRB) of Faculty of Medicine, Zagazig University. The 40 adult male albino rats were obtained from the animal house of the Faculty of Veterinary Medicine, Zagazig University; their weights ranged between 290 and 310 gm. The guidelines stated in "The Guide for the Care and Use of Laboratory animals" (Institute of laboratory animals resources, 1996), were used to care for the experimental animals used in the current study.

2. Chemicals:

Sodium citrate solution (2.9-3%) and physiological saline solution (0.9%) were used for epididymal spermatozoal examination. Ketamine hydrochloride and diazepam were used in the burn model. All chemicals were obtained from El- Nasr Co., Egypt.

METHODS

1. Scald model:

Each rat was anesthetized, and the analgesic was given with intraperitoneal injection of ketamine hydrochloride (20 mg/kg BW) and diazepam (0.1 mg/kg BW). The back and flank skin of the rats was shaved. Rats were placed in supine position in a plaster cast exposing an area in their backs through an opening in the cast, then immersed in a hot water bath (100°C) for 10 seconds (Jewo et al., 2011). This should produce a nonlethal full thickness injury to the skin that covers 20% of the total body surface area which was calculated by using the formula of Lee which is: Total body surface area (TBSA) = (body weight in grams x 0.78) + 148 (Gouma et al., 2012). TBSA of a rat
weighing 300 grams is 382 cm$^2$. So that 20% of TBSA equals 76.4 cm$^2$.

2. Histopathological Examination:
Light microscopic examination was performed to detect histopathological changes of the skin with scald injury and the testis. After 2 days, 7 days, 1 month and 3 months of scald infliction, the scald area in the skin was immediately dissected out and fixed in Bouin's solution (Prophet et al., 1992), while testicular specimens were fixed in 10% formalin saline. After fixation, specimens were embedded in paraffin blocks and processed for the preparation of sections in 5 μ thickness. These sections were subjected to:

1- Hematoxylin and Eosin staining according to the method described by Wilson & Gamble (2002).

2- TNF-α Immunohistochemical detection according to the method described by Carreiraa et al. (2012). Staining was considered positive if the tissue demonstrated brown staining.

3. Epididymal Sperm Analysis:
Spermatozoa collection was done as described by Klinefelter et al. (1991). Epididymal content was obtained by cutting the tail of epididymis and squeezing it gently to get the fresh undiluted semen in a clean Petri dish to perform the following examinations:

1- Sperm motility:
Sperm motility was estimated by mixing an undiluted semen droplet to a drop of sodium citrate solution 2.9-3% on warm slide, several fields were inspected under light microscope, then the incidence of progressive motile sperms were counted and recorded (Tardif et al., 1999).

2- Sperm count:
Sperm cell concentration was determined by withdrawing undiluted semen up to the mark 0.1 and a hemocytometer pipette was filled up to the level 101 by normal saline, then stained with eosin, and shook vigorously. A drop of diluted semen was spread between the haemocytometer chambers after placing over the counting chamber cover slide. Using the high power lens of light microscope (40x), the sperms in 5 large squares (80 small squares) were counted. The sperm cell concentration is calculated by multiplying the number of sperms by 100 (depth) and 1000 (dilution) (Blazak et al., 1993).

3- Sperm abnormal forms:
Two equal drops of epididymal content and eosin-nigrosin stain were mixed, and spread on clean and grease free slides. Two hundred sperms were randomly examined for each rat samples under the light microscope high power lens to record both the percentages of abnormal sperms and the abnormal forms. Moraes’s classification was used to describe the sperm abnormal forms observed in the current study (Moraes et al., 2008), which were classified mainly into: sperms with deformed or absent tails (tailless sperm) and sperms with abnormal heads.

4. Biochemical Measurement for Testosterone Hormone:
Venous blood samples were collected from the retro-orbital plexus of the animals by capillary glass tubes using light ether anaesthesia according to procedure described by Joslin (2009). According to the, manufacturer's guidelines, for each animal, not less than 2 ml of blood was collected into a glass tube for the quantitative measurement of testosterone in rat and mouse by ELISA.

5. Statistical Analysis:
For statistical analysis, SPSS 13.0 for windows programme was used. Data was represented in terms of means ± SD. The differences were compared for statistical
RESULTS

Histopathological Examination

Skin Examination (Table-1)

In H & E skin sections (Figure-1), control group showed intact dermal epidermal layers with normal adnexal structures after 2 days, 7 days, 1 month and 3 months. In Scald injured group, skin with scald injury revealed ulceration of the epidermis and dermal inflammatory infiltrate that invade muscles in some area by the end of the 2nd day of scalding injury. By the end of 7th day, there was destruction of adnexal structures, and area of coagulative necrosis invaded by inflammatory cells.

By the end of 1st month, a decrease in the acute inflammatory response findings were observed with deposition of multiple collagen layers. By the end of 3 months, re-epithelialization was observed in the form of multiple epithelial cells islands migrating towards the wound surface from underlying dermal appendages.

For TNF-α immunohistochemical expression (Figure-2), it was negative in the dermis, endothelial cells and perivascular cells of the skin of the control group after 2 days, 7 days, 1 month and 3 months. In Scald injured group, skin showed a positive expression of TNF-α in the dermis, endothelial cells and perivascular cells after 2 days and 7 days. By the end of 1 month and 3 months, there were a recorded improvement as all skin specimens of the rats of both groups showed a negative expression of TNF-α in the dermis, endothelial cells and perivascular cells, respectively.

Testes (Table-2)

In H & E sections (Figure-3), the testes of the control group showed normal cell arrangement and the lumen is full of mature sperm cells after 2 days, 7 days, 1 month and 3 months. In scald injured group, microscopic examination of the testes revealed reduction of number of germ cell layers and dissociation of the germ cells from the tubular basement membrane by the end of the 2nd day of burn application. By the end of 7th day of burn application, there was germ cell atrophy, absence of free spermatzoa, the tubules showed scanty cells even in their basal areas which lined only by Sertoli cells. By the end of 1st month the same histopathological changes as those described by the end of the 7th day were noted. Widespread cyto-architectural disruption of the seminiferous tubules, the tubules lined only by Sertoli cells. By the end of 3 months microscopic examination of testes showed considerable seminiferous tubular damage.

For TNF-α immunohistochemical expression (Figure-4), the control group showed normal appearance of testes specimens with negative expression of TNF-α in the cytoplasm of spermatogonia, spermatocytes and the interstitial leydig cells after 2 days, 7 days, 1 month and 3 months. In Scald injured group, the testes showed a positive expression of TNF-α in the cytoplasm of spermatogonia, spermatocytes and the interstitial leydig cells by the end of the 2nd and 7th days, while by the end of 1 month and 3 months, a recorded improvement was detected with a negative expression of TNF-α in the cytoplasm of spermatogonia, spermatocytes and the interstitial leydig cells.

Epididymal Sperm Analysis (Tables-2, 3, Figure-5)

Scald injury was associated with significant decreases (p<0.05) in the mean values of epididymal sperm analysis parameters (sperm count (106/mm3), the percent (%) of sperm motility, and abnormal forms) after 1 and 3 months of injury infliction. Abnormal sperm forms...
were: coiled tailed, bent tail, 2-tailed and flat head. Scald injuries of 2 and 7 days of age were not associated with any significant changes (p>0.05) in the mean values of epididymal sperm analysis parameters compared to the control group mean values on one side, and between one another on the other side.

- Testosterone Measurements (Table-4)
Scald injury was associated with progressive significant decreases (p<0.05)

Table-1: Characteristic light microscopic findings observed in skin in terms of absent (-), mild (+), moderate (+++) and severe (++++) after 2 days, 7 days, 1 month and 3 months from exposure to antemortem scald injury in adult male albino rats

<table>
<thead>
<tr>
<th>Light Microscopic Finding</th>
<th>Age of Scald Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2 Days</td>
</tr>
<tr>
<td>Destruction of Adnexal Structures</td>
<td>+</td>
</tr>
<tr>
<td>Ulceration in the Epidermis</td>
<td>+</td>
</tr>
<tr>
<td>Inflammatory Infiltrate in the Dermis</td>
<td>++</td>
</tr>
<tr>
<td>Deposition of Multiple Collagen Layers</td>
<td>-</td>
</tr>
<tr>
<td>Re-epithelialization</td>
<td>-</td>
</tr>
<tr>
<td>Multiple Cell Islands</td>
<td>-</td>
</tr>
<tr>
<td>Epithelial Cell Migration to the Scald</td>
<td>-</td>
</tr>
<tr>
<td>TNF-α Immunoexpression</td>
<td>+++</td>
</tr>
</tbody>
</table>

Table-2: Characteristic light microscopic findings observed in the testis in terms of absent (-), mild (+), moderate (+++) and severe (++++) after 2 days, 7 days, 1 and 3 months from exposure to antemortem scald injury in adult male albino rats

<table>
<thead>
<tr>
<th>Light Microscopic Finding</th>
<th>Age of Scald Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2 Days</td>
</tr>
<tr>
<td>Reduction in Germ Cell Layers Number</td>
<td>+</td>
</tr>
<tr>
<td>Dissociation of Germ Cells</td>
<td>+</td>
</tr>
<tr>
<td>Germ Cell Atrophy</td>
<td>-</td>
</tr>
<tr>
<td>Absent Free Spermatozoa</td>
<td>-</td>
</tr>
<tr>
<td>Scanty Sertoli Cells Layer</td>
<td>-</td>
</tr>
<tr>
<td>Tubular Cyto-architectural Disruption</td>
<td>-</td>
</tr>
<tr>
<td>Sperm Cell Count</td>
<td>-</td>
</tr>
<tr>
<td>Decreased Sperm Motility</td>
<td>-</td>
</tr>
<tr>
<td>Presence of Sperm Abnormal Forms</td>
<td>-</td>
</tr>
<tr>
<td>TNF-α Immunoexpression</td>
<td>+++</td>
</tr>
</tbody>
</table>
**Table-3:** Mean values of epididymal sperm analysis (sperm count in 106/mm3, sperm motility percent and percent of abnormal sperm forms) of control and scald injury groups after 2 days, 7 days, 1 month and 3 months

<table>
<thead>
<tr>
<th>Testicular Function Parameters</th>
<th>Control Group</th>
<th>Scald Injury Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>After 2 days</td>
<td>After 7 days</td>
</tr>
<tr>
<td><strong>Epididymal Sperm Analysis</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sperm Count (10^6/mm^3)</td>
<td>118.85 ± 2.4</td>
<td>117.6 ± 2.4</td>
</tr>
<tr>
<td>Epididymal Sperm Motility (%)</td>
<td>85.3 ± 1.7</td>
<td>82.9 ± 2.2</td>
</tr>
<tr>
<td>Sperm Abnormal Forms (%)</td>
<td>32.98 ± 2.6</td>
<td>36.2 ± 4.3</td>
</tr>
</tbody>
</table>

Data are expressed in terms of mean ± standard deviation for each parameter, (%): percent, Significance is considered when \(p < 0.5\), (\(\): increase, (\(\): decrease, a: significant difference when compared to the control group, b: significant difference when compared to scald injury group after 2 & 7 days, c: significant difference when compared to scald injury group after 1 month.

**DISCUSSION**

Scald injury is the most common type of burn (Singh et al., 2017), whether accidental or non-accidental as in case of deliberate self-immolation (attempt suicide) or due to assault (attempt homicide) (Silverstein and Lack, 1987). Scald injury has proven to elicit an immediate response in almost all body systems due to vascular permeability changes that lead to fluid and colloid loss, and pathophysiologic changes can occur in several body systems in the proceeding days (Jewo et al., 2011).

In the current study, skin with scald injury showed histopathological findings in the form of ulceration of the epidermis and severe dermal

**Table-4:** Testosterone mean serum levels of scald injury group after 2 days, 7 days, 1 month and 3 months, and the percentage of change compared to mean serum level of control group

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control Group</th>
<th>Scald Injury Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>After 2 days</td>
</tr>
<tr>
<td>Testosterone Serum level (\text{ng/ml})</td>
<td>3.26 ± 0.26</td>
<td>0.59(^a) ± 0.03</td>
</tr>
</tbody>
</table>

Data are expressed in terms of mean ± standard deviation in terms of ng/ml, Change %: percent of change compared to control level, \(\downarrow\): decrease, Significance is considered when \(p < 0.5\), (\(\): increase, (\(\): decrease, a: significant decrease when compared to the control group, b: significant difference when compared to scald injury group after 2 days, c: significant difference when compared to scald injury group after 7 days, d: significant difference when compared to scald injury group after 1 month.
inflammatory infiltrate that invades muscle layer in some areas by the end of the 2nd day of scalding injury, and then progressed to destruction of adnexal structures, and area of coagulative necrosis invaded by inflammatory cells by the end of 7th day. Healing process was evident by deposition of multiple collagen layers were observed by the end of the first month and re-epithelialization was observed in the form of multiple epithelial cells islands migrating towards the wound surface from underlying dermal appendages by the end of 3 months. The previously described inflammatory response is supported by the TNF-α immunohistochemical expression which was strongly positive during the early age of scald injury (after 2 and 7 days), and became negative in scald injury of 1 and 3 months of age.

The previously described histopathological changes can be referred to the study of Ipaktchi et al. (2014) who stated that burn injury strongly stimulates dermal release of proinflammatory mediators, resulting in progressive wound inflammation and tissue edema. This can be demonstrated, microscopically, by blistering of the epidermal layers, epithelial cells with pyknotic nuclei, injured adnexal cells in the deep dermis, and destruction of the superficial dermal appendages (Cribbs et al., 1998). Adeteye et al. (2011), also, stated that the microscopic examination of 3rd degree full-thickness thermal injury showed layers of immature collagen fibers amidst fibroblasts, dermal ulceration, areas of destruction of skin adnexal structures and replacement with coagulative necrosis.

Yongqiang et al., (2016) studied a mouse model of scald wounds in which a full-thickness scald injury was developed by exposing the dorsal skin to a 90°C water for 9 seconds. It was found that the epidermis of scalded mouse skin was broken and become separated from the dermal layer. The dermal hair follicles were, severely, damaged and almost unviable. In addition, there were homogenization and coagulative necrosis of some areas in the subcutaneous adipose tissue, and damage of superficial intradermal muscle layer.

During identifying the age of the scald, it has to be noted that it is a progressive injury, as stated earlier in the current study, during the further days after infliction i.e. extending in the area and depth in the days following the accident. This 2nd damage, as stated by Winter (1975) which occurs within 5-8 days, is, primarily, due to the heat coagulation of the contents of venules and capillaries with stagnation of the tissue fluid leading to inability of the surrounding tissues to provide the injured area with vital supplies of oxygen and glucose to the cells on the rim of the zone of severe and irreversible damage, and, secondarily, to loss of water vapour through the injured surface causing dehydration of the exposed dermis.

According to the results of the current study, a TNF-α immunohistochemical expression is positive in scald injury till 7 days of infliction, and becomes negative after 1 month of scald injury age. This can be explained by the fact that cytokines are mediators in the post-burn pathophysiological process and as an important pro-inflammatory cytokine, TNF-α is a key product released following a cutaneous thermal injury (Clark et al., 1995). The release pattern of TNF-α in the scald area of the current study is confirmed by the work of Kubo et al. (2014) who stated that TNF-α gene expression (together with other cytokines) increased significantly in a biphasic pattern from
3 or 6 hours to 12 hours or 1 day (inflammatory phase) and from 3 or 5 days to 7 days (proliferative phase).

**Jeschke et al. (2007)** reported the strength of the inflammatory and hypermetabolic responses is determined by the burn size, in which an increase in the later is associated with increased hyper-metabolic state, persistent inflammation, catabolism and organ dysfunction. This study explains the significant testicular dysfunction detected in the scald injured albino rats of the current study, on the histopathological, epididymal semen analysis and serum testosterone levels. **Yang et al. (2011)** studied a quantitative model of thermal injury-induced acute inflammation and postulated that the initiator of the inflammatory trajectory after was the release of TNF. The same authors added that a 20 % TBSA scald injury can cause marked inflammatory response and elevation in catecholamines, where both of which are associated with increased metabolic rate. The postulation of **Yang et al. (2011)** can, also, explain the positive immunohistochemical expression of TNF-α in the cytoplasm of spermatogonia, spermatocytes and the interstitial Leydig cells by the end of the 2nd and 7th days of scald infliction, and became negative in later scald ages (after 1 and 3 months).

In scald injured group of the current study, microscopic examination of the testes revealed reduction of number of germ cell layers and dissociation of the germ cells from the tubular basement membrane by the end of the 2nd day of scald application. Histopathological changes progressed to germ cell atrophy, absence of free spermatozoa, the tubules showed scanty cells even in their basal areas which lined only by Sertoli cells by the end of the 7th day, widespread cyto-architectural disruption of the seminiferous tubules and the tubules lined only by Sertoli cells by the end of 1 month, and considerable seminiferous tubular damage was present by the end of 3 months of scald infliction. The previous histopathological changes were accompanied by progressive significant decreases in the mean values of serum testosterone after 2 & 7 days and 1 month of injury infliction, with a percentage of decrease compared to the control level by 82%, 87% and 90%, respectively. After 3 months of injury infliction, significant improvement in the mean values of serum testosterone was recorded when compared to the control level and the mean levels recorded in the earlier periods.

These results correlated with the study of **Jewo et al. (2011)** who studied the histopathological changes and affection of testicular function in severely burned rats (after 8 and 16 weeks since scald application) and have reported that severe burns produced significant seminiferous tubular damage, atrophy of germ cell in the adluminal area, with tubular atrophy almost three times more than that found in the control group. Other histological changes were in the form of sloughing leaving only basal cells such as spermatogonia and Sertoli cells in many tubules. Serum testosterone showed significant progressive decline till after 16 weeks since scald application. Significant decrease in serum testosterone level recorded in the current study is supported by the results of previous researches. **Emanuele et al. (2005)**, in their study, have subjected young adult male mice to a 15% total body surface area and full thickness scald, and were sacrificed 48 hours later. Scald injury has reduced serum testosterone with an increase in hypothalamic concentrations of TNF-α (with other proinflammatory cytokines).

**Fadeyibi et al. (2010)** reported that a significant fall in serum levels of testosterone, luteinising hormone (LH) and
follicle-stimulating hormone (FSH), and the fall correlated with burn size. Also, Bergquist et al. (2016) have reported that burn injury has altered endogenous steroid biosynthesis, with decreased testosterone concentrations and elevated estrone concentrations, during the first 21 days after the burn injury.

CONCLUSION
According to the finding of the current study, identifying the age of scald injury can be performed using routine histopathological examination. For TNF-α Immunoeexpression, it can be used differentiating early and long term scald injury. A full-thickness scald injury involving 20% of TBSA can cause long term impairment of testicular function. Further studies for longer durations are required to confirm or deny this cause-effect relationship.

Figure 1: An H & E photomicrograph of a section in the skin of adult male albino rat. a, b, c: control group showing normal epidermis, dermis and adnexal structures, d: scald injury after 2 days showing heavy dermal inflammatory infiltrate, e: scald injury after 2 days showing destruction of epidermis and dermis heavy dermal inflammatory infiltrate invading muscle layer, f: ulceration of the epidermis and dermis, g: scald injury after 7 days showing coagulative necrosis, h: scald injury after 1 month showing destruction of skin adnexa and deposition of multiple layers of collagen, i: scald injury after 1 month showing re-epithelialization with multiple islands of epithelial cells migrating to the wound surface (x 200).
Figure 2: A DAB photomicrograph of a section in the skin of adult male albino rat showing the brown TNF-α Immunexpression (→). a: control group showing negative expression (x 200), b: scald injury after 2 days showing positive expression (x 200), c: scald injury after 7 days showing positive expression (x 400), d: scald injury after 1 month showing negative expression (x 200), e: scald injury after 3 months showing negative expression (x 200).

Figure 3: An H & E photomicrograph of a section in the testis of adult male albino rat. a, b: control group showing normal testicular tissue with normal cell arrangement and the lumen is full of mature sperm cells, c, d: scald injury after 2 days showing reduction of the number of germ cell layers, most tubules show no free mature spermatozoa in their lumen and dissociation of the germ cells from the tubular basement membrane, e, f: scald injury after 7 days showing germ cell atrophy and absence of free spermatozoa. The tubules lined only by Sertoli cells, g: scald injury after 1 month showing marked germ cell loss. The tubules show scanty cells even in their basal area. Some tubules show sloughing of the cells in their lumen, h: scald injury after 3 months showing considerable seminiferous tubular damage (→) (x 200).
Figure 4: A DAB photomicrograph of a section in the testis of adult male albino rat showing the brown TNF-α Immunoeexpression (→). a: control group showing negative expression (x 200), b: scald injury after 2 days showing positive expression (x 200), c: scald injury after 2 days showing positive expression (x 400), d: scald injury after 7 days showing positive expression (x 200), e: scald injury after 1 month showing negative expression (x 400), f: scald injury after 3 months showing negative expression (x 400).

Figure 5: A Nigrosin and Eosin photomicrograph of sperm abnormal forms detected in scald injury group after 1 and 3 months. a: showing coiled tail sperm b: bent tail sperm, c: 2-tailed sperm, d: flat head and bent tail sperm (→), (x400).

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المخصّص العربي

تقدير عمر إصابات الحروق لدراسة التأثير المحتمل على وظيفة الخصية لذكور الجرذان البيضاء

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قسم الطب الشرعي و السموم الإكلينيكية كلية الطب البشري - جامعة الزقازيق

الخلفية:
من وجهة النظر الطبية الشرعية ، يجب تحديد العواقب طويلة الأمد لإصابات الحروق بالسوائل الساخنة من أجل تحديد التعويض المناسب والإجراء القانوني ، خاصةً لإصابة الحرارية التي تتضمن سمك كامل للجلد في 20% من إجمالي مساحة سطح الجسم. وهو المعروف أن لها عواقب خطيرة على المدى الطويل على الصحة العامة. الهدف من الدراسة: كان الهدف من هذا العمل التجريبي هو التعرف على التغيرات النسيجية والتناسلية المناعية لتحديد عمر الإصابات الحرارية التي تشمل 20% من إجمالي مساحة سطح الجسم. إلى جانب ذلك ، تم دراسة التأثير طويل المدى لهذا الإصابة على الوظائف التناسلية لذكر الجرذان البيضاء. المواد والطرق:

تم استخدام عدد 40 من ذكور الجرذان البيضاء في الدراسة. أجريت فحوص في نهاية اليوم الثاني، اليوم السابع، الشهر الأول، والشهر الثالث من الإصابة تم ذبح الفئران لأخذ جزء الجلد المصابة بالحرق والخصيتين لعمل دراسة هистوباثولوجية باستخدام المجهر الضوئي، ودراسة حيوية وعدد الحيوانات المنوية، وقياس مستوي هرمون التستوستيرون. وتم عمل دراسة هيستوكيميائية مناعية لقطاعات من الجلد والخصية للكشف عن البروتين عامل النخر الورم (تي-إن-إيه).

النتائج:
لوحظت تغيرات نسيجية تقدمية في حين بدأت مظاهر الشفاء وتحسن تظهر بعد 1 و 3 أشهر من الإصابة. الاستنتاج: يمكن استخدام التغيرات الهيستوباثولوجية والهستوكيميائية المناعية لإصابة في تحديد عمر حرق يشمل 20% من إجمالي مساحة سطح الجسم والذي قد يتسبب ذلك في حدوث اختلال على المدى البعيد في الوظيفة التناسلية للذكور.