Evaluation of two biological matrices for repairing of ventral hernia in bucks

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Summary
The aim of this study was to estimate the changes in ventral hernia repairing in Iraqi bucks by using two biological matrixes derived from bovine (pericardium and urinary bladder matrix) through histopathological examination. All bucks right lower flank awas prepared surgically, sedation were done by using (2% Xylazine hydrochloride) at a dose of 0.2mg/kg intramuscular, and surgical site anesthetized through an inverted (L) shape local infiltration technique using lidocaine hydrochloride (2%) at a dose of 8mg/kg. Ventral abdominal hernias were induced in (24) bucks through elliptical resection of abdominal muscles to made hernia ring (6-8cm) with avoiding peritoneum perforation. After 30 days of operation bucks were divided into two equal groups. cellular Bovine pericardium group and cellular urinary bladder matrix group.
In two groups hernias were treated with only replacement of acellular bovine pericardium and cellular urinary bladder matrix respectively which fixed with interrupted horizontal mattress 2cm far from hernia ring by Polypropylene (No.1) suture material. Histopathological biopsies were taken at 2³, 8³ and 16³ week post treatment (4 bucks\ period). Both groups’ successes in reconstruction of large hernia in bucks through prevent recurrent or others post-operative complications. In addition the histopathological examination showed that the acellular urinary bladder matrix superior than acellular bovine pericardium matrix in enhance healing based on cellular urinary bladder matrix role in augment early initiation of inflammatory cells infiltration, fibroblast proliferation and marked collagen deposition, in addition to its early degradation, incorporation and remodeling.

Keywords: Hernia, Bucks, Bioimplants, Histopathological.

Introduction
Abdominal wall defects (hernia) correspond a difficult trouble, considered a common acquired condition in ruminant's which has some harmless effects, such as lowering the productivity and reproductively (1 and 2). Furthermore, large full-thickness, abdominal wall defects secondary to wide resection of malignant tumors, traumatic injuries or congenital abnormalities cannot be closed primarily (3). Surgical intervention (herniorrhaphy) is useful in these cases but large ventral abdominal hernia may require hernioplasty such as (Mesh repairs) to minimize the amount of tension that must be put on the abdominal wall in order to cover the hernia (4). However, these materials may in addition to cause infection and chronic pain in the surgical area (5), it may perhaps contribute to the dysfunction of other organs, and cause complications such as internal adherence, obstructions, and fistula development (6). To avoid the potential squeal of synthetic non absorbable materials, biological materials are being developed by the surgeons and used for abdominal wall defect repairs and other applications, such as porcine small intestinal sub mucosa, a cellular dermal matrix and human dura mater (5). The composition and structure of these biomaterials contrast according to the source of the tissue that can provide a reservoir of active molecules like growth factors that can be rapidly mobilized following injury to stimulate cell proliferation and migration (7).
The current study aimed to compare the efficacies of using of two natural bio-scaffolds including: acellular bovine pericardium matrix (BP) and acellular urinary bladder matrix (UBM) in the reconstruction of experimentally induced large ventral hernias in Iraqi bucks.

Materials and Methods

After obtaining an official approval from the ethical committee of the college. Twenty four apparently healthy adult local bucks aged 1-2 years and weighing (25-30) kg were used. Bucks were acclimatized for 2 weeks in pens at the College of Veterinary Medicine, University of Baghdad, Iraq before operation. The animals were divided and numbered according to the experimental design and each animal was injected s.c with Ivermectin as antihelminthic in a dose 0.2mg/kg., B.W and s.c vaccinated with 5ml of enterotoxaemia vaccine. Ventral abdominal hernias were induced in the right lower flanks of 24 bucks before herniation, food was withheld for 24 hours and water for 12 hours. Buck was sedated with (Xylazine hydrochloride 2%) at a dose of 0.2mg/kg B.W IM. In addition surgical site were anesthetized locally by using lidocaine hydrochloride (2%) at a dose of 8mg/kg B.W through an inverted (L) shape local infiltration technique. A vertical incision 10-12cm was done through skin and subcutaneous tissue, then abdominal muscles were separated bluntly, to create hernia 6-8cm of full-thickness abdominal muscles were resected with avoiding opening the peritoneum (Fig.1). Finally, skin and subcutaneous layers were sutured using simple interrupted pattern with non-absorbable suture materials (Silk No.1) and wounds were covered with sterile gauze.

Figure 1: Shows hernia ring after excision of abdominal wall muscles.

Whole fresh urinary bladders of cows were excised immediately after slaughtered at local abattoir. The matrix was prepared as described by (7 and 8) to make it acellular to prevent rejection. The urinary bladder was filled with tap water to facilitate trimming of external connective tissues and adipose tissue with scissors and washed in tap water. The tunica serosa, tunica muscularis and most of the muscularis mucosa were mechanically delaminated from bladder tissue by scraping with blunt knife. The remaining basement membrane of the tunica mucosa and the subjacent tunica propria became a urinary bladder matrix (UBM), decellularized and disinfected by immersion in peracetic acid (0.1%) and ethanol (4%) for two hours. Traces of peracetic acid were removed and pH adjusted to ~7.4 by rinsing in phosphate buffered saline (PBS) with 100 IU/ml penicillin, 100 µg/ml streptomycin and 100 µg/ml amphotericin. At room temperature the scaffold was rinsed and shaken in deionized water twice, and once in PBS for 15min. The scaffold was then sterilized by immersion in peracetic acid solution (0.1%) titrated to pH 7.0 at room temperature for five hours.

Bovine pericardium was excised from slaughtered cows at local abattoir and submerged in PBS (pH 7.4) and rinsed in saline until clean of blood. Fat and connective tissues was removed with dry gauze. The tissue was decellularized as mentioned by (9) with the same steps were done for UBM decellularization. After 30 days post inducing of hernia, all animals were allocated into two main equal groups. In Bovine pericardium group (BP), the hernias ring was closed with a sheets of acellular BP sheets. Under the effect of sedation and local anesthesia, as mention before, the area of the operation was ready for aseptic operation and vertical skin incision was made parallel to the original skin incision. Gentle blunt dissections of the underlying tissues to free the adhesions until defined the borders of the hernial ring and then the hernial sac was inverted into the abdominal cavity. Implants were soaked in sterile PBS (containing antibiotics and antifungal) for
three hours before operation, onlay repair technique was done and BP implant firmly fixed with the muscular layers of the hernia ring by interrupted stitches of horizontal mattress with Polypropylene (No.1) no less than 2cm far from hernia edges. The access skin and s.c tissue was sharply excised then skin closed. The same procedures as mentioned for first group were performed in urinary bladder group (UBM); with exception that the hernias were closed with acellular UBM implant (Fig.2). Post-treatment of hernia, sterile bandage truss were used for each buck to care the surgical site and to lessen postoperative edema. To reduce the activity of animals and avoid the trauma all animals were housed in separated pen. During first five days, the animals received soft food which increased gradually with the time associated with daily administration IM of a combination of penicillin and streptomycin in a dose of 10,000 IU/ kg and 10 mg/kg body weight respectively for 5days.

The histopathological evaluation was performed at 2, 8, and 16 weeks post-implantation (four animals/period). Segments of (1x1) cm³ were collected from three areas; edge of the implant, center and between them. The samples were fixed in neutral buffered formalin solution (10%) and set in paraffin and then sectioned longitudinally and transversally at (5-7) µm and staining with Hematoxylin-Eosin (H&E) and Mallory trichrome stain which stain collagen blue color and granulation tissue yellowish color (10).

The results of the histopathological score of progression of healing process in the site of hernias of both treatment groups were evaluated through using a scoring system, as mentioned in (Table,1), illustrated by (11). Higher scores within the scoring system represent more approving outcomes with rating to remodeling, as evidenced by cellular infiltration, host ECM deposition, neovascularization, scaffold degradation, fibrous encapsulation and cell types that represent low levels of inflammation.

Data were analyzed using SAS (Statistical Analysis System-Version 9.1). Two ways ANOVA and least significant differences (LSD) post hoc test were performed to assess significant difference among means P≤0.05 was considered statically significant.

![Figure 2: Shows the onlay fixation of the UBM sheet with interrupted horizontal mattress using Polypropylene (No.1).](image)

**Table,1: Histopathological scores of progression of healing process at the site of hernias (11).**

<table>
<thead>
<tr>
<th>scores</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cellular infiltration</strong></td>
<td>Zero cells in contact with Scaffold.</td>
<td>Cells contact periphery, no penetration into scaffold.</td>
<td>Cells infiltrate scaffold, not reach center</td>
<td>Cells penetrate into center of scaffold</td>
</tr>
<tr>
<td><strong>Cell types (neutrophiles, macrophages, and foreign body giant cells)</strong></td>
<td>Inflammatory cells present, no fibroblasts</td>
<td>Primarily inflammatory cells, few fibroblasts</td>
<td>Primarily fibroblasts few inflammatory cells</td>
<td>Fibroblasts only, no inflammatory cells</td>
</tr>
<tr>
<td><strong>Host extracellular matrix (ECM) deposition.</strong></td>
<td>No host ECM deposition</td>
<td>Host ECM deposited at periphery of scaffold</td>
<td>Host ECM deposited inside scaffold, but not at the center</td>
<td>Host ECM deposited inside scaffold, including the center</td>
</tr>
<tr>
<td><strong>Scaffold degradation</strong></td>
<td>Original scaffold intact, Borders clearly demarcated</td>
<td>Scaffold partially degraded, layers separated by cells, blood vessels, host tissue, etc.</td>
<td>Scaffold extremely degraded, difficult to distinguish scaffold from host tissue</td>
<td>Scaffold completely degraded, no evidence of original scaffold</td>
</tr>
<tr>
<td><strong>Fibrous encapsulation</strong></td>
<td>Extensive encapsulation (50-100% of periphery)</td>
<td>Moderate encapsulation (25-50% of periphery)</td>
<td>Mild encapsulation (≤25% of periphery)</td>
<td>No fibrous encapsulation</td>
</tr>
<tr>
<td><strong>Neovascularization</strong></td>
<td>Zero blood vessels presence</td>
<td>Vessels present at scaffold periphery, no penetration into scaffold</td>
<td>Vessels infiltrate scaffold but not reach center of scaffold</td>
<td>Vessels penetrate into center of scaffold</td>
</tr>
</tbody>
</table>
Results and Discussion

Clinically, all experimental bucks were kept under inspection along the period of the study to record general health, behavior and alertness, with no death of any animal. Ventral hernia was noticed directly post-operations of inducing hernias, there were appeared as clear tense swelling in the right low flank of the abdominal wall. Post-treatment of hernias, the hernia sac was disappeared, and local inflammatory reaction was gradually disappeared within 5 to 7 days, as well as, clinical followed-up of both treatment groups not recorded any complications, except different sizes of seromas were formed in three bucks of each treatment groups which were detected during 3 days post-implantation by gradual formation of sac like swelling at the site of implantation and treated by exploratory aspiration of the serous fluids under aseptic technique. Along the period of the study, one case of reherniation was noticed in UBM group at 5 days may be due to severe and continues coughing which associated to aspiration pneumonia.

In the present study, hernias were treated after 4th weeks post-inducing to create complicated hernia and fibrosis of hernia ring. All implants fixation were success without tearing through suturing with ring site. This result closed to (12), whom referred that the hernia repairing should be performed at least four weeks post-inducing may be due to it suitable time for resolution of active inflammation.

Alternatively, only replacement was used in this study is a successful technique with this biological implant and hernia size, it gave improvement of tissue healing as showed in histopathological results, without complications or recurrent. The same observations were noticed by (13), whom explained that onlay implantation allows of tissue in growth by two directions and (14), demonstrated that the contact of biomaterial with the fascia over or under the rectus muscles stimulates re-growth of the host tissue, and promotes the production and deposition of collagen on the suture line in addition to acceleration of wound healing.

In the current study, seroma was occurred in three animals for each treated group during 72 hours post-hernia treatment. Many previous studies like (15 and 16) recorded the formation of seromas in more than one case at the same period of the present study post-hernioplasty. The causes of seroma formation in the current study may be due to dissections that form separation between skin and subcutaneous tissue, as well as, the manipulation at surgical site for implantation that cause aseptic inflammation. These reasons are the same as recorded by (17), who referred to seroma formation due to local inflammatory response to a mechanical injury during surgical dissection and the presence of foreign bodies. While, (18) noticed that the abdominal wall approaching (ventral incision) lead to accumulation of blood and serum among the various level of wound resulting in hematoma or seroma or dead space. other authors (19) referred to the seroma formation are, the blood and lymphatic vessel injury during dissection, dead-space formation, shear forces among layers and the release of inflammatory mediators.

Wound infections weren’t noticed along the fallowed up period, it may be related to the biological implants components. Many studies were confirmed the antimicrobial effects of natural bio-scaffolds therefore increased frequency of their using to repair contaminated abdominal wall defects (20 and 21). Other result chers (22) suggested the presence of several different low molecular weight peptides with antibacterial activity exist within ECM and such peptides can resist the bacterial infection, and there were differences between antimicrobial peptides in each bioscaffolds type. Histopathological section at 2 weeks post-implantation in BP group revealed presence of highly cellular and vascular immature granulation tissue with severe hemorrhage and numerous congested capillary and moderate mononuclear cells (MNCs) (Fig.3), in another section there were a multifocal MNCs aggregation consist mainly of macrophages and lymphocytes with proliferation of fibroblasts producing collagen fibers (Fig.4), and identification of a fragment of muscles fibers observed within the necrotic area with calcium precipitation (Fig.5).
Figure, 3: Histopathological section in BP-treatment group at 2nd week post implantation, shows the presence of highly cellular vascular immature granulation tissue with severe hemorrhage (long arrow) numerous congested capillary (short arrow) and moderate mononuclear cells (MNCs) (H&E stain, 100X).

Figure, 4: BP-treatment group at 2nd week post implantation, shows a multifocal MNCs aggregation (long arrow) consist mainly of macrophages and lymphocyte with proliferation of fibroblast producing collagen fibers (H&E stain, 100X).

Figure, 5: BP-treatment group at 2nd week post implantation, shows fragment of muscles fibers observed within the necrotic area (long arrow) with calcium precipitation (short arrow) (Mallory stain, 100X).

Figure, 6: BP-treatment group at 8th week post implantation shows fragments of pericardial implant (long arrow) and high vascularization, presence of immature collagen fibers and blood vessels in the host tissue (short arrow) (Mallory stain, 200X).

Figure, 7: BP-treatment group at 8th week post implantation shows vascular (immature) granulation tissue (H&E stain).

Figure, 8: BP-treatment group at 8th week post implantation, shows presence of thick collagen fibers (long arrow) with moderate vascular granulation tissue (short arrow) (Mallory stain, 200X).

The histopathological section at 8 weeks post implantation in BP group showed a fragments of BP and high vascularization, presence of immature collagen fibers and blood vessels (BV) in the host.
tissue (Fig.6), in another section, vascular (immature) granulation tissue was seen (Fig.7), with presence of thick collagen fibers with moderate vascular granulation tissue (Fig.8).

Histopathological study at sixteen weeks post implantation in BP group showed BP implant surrounded (encapsulate) by thin zone of mature fibrous connective tissue with calcium precipitation and accompanied with moderate vascular response (Fig.9). The center of implant was invaded by moderate (MNCs) and collagen fibers (Fig.10), while, moderate vascular granulation tissue with moderate cellular infiltration was identified (Fig.11).

The histological sections at 2weeks of UBM revealed the formation of granulatin tissue consisting from dense collagen fibers with numerous dilated and congested BVs with mild MNCs response (Fig.12), as well as, vascular granulation tissue infiltrated with MNCs and myofibroblasts extended from muscular borders of implantation site (Fig.13). While in (Fig. 14) there were depositions of newly form collagen fibers mainly around tissue debris.
Figure, 13: UBM-treatment group at 2nd week post implantation, shows vascular granulation tissue was infiltrated with MNCs and myofibroblasts extended from muscular borders of implantation site (H &E stain, 400).

Figure, 14: UBM-treatment group at 2nd week post implantation shows deposition of newly form collagen fibers mainly around tissue debris (long arrow) (Mallory stain, 100X).

The host tissue at 8 weeks post implantation exhibited peripheral aggregation of MNCs forming granulomatous like reaction as shown in (Fig.15). In addition shows the construction of implant with delicate irregular collagen fibers (Fig. 16).

Figure, 15: UBM-treatment group at 8th week post implantation, shows that host tissue (long arrow) exhibited peripheral aggregation of MNCs (short arrow) forming granulomatous like reaction (H&E stain, 200X)

Figure, 16: UBM-treatment group at 8th week post implantation shows the construction of implant with delicate irregular collagen fibers (long arrow) (H&E stain, 400X).

At sixteen weeks post implantation, the histopathological finding characterized by formation of thick regular collagen fibers infiltrated with MNCs which attached to host muscular layer and incorperation of new collagen bandle within muscular coat (Fig.17). In addition, areminante of implant near the suture material surrounded by new fibrous tissue (Fig.18).

Figure,17: UBM-treatment group at 16th week post implantation, shows regular collagen fibers infiltrated with MNCs which attached to host muscular layer and incorporation of new collagen (H&E stain,200X)

Figure, 18: UBM-treatment group at 16th week post implantation, shows the remnant of implant (long arrow) near the suture material surrounded by new fibrous tissue (Mallory stain, 400X).
The analysis results of the histopathological score (Table, 2) gives well information about the role of both implants in enhanced healing, and reflected that despite the significant difference (P≤0.05) between both group at the 2nd and 8th week, while no significant differences was confirmed at 16th week.

Table 2: Shows the Means± SE of histopathological score results for both treatment groups.

<table>
<thead>
<tr>
<th>Reaction score</th>
<th>Group</th>
<th>2 weeks</th>
<th>8 weeks</th>
<th>16 weeks</th>
<th>LSD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cellular Infiltration</td>
<td>BP</td>
<td>C 1.00±0.28 b</td>
<td>B 2.00±0.14 a</td>
<td>A 3.00±0.57 a</td>
<td>0.9945</td>
</tr>
<tr>
<td></td>
<td>UBM</td>
<td>B 2.00±0.14 a</td>
<td>A 3.00±0.28 a</td>
<td>A 3.00±0.14 a</td>
<td></td>
</tr>
<tr>
<td>Cell type (inflammatory cells)</td>
<td>BP</td>
<td>B 1.00±0.14 b</td>
<td>A 2.00±0.28 b</td>
<td>A2.00±0.28 b</td>
<td>0.6022</td>
</tr>
<tr>
<td></td>
<td>UBM</td>
<td>B 2.00±0.14 a</td>
<td>A 3.00±0.00 a</td>
<td>A 3.00±0.14 a</td>
<td></td>
</tr>
<tr>
<td>Host (ECM) deposition</td>
<td>BP</td>
<td>B 1.00±0.28 b</td>
<td>A 2.00±0.28 b</td>
<td>A2.00±0.28 b</td>
<td>0.9945</td>
</tr>
<tr>
<td></td>
<td>UBM</td>
<td>B 2.00±0.28 a</td>
<td>A 3.00±0.28 a</td>
<td>A 3.00±0.14 a</td>
<td></td>
</tr>
<tr>
<td>Scaffold Degradation</td>
<td>BP</td>
<td>C 1.00±0.14 b</td>
<td>B 2.00±0.28 a</td>
<td>A2.00±0.28 b</td>
<td>0.7032</td>
</tr>
<tr>
<td></td>
<td>UBM</td>
<td>B 2.00±0.14 a</td>
<td>B 2.00±0.14 a</td>
<td>A3.00±0.28 a</td>
<td></td>
</tr>
<tr>
<td>Fibrous Encapsulation</td>
<td>BP</td>
<td>A 3.00±0.28 a</td>
<td>C 1.00±0.00 a</td>
<td>B 2.00±0.00 b</td>
<td>0.629</td>
</tr>
<tr>
<td></td>
<td>UBM</td>
<td>B 2.00±0.28 b</td>
<td>C 1.00±0.00 a</td>
<td>A 3.00±0.28 a</td>
<td></td>
</tr>
<tr>
<td>Neovascularization</td>
<td>BP</td>
<td>B 2.00±0.00 a</td>
<td>A 3.00±0.00 a</td>
<td>A 3.00±0.28 a</td>
<td>0.7263</td>
</tr>
<tr>
<td></td>
<td>UBM</td>
<td>B 2.00±0.28 a</td>
<td>A 3.00±0.28 a</td>
<td>C 1.00±0.28 b</td>
<td></td>
</tr>
</tbody>
</table>

The analysis results of ECM deposition and host degradation illustrated significant differences (P≤0.05) in both groups along the period of the study the mean values were lower at 2nd week and increased with time in both treatment groups. In addition in UBM-implants were faster than BP-implants, histopathological sections appeared the deposition or invited of new tissue to replace the scaffold in UBM group at 2nd week. While, in BP treated group the deposition showed periphery and not reach the center till 16th week post-implantation. Alternatively, histopathological score of presence study showed different between both treatment groups in inflammatory cells infiltration, host ECM deposition and matrix degradation in which UBM-implants were faster than BP-implants in degradation and host deposition, as well as, early cellular infiltration. These results enclosed with (25 and 26) referred that the degradation of BP implants occurs in uniform and gradual. While (27), confirmed that the UBM-implants rapidly degraded and turnover (remodeling). In addition (28), referred that the physical and chemical properties of biomaterials could influence on the intensity and duration of the inflammatory response. The
result of degradation of the implants showed significant differences ($P\leq 0.05$) in both group for each period of study. The mean values in both groups were lower at 2nd week and progress with time. The means in BP animals group were lower than UBM treated group for all periods. The result of fibrous encapsulation also recorded significant differences ($P\leq 0.05$) for both groups along the studied periods, that mean early encapsulation in UBM treated group compared to BP treated group that mean well incorporation were occur in early time in UBM group.

The analysis neovascularization score reflect the site of blood vessels were distributed, there were a significant differences ($P\leq 0.05$) along the studied period in both groups, while no significant differences between both groups at each period except that at 16th week which shows higher means in BP group than that in UBM group. In the current study showed early and faster degradation of UBM implants compared to BP implants, this fact may related to early infiltrations of inflammatory cells in UBM treatment group that may be enhanced more attraction to fibroblasts cells and collagen deposition. Realy bioscaffolds degradation can stimulates the releasing of the inherent bioactive constituents and promote host cells deposition. This fact was noticed by (29), referred the degradation process has a significant biological activity which stimulates the releasing of the inherent bioactive constituents subsequently promote tissue neovascularization and host-cell deposition (30 and 31) through encouraging cells attachment, proliferation, differentiation, maturation and angiogenesis.

Histopathological results also reflect the role of UBM implant in augmented remodeling process, it may be related to UBM and BP matrix composition and contains like growth factors or cytokines which retained in implant that causes change in the cells quantity and quality that’s precipitated in the healing process and augment remodeling when compared to BP implants group. This fact explained by (22 and 32), indicated about the different in the products of each ECM that related to its source, these products have multiple biological properties including angiogenic, chemotactic and antibacterial activity when liberating growth factors and cytokines during their degradation process by the host proteolytic enzymes.

The results of histopathological score of the present study appeared that UBM implant encapsulated started at 2nd week post-implantation prior to BP and progress with time to thick regular collagen fibers while in BP treated group showed it encapsulated by thin zone of mature fibrous connective tissues with calcium precipititation, irregular collagen fibers. This result support the idea of UBM implant cause or induced early initiation of inflammatory response and fibroblast attraction compared to BP implants. The same outcomes were recognized by (33 and 34), who proved that the biological scaffold encapsulation indicates an accelerated progression of the inflammatory response. In addition (24), confirmed that the dense fibrous capsule which forms around implant during remodeling was responsible for the firm incorporation between the implant and the host tissue, as well as, the zone of capsule did not interfere with the progression of implant degradation. All that change in the results of histological score included rate of inflammatory cells infiltration and implant degradation or deposition may be related to the change in the implant ultra-structure. This result agree with results of a study by (35 and 36), were mentioned that UBM implants allowed or augmented fibroblasts proliferation and collagen production due to it contains collagen more than 90 % of whole matrix, specially collagen type-III that consider a homo-trimeric fibrillar collagen which providing initial support for cell migration and adherence and facilitates ECM turnover and remodeling. While, BP implants augments neovascularization due to collagen type-I is the predominant component of pericardial tissue which arranged hierarchically in different levels, as well as, it has been shown to induce activation of mitogen activate protein kinase pathways that promote angiogenesis (37).

In addition, differences in collagen orientation between BP and UBM-implants may have a role in change of host response and healing process, BP matrix has a wavy shape of collagen fibers in all species, more intense curly appearance in bovine than other
species of mammals but, the collagen in UBM implants have unidirectional as showed by (38). While (39 and 40), explained, that the orientation of ECM components, such as collagen fibers, can profoundly influence the directed migration of cells, possibly by potentiating growth factor receptor signaling or by mechanically reinforcing cells migration. It is concluded that, the present study demonstrated that the only implantation technique of acellular BP and UBM were a simple, less cost-effective and safe methods with minor non-serious complications to support large abdominal wall hernias. In addition UBM implant superior than BP implant in enhance healing based on early initiation of inflammatory cells infiltration, fibroblast proliferation and marked collagen deposition, in addition to its early degradation, incorporation and remodeling.

References


تقييم لأثنين من الأغشية الإحيائية لترميم الفتق البطنية في ذكر الماعز

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الخلاصة

الهدف من هذه الدراسة هو تقييم التغيرات في ترميم الفتق البطنية باستخدام اثنين من الأغشية الإحيائية الأخوية من الأبقار، شغاف القلب البقر والنسج الغشائي. كان من خلال الفحص النسيجي، حيث تم تقييم الفتق البطني في 24 (ذكر من الماعز) بعد معالجة 4 فترات. تتم تقييم الفتق البطني والأنسجة المصابة بواسطة مكونات الفتق البطني و과정 التخليص. أتبعت مجموعة المعالجة (الغشاء الباطني) وتمارين التدريبية بشكل متكرر على النمط الأفقي للغشاء النسيجي. النتائج:

أ. الفتق البطني يتم ترميمه بنجاح في جميع الحوزات.

ب. الفتق البطني يمكن تعويده بنجاح في جميع الحوزات.

ج. الفتق البطني يتم ترميمه بنجاح في جميع الحوزات.

ك. الفتق البطني يتم تعويده بنجاح في جميع الحوزات.

الكلمات المفتاحية: الفتق، ذكور الماعز، الغرس الإحيائي، النسيج البطني، الغشاء الباطني


