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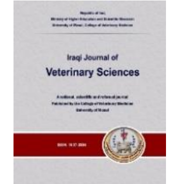

Repair of the cerebral dura mater defect by temporalis muscle fascia or peritoneum as autografted tissues in dogs

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Abstract

This article was designed to compare the efficiency of temporalis muscle fascia or peritoneum as autografted tissues with fibrin glue to reconstruct induced dural defects in dogs. Eighteen adult dogs were used. The animals were divided randomly into two equal groups. The cerebral dura mater induces a defect about (1x 1cm) in diameter in all these experimental animals. The defect was substituted with temporalis muscle fascia in the first group, while in the second group, the defect was substituted by a piece of the peritoneum. The results were evaluated through monitoring postoperative clinical signs, gross features, histopathological changes, and estimation of cerebrospinal fluid pressure with analysis of total cell count, glucose, and protein level of cerebral spinal fluid in both groups at the 15th, 30th, and 60th postoperative days. The experimental animals stayed alive postoperatively without any development of complications primarily related to the appearance of abnormal nervous signs. Grossly, the craniectomy site in both groups was occluded completely, and no cerebrospinal fluid leaks were noticed. Additionally, the dural defect was closed internally, where the grafted tissues were entirely sealed with the dura mater of the host. The histopathological changes were represented by connective tissue proliferation with infiltrations of mononuclear inflammatory cells and angiogenesis. The cerebrospinal fluid pressure, total cell count, glucose level, and protein appeared within the normal range during all experiment periods in both groups. The conclusions of this study can be used in both autograft tissues with fibrin glue to repair dural defects in dogs.

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Introduction

The term meninx is a Greek word meaning the membrane (1). Therefore, the membranes surrounding and covering the brain and spinal cord are called the meninges. There are three layers of meninges. The outer layer is called the dura mater, characterized by thick, challenging, and fibrous dense connective tissue in nature (2). The second and intermediate layer of the meninges arachnoid is very close to the dura mater and has delicate properties (3). The third and inner layer is the pia mater, which has a rich blood supply. The space between the pia mater and arachnoid is subarachnoid, which contains the cerebral spinal fluid (CSF) (4). Generally, the dura mater consists of periosteal, meningeal, and border cell layers. The periosteal layer is attached to the skull and contains blood vessels, and nerves with elongated fibroblast and intercellular space (5,6). The meningeal layer contains more fibroblast with less collagen (7), while the dural border cell, also called mesothelial layer, has flattened fibroblast with very little intercellular space (8). The dural tear is the main problem of the dura mater because of injury produced by head punctured wounds or during surgical operations of the brain. The cerebrospinal fluid leakage after tearing of the dura mater, which is manifested by some neurological signs,

may lead to many problems, such as the formation of cerebrospinal fluid fistula, pseudo meningocele, epidural abscess, meningitis, and arachnoiditis (9). However, dural defects should be repairing during the same surgical operation by either suturing, sealants, or using different grafted materials and patches (10). Several dural substitutes and sealants in neurosurgical operations close the dura mater (11). Many studies were done on different animals, such as canine, ovine, and feline, to evaluate the efficiency of several medical devices to repair the dural defect (12). Generally, the simple dural tear is closed by suturing, but the suture application is limited due to the location and condition of the damaged dura (10). Fibrin glue is the most used material to repair simple dural defects (13). Fibrin glue may improve and accelerate the repair of the dura and limit cerebrospinal fluid leakage by providing good binding between the graft materials and the dura (14). Tissue adhesives and sutures with transplant materials may be used together where they are available, easy to apply, not cost, and relatively rapid technique (15). Several materials are used to graft the dural defect, like fascia lata, autologous pericranium, and bovine pericardium (16,17).

Therefore, this work aims to know the ability of temporalis muscle fascia and peritoneum as autografted tissues with fibrin glue instead of suturing to reconstruct induced cerebral dura mater defect in dogs.

Materials and methods

Animals

This experimental work, used eighteen adult local breed male dogs aged 1-2 years with an average weight of 25±1.8 kg. The animals were divided into equal groups. The animals were examined clinically to ensure their safety from diseases related to the nervous system. The animals were kept in specific cages in the animal house at the College of Veterinary Medicine, University of Mosul.

Ethical approve

The research was approved by the Ethics Committee of the Faculty of the College of Veterinary Medicine, University of Mosul. No. UM. VET. 2023. 003.

Anesthesia

The protocol of general anesthesia was accomplished by injection of a mixture of 10 % ketamine HCL at a dose of 10 mg/kg and 2% xylazine at a dose of 2mg/kg intramuscularly with administration of atropine sulfate as a pre-anesthetic agent at a dose 0.05 mg/kg subcutaneously (18).

Surgical procedure

The animals were fasted from food and water intake about 12 hours before the surgical operation. After induction of general anesthesia, a craniectomy was performed to reach the cerebral dura mater. In this surgical operation, a piece of skull from the parietal bone should be excised and removed. However, the skin incision was made a V shape on the forehead of the animal. Then, the incised skin was elevated caudally to expose the subcutaneous tissue. The exposed subcutaneous tissue was incised and reflected laterally to expose the temporalis muscle. Then, the temporalis muscle was incised along the sagittal suture, dissected with blunt scissors, and expanded by a wound retractor to facilitate exposure of the parietal bone. After, opening the parietal bone as a square shape using an electrical saw, the excised bone piece of the skull was elevated using a bone chisel to show the dura mater. The craniectomy site exactly between the lambdoid and coronary suture of the skull (19). The hemorrhage during craniectomy was controlled through the application of bone wax. After exposing the dura mater, an induced defect, a square shape about (1x1cm) in diameter, was established on the dura mater (Figure 1). Immediately after incising the dura mater, cerebral spinal fluid was leaked. The substitute of induced dura mater defect was accomplished in the first group with a piece of temporalis muscle fascia taken from the same animal (Figure 2), while in the second group, a piece of peritoneum taken from the same animal was used to close the site of the dural defect. In both groups, the fibrin glue was used to seal the grafted piece of peritoneum and fascia of temporalis muscle with the hosts dura mater by applying a few drops of fibrin glue around the edges of each grafted piece of tissue. Then, the operation site was closed by suturing the temporalis muscle and subcutaneous tissue by polyglactin suture using a simple continuous pattern and skin with silk using a simple interrupted pattern.

Postoperative care

The experimental animals were injected intramuscularly with systemic antibiotic and analgesic postoperatively using penicillin streptomycin (1ml /10 kg. and metalgen 1ml/ day respectively) (20) for seven days after surgical operation. Also, the site of operation was dressing daily with the application of oxytetracycline spray until complete skin wound healing.

Assessment of dural defect healing

The follow-up examinations include, monitoring the clinical signs postoperatively, estimation of CSF pressure, and analysis of CSF total cell count with CSF glucose and protein level in both groups at the 15,30, and 60 postoperative days. Specimens of the graft area with the cortex of the brain were obtained on the 15th, 30th, and 60th days after euthanizing three animals of each group in each period for macroscopic observation, and histopathological evaluation where the samples fixed in 10% formalin and stained with hematoxylin and eosin (H&E) stain (21). The histological sections were scored according to the following criteria [1] Intensity of connective tissue, [2] Intensity of angiogenesis, and [3] Intensity of inflammatory reaction (Table 1).

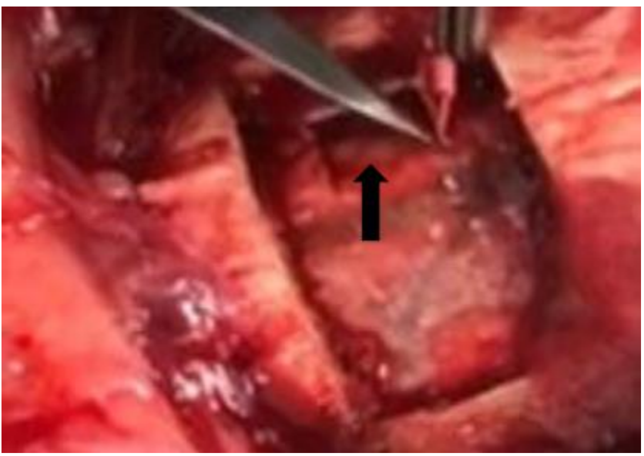

Figure 1: Cerebral dural defect.

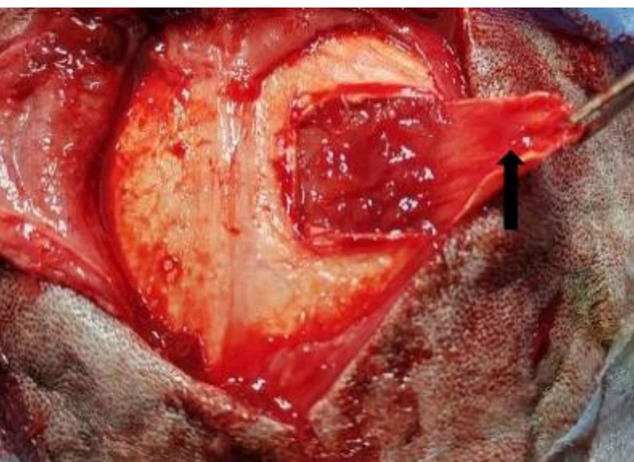

Figure 2: Fascia of temporalis muscle.

Table 1: Showing the histopathological scoring (22)

Criteria	0	1	2	3	4
Connective tissue	Absent	Discrete	Moderate	Intense	Complete
Angiogenesis	Absent	Discrete	Moderate	Intense	Complete
Severity of inflammation	Absent	Few	Few	Moderate	Sever

The histopathological scores were analyzed statistically by pathologists using the Kruskal-Wallis One Way Analysis of Variance on Ranks test and using Pairwise Multiple Comparison Procedures (Tukey Test) at $P \leq 0.05$. We used the Sigma Plot software program for statistical analysis. Also, the statistical analysis of CSF protein and glucose level with total cell count was dependent.

Collection of cerebral spinal fluid

This study, collected CSF under general anesthesia from the cerebellomedullary cistern at the back of the animal head. The main landmarks depended collecting cerebrospinal fluid from the cistern, including the external occipital protuberance, the cranial aspect of the dorsal spine of the axis, and the transverse processes of the atlas. However, after induction of general anesthesia, the back of the neck was shaved and sterilized. Then, a spinal needle is inserted at the base of the skull, and into the spinal column, penetrating the dura mater and arachnoid membranes to the subarachnoid space. Then, CSF will escape or leak through a spinal needle into a sterile plain tube. About 5 ml was collected to make CSF analysis (23).

Estimation of cerebral spinal fluid pressure

Generally, the best methods to diagnose any CSF leaks were accomplished by magnetic resonance cisternography (MRCG) and computerized tomography myelography (CTMG). Due to any of these techniques unavailable, monitoring of CSF pressure by using a catheter as the modified technique was depended to ensure there were no CSF leaks where a small, flexible tube was placed and fixed on the calculated ruler and connected with the spinal needle after insertion of it is in the subarachnoid space. The animal must be placed on one side and one level. Additionally, the tube used to collect CSF should be placed vertically on the animal and spinal needle and left for a short time to track changes in the CSF pressure.

Cerebral spinal fluid analysis

The main components of the CSF adopted during the analysis of CSF to evaluate the efficiency of each type of grafted tissue used in this work include the protein and glucose levels with an estimation of the total cell count.

Estimation of CSF glucose

1 ml of CSF was taken from the collected sample of CSF and put in a sterile glass tube. Then, a glass tube that contained a CSF sample was placed inside a device spin 120. The reagent used in this study to estimate the CSF glucose level is the glucose spin reagent. The reaction between the sample of CSF and glucose spin reagent will provide the data for analysis.

Estimation of CSF protein

The device used in this work to estimate the level of CSF protein was the device Fujifilm. However, 1 ml of CSF was put in the Eppendorf tube. Then, a disposable fuji dri-chem slide and auto tips with Eppendrof tube containing the CSF sample were put in Fujifilm. The chemical reaction between the CSF sample and the fuji dri-chem slide will be given as the analysis result.

Estimation of CSF total cell count

A hemocytometer chamber was used to estimate the total cell count of CSF. Each chamber was composed of 5 mini chambers in a square shape, four peripheral and one central. To read white cells (lymphocyte, monocyte, neutrophils, and eosinophils), 25nmicrons of the CSF sample were applied on one peripheral mini chamber in addition to its 75 microns of new methylene blue. Then, the sample was left for 10 minutes to fix, and then the hemocytometer chamber was washed with tap water and the sample to dry. After that, 25 microns of oily drop was applied on the chamber with putting cover slide. The results were recorded under a microscope using a lens with power (100x). While, the central mini chamber was used to read only the red cells. The methylene blue was not used (only the CSF sample).

Results

Clinical signs postoperatively

There was a swelling at the site of operation in all operated animals. The swelling disappeared within the first week postoperatively. However, animals of both groups were still alive without any complications, especially related to the appearance of abnormal nervous signs such as epilepsy, muscle tremors, abnormal herring, or visions. In addition, the animals were eaten and drank usually, and no change was shown in their behaviors.

Gross appearance

The craniectomy site was entirely sealed with granulation tissue during all study periods, and no cerebral spinal fluid leaks were noticed (Figures 3 and 4). The size of the dural defect was diminished internally at the end of the study in both groups, where the grafted tissues connected with the dura mater of the host completely (Figures 5 and 6).

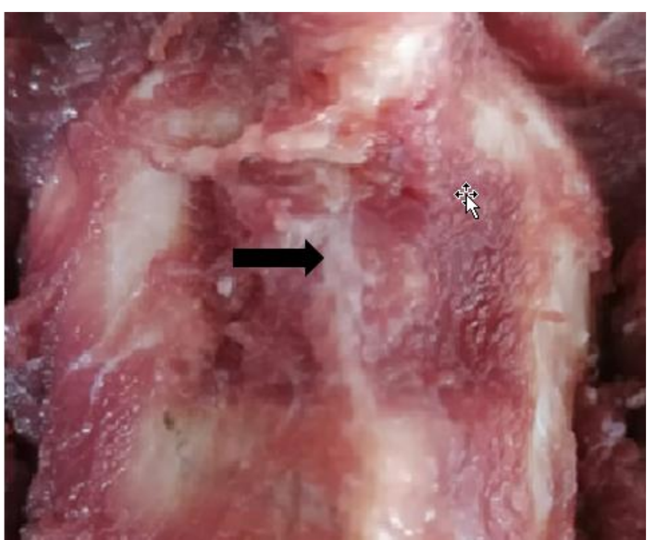

Figure 3: Operation site at 60 PO days in the first group.

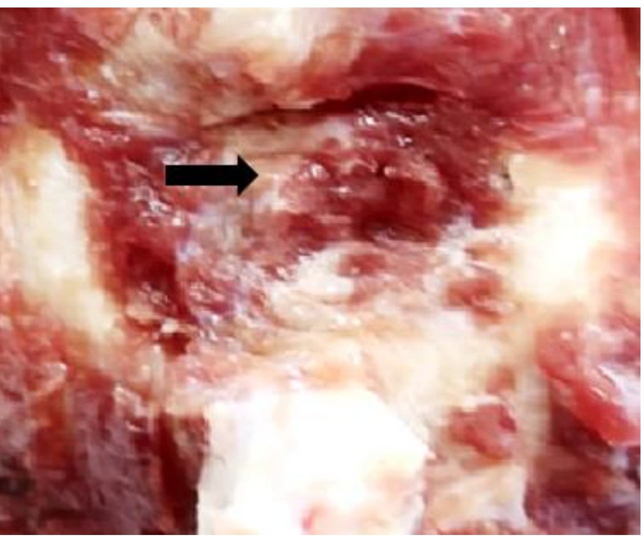

Figure 4: Operation site at 60 PO days in the second group.

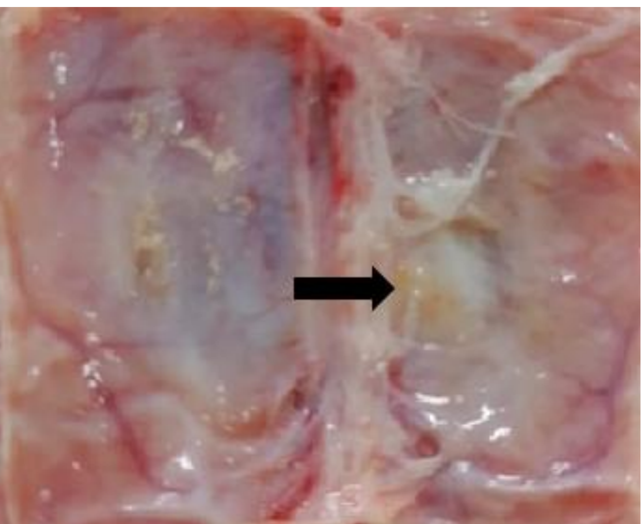

Figure 5: Dural defect at 60 PO days in the first group.

Histopathological examination

In the first group, the histopathological section at the grafting site after the 15th postoperative day was characterized by the proliferation of connective tissue with new blood vessels formation, infiltration of inflammatory cells, and deposition of eosinophilic pretentious material within the connective tissue (Figure 7). On the 30th postoperative day, the changes showed connective tissue and blood vessels maturation with reduced infiltration of inflammatory cells (Figure 8). On the 60th postoperative day, the histopathological sections revealed more connective tissue maturation with mild angiogenesis and infiltration of inflammatory cells (Figure 9). The normal histological architecture of the brain cortex section during the study period was shown (Figure 10).

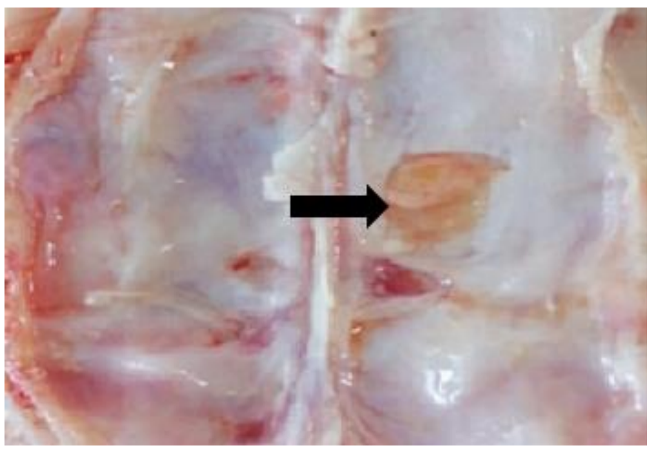

Figure 6: Dural defect at 60 PO days in the second group.

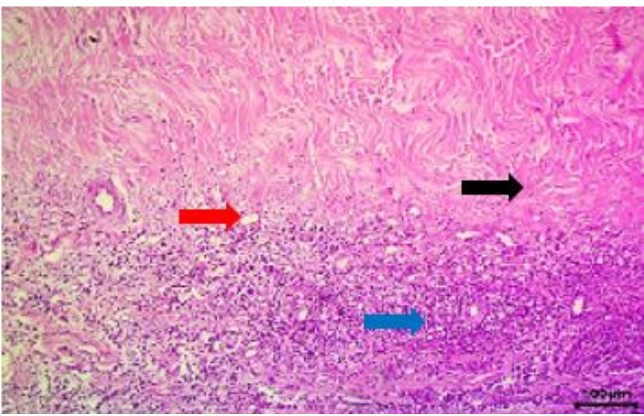

Figure 7: Micrograph at 15th postoperative days in the first group shows proliferation of connective tissue (black arrow), angiogenesis (red arrow), and infiltration of inflammatory cells (blue arrow) (H&E.100X).

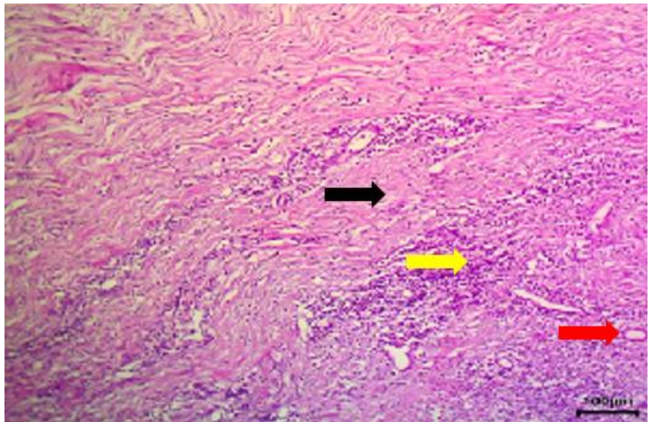

Figure 8: Micrograph at 30th POD in G1 shows a maturation of connective tissue (black arrow), new blood vessels (red arrow), and infiltration of inflammatory cells (yellow arrow). (H&E.100X).

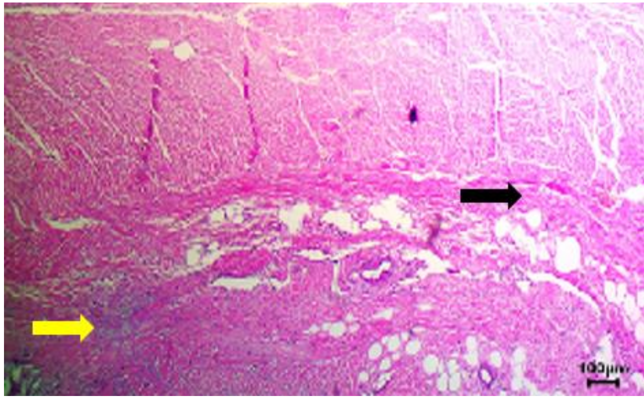

Figure 9: Micrograph at 60th postoperative days in the first group shows a maturation of connective tissue (black arrow) and very infiltration of inflammatory cells (yellow arrow) (H&E.100X).

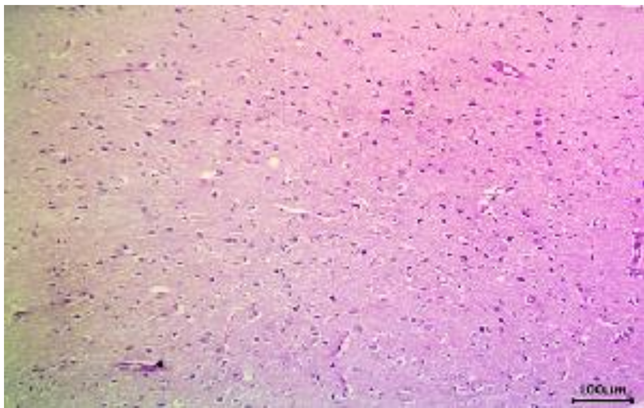

Figure 10: Micrograph at 60th POD in the first group shows normal brain cortex. (H&E.100X).

In the second group, the histopathological section at the grafting site after the 15th postoperative day was characterized by the proliferation of connective tissue, severe inflammatory cell infiltration, more angiogenesis, and deposition of eosinophilic pretentious materials within the proliferative connective tissue (Figure 11). On the 30th postoperative day, there is maturation in the connective tissue, pronounced angiogenesis, and reduced inflammatory cells infiltration (Figure 12). On the 60th postoperative day, the histopathological section showed mature connective tissue with few infiltrations of inflammatory cells and angiogenesis (Figure 13). In addition, the normal histological architecture of the brain cortex was shown during the period of the study (Figure14).

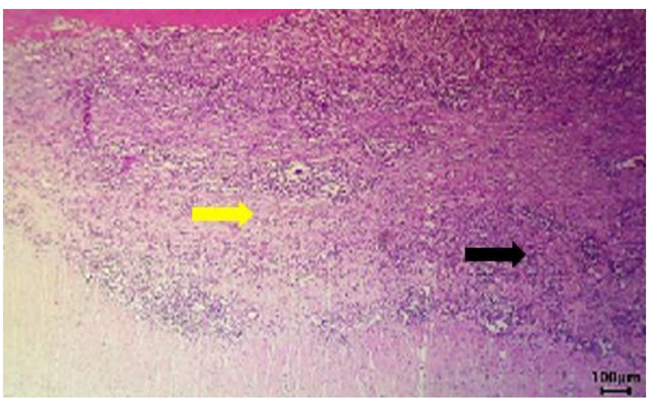

Figure 11: Micrograph on the 15th postoperative day in the second group shows proliferation of connective tissue (yellow arrow) and severe infiltration of inflammatory cells (black arrow) (H&E.40X).

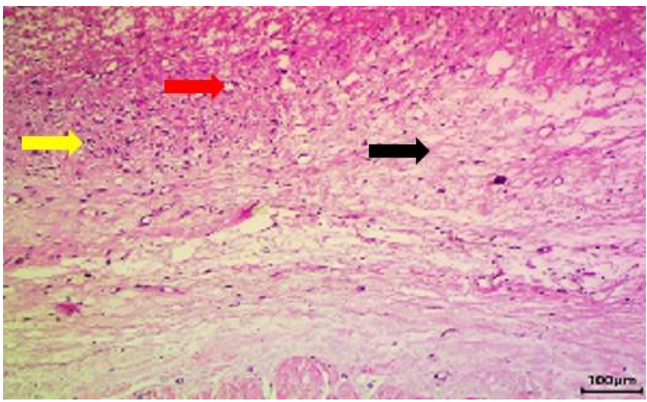

Figure 12: Micrograph at 30th POD in G2 shows mature connective tissue (black arrow), angiogenesis (red arrow), and little infiltration of inflammatory cells (yellow arrow). (H&E.100X).

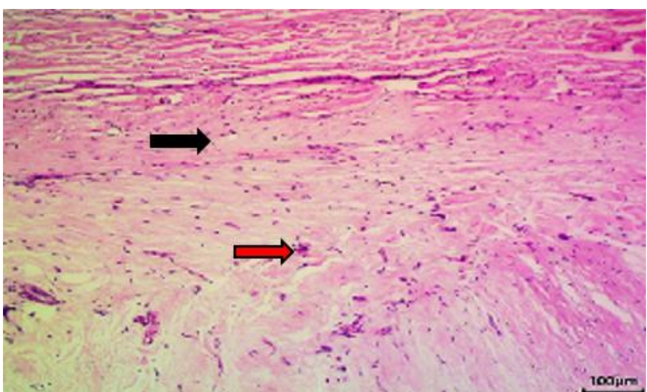

Figure 13: Micrograph at 60th postoperative days in the second group shows mature connective tissue (black arrow) and few infiltrations of inflammatory cells (red arrow). (H&E.100X).

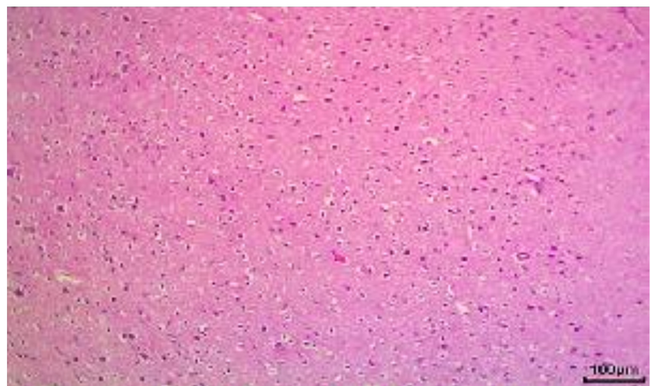

Figure 14: Micrograph at 60th POD in the second group shows normal brain cortex (H&E.100X).

The statistical analysis of the histopathological scoring

The statistical analysis of the intensity of connective and angiogenesis revealed no significant difference at $P\leq0.05$ between both groups and each group itself during the study periods (Tables 2 and 3). The statistical analysis of the intensity of inflammation revealed significant differences at $P\leq0.05$ between both groups at 15th postoperatively, but there is no significant difference at $P\leq0.05$ in each group during the study periods (Table 4).

Table 2: Intensity of connective tissue scores

Groups	15th P.O.Ds. / n=9 / Median	30th P.O.Ds. / n=6 / Median	60th P.O.Ds. / n=3 / Median
G1	3	3	4
G2	2	3	4

Data expressed as Median of the scores (Kruskal-Wallis test). The difference letters mean there are significant differences between groups at $P\leq0.05$.

Table 3: Angiogenesis scores

Groups	15th P.O.Ds. / n=9 / Median	30th P.O.Ds. / n=6 / Median	60th P.O.Ds. / n=3 / Median
G1	3	2	0
G2	4	2	1

Data expressed as Median of the scores (Kruskal-Wallis test). The difference letters mean there are significant differences between groups at $P\leq0.05$.

Table 4: Intensity of inflammatory response scores

Groups	15th P.O.Ds. / n=9 / Median	30th P.O.Ds. / n=6 / Median	60th P.O.Ds. / n=3 / Median
G1	3	2	1
G2	4 A	1	1

Data expressed as Median of the scores (Kruskal-Wallis test). The difference letters mean there are significant differences between groups at $P\leq0.05$.

Cerebrospinal fluid analysis with pressure

The cerebrospinal fluid pressure in both animal groups during periods of the study, in addition to the total cell counts and level of protein and glucose appeared within the normal range, and there is no significant difference at P≤0.05 between both groups (Tables 5-8).

Table 5: Cerebral spinal fluid pressure

Groups	CSF pressure (mm Hg)±St. E.			
	Before operation (N=9)	15th P.O.Ds. (N=9)	30th P.O.Ds. (N=6)	60th P.O.Ds. (N=3)
G1	10.7±0.92 A	10.6±0.47 A	10.3±0.47 A	10.6±0.33 A
G2	11.8±0.85 A	10.8±0.35 A	11.3±0.61 A	10.0±0.57 A

The difference letters mean there are significant differences between groups at P≤0.05.

Table 6: Cerebral spinal fluid protein

Groups	CSF protein (mg/dl)±St. E.			
	Before operation (N=9)	15th P.O.Ds. (N=9)	30th P.O.Ds. (N=6)	60th P.O.Ds. (N=3)
G1	28.1±1.7 A	30.4±1 A	29.5±1.4 A	31±0.5 A
G2	27.1±2.2 A	32.2±0.4 A	31.6±0.4 A	30.3±0.3 A

The difference letters mean there are significant differences between groups at P≤0.05.

Table 7: Cerebral spinal fluid glucose

Groups	CSF glucose (mg/dl)±St. E.			
	Before operation (N=9)	15th P.O.Ds. (N=9)	30th P.O.Ds. (N=6)	60th P.O.Ds. (N=3)
G1	64±4.2 A	66.6±4.7 A	63.1±0.8 A	63±1.7 A
G2	65.2±5.2 A	68±1.2 A	66.3±1.2 A	66.6±3.5 A

The difference letters mean there are significant differences between groups at P≤0.05.

Table 8: The total cell counts

Groups	CSF cells count (%)±St. E.			
	Before operation (N=9)	15th P.O.Ds. (N=9)	30th P.O.Ds. (N=6)	60th P.O.Ds. (N=3)
G1	1±0.3 A	2.2±0.3 A	2.1±0.4 A	0.6±0.3 A
G2	0.8±0.3 A	2.1±0.3 A	2.4±0.6 A	0.3±0.3 A

The difference letters mean there are significant differences between groups at P≤0.05.

Discussion

The dural defect was considered a primary problem during brain surgery that can result from different causes such as trauma, tumors, or congenital malformations (24-27). The authors Fontana *et al.* (28) Van Calenbergh *et al.* (29) mentioned that it is essential to substitute the dural defect with different biomaterials to avoid some significant complications, for example, leaks of cerebrospinal fluid, herniating of the brain, infections, adhesions, and scarring of the brain cortex. In this research, the dural defect was substituted by autografting with fibrin glue because sometimes the defect of the dura cannot be closed by suturing. Yu *et al.* (30). They said the suturing is time-consuming and no thread has all perfect qualities; a dural graft is indicated. The grafted materials are used to substitute the dural defect with either autologous tissue grafts, such as fascia of femoris and periosteal flaps, or allografts, as in lyophilized cadaveric dural grafts, or xenografts, such as using bovine pericardium and submucosa of porcine small intestinal (31). The fibrin glue also provided excellent and smooth fixation between the grafted materials and the hosts dura mater preventing cerebral spinal fluid from leaking with less complication (32). However, in this article, we used two types of autograft tissues, including the temporalis muscle fascia and peritoneum, with fibrin glue to bind both grafted tissues with the dura of the host.

The use of the peritoneum and fascia of temporalis muscle as autografted tissues to repair dural defect in this work, provided relatively good reconstruction for dural defect where all animals postoperatively showed normal levels of exercise with normal appetite, and they recovered entirely and still lived during all periods of the study without any clinical complication. Additionally, no cerebrospinal fluid accumulation or discharge at the site of operation appeared, and this may be due to the grafted materials had

healed entirely with the host dura mater and prevented the occurrence of some complications, especially cerebrospinal fluid leaks and brain tissue infection or damage. The Klekamp (33) reported that clinical improvement appeared after duraplasty in a short time regardless of the type of grafted materials, whether it is autologous or non-autologous subjects. Both grafted tissues are characterized by absorption, good tensile strength, less cost, and inflammatory reaction. It can be obtained at any time, and this resembled Wang *et al.* (27) Fontana *et al.* (28) Van Calenbergh *et al.* (29) Forseth *et al.* (30) Yu *et al.* (31) Sharma *et al.* (32) Klekamp (33), Pogorielov *et al.* (34), Grotenhuis (35), Rosen *et al.* (36), Biroli *et al.* (37), Yamada *et al.* (38), where many grafted materials utilized to close the induced dural defect produced excellent results. These grafted materials must have some essential characteristics, such as they should be improved the regeneration of tissues and resorb, providing a good scaffold for the formation of neodura, having less inflammatory reactions, not being rejected, and being strong enough to provide a watertight seal without tearing, and easy to apply and less costly.

Any subcutaneous cerebrospinal fluid collection may prevent appropriate wound healing and develop into infection and cutaneous fistula. In addition, the formation of pseudo meningocele leads to abnormal neurological signs (39). In all groups, no cerebral spinal fluid leaks were observed where the results of cerebral spinal fluid pressure were located within the normal range, and no significant difference appeared in all groups. This good result may be due to the effectiveness of fibrin glue with dural substitutes that are used in this work, where the authors De almeida *et al.* (40), Nishihira and McCaffrey (41), Siedentop *et al.* (42), and Jankowitz *et al.* (43) mentioned that the fibrin sealant could be used alone or with sutures and dural substitutes to provide correct closure to the dural defect and prevent cerebral spinal fluid leaks and other complications.

The gross changes in both groups showed complete healing where the craniectomy site was entirely sealed with granulation tissue externally and both grafted tissues connected with host dura mater internally. Also, no adherence showed between grafted tissues and the brain relatively. In addition, the dural defect size was reduced at the end of the study, where the authors Zhi-Dong *et al.* (44) revealed that the boundary between the implanted dura mater and surrounding host dura mater was visible three months after implantation. However, there is good adherence between grafted tissue and host dura, and these boundaries between the hosts dura mater and implanted material became distinguishable morphologically for ten to twelve months. Also, the authors' Bosacco *et al.* (39) reported that any cerebral spinal fluid collected subcutaneously may retard the healing process.

Microscopically, in both groups, the histological sections during the early stages of the healing process were represented by the proliferation of connective tissue, fibroblast proliferation, infiltration of inflammatory cells, and new blood vessel formations, but the rate of inflammatory cell infiltration was higher relatively in the second group than the first group. At the end of the study, both grafted materials were interlocked with the dura mater of the host and covered with mature connective tissues. In addition, there is a reduction in inflammatory cell infiltration. Generally, the grafted materials used to repair dural defects in dogs are gradually biodegraded and replaced by endogenous tissue relatively equal to the host dura mater without developing any abnormal pathological feature. These good results were mentioned by Zhi-Dong *et al.* (44), where the material used for grafting should have as much as possible good tissue compatibility with ideal mechanical characteristics to facilitate invasion of host cells and finally replace the implanted by new biological tissue having the same functions of normal dura mater.

Generally, the fascia of the temporalis muscle provided promising results in repairing the dural defect, and this agreed with Dufrane *et al.* (45), Zerris *et al.* (46), and Shimada *et al.* (47) that the fascia temporalis belonged to the native autologous tissue grafts and was used as a dural substitute because it did not provoke intense inflammatory or immunological reactions. However, it may cause more scar tissue formation. Also, the Tachibana *et al.* (48) showed that fascial tissue graft can be healed with the fibrous tissue without blood supply from an overlying vascularized flap.

Application of the peritoneum piece also provided an excellent seal to the dural defect, where the histopathological section revealed good fibroblast proliferation and deposition of connective tissue with more angiogenesis. These features resemble (49,50) where the peritoneum tissue can restore tissue because the cells of peritoneal tissue can be differentiated into other cells such as fibroblasts and myoblasts. In addition, the Allawi and Alkattan (50), and Nebras and Asmaa (51) showed the peritoneum cells can secrete several growth factors, such as fibroblast growth factor (FGF), transforming growth factor (TGF), and vascular endothelial growth factor (VEGF), which have an essential role in the healing process. Applying a piece of the peritoneum as an autograft was characterized by being easy to obtain, very safe, and suitable size can be obtained quickly, especially in emergencies, and cheaply. However, an additional incision was needed to obtain the graft tissue (52-54). The Yin (54), Suwajo *et al.* (55), and Seung-Hwan *et.al* (56) reported that the peritoneum autograft was also used to repair some conditions in blood vessels, urogenital organs, and lungs. Additionally, the authors Nicolas (52), Castillo *et al.* (53), Yin (54), Suwajo *et al.* (55), Seung-Hwan *et al.* (56), and Uema *et al.* (57) mentioned that applying the peritoneum as a piece of auto graft on the anastomosis site accelerates and improves the healing process and prevents leakage.

In both groups, the percentage of protein, sugar, and total cell counts are located within the normal range after

implantation of the different types of biomaterials, and there is no significant difference between both groups statistically, where any increase or decrease in the protein or glycose of cerebral spinal fluids was indicated to the presence of leaks, infection, bleeding and inflammation (58).

Although the use of some autologous tissues is limited due to disruption of graft supply, the complexity of the surgical operation, time-consuming, applied additional trauma, and adhesion between the piece of autologous tissue with brain tissue (59), good results were obtained through using fascia of temporalis muscle and peritoneum with fibrin glue during this study.

Conclusions

We could use temporalis muscle fascia and peritoneum as autografted tissues with fibrin glue to substitute cerebral dura mater defects in dogs.

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Conflict of Interests

The authors declare that there is no conflict of interest regarding the publication of this paper.

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إصلاح عيب الجافية الدماغية عن طريق لفافة العضلات الصدغية والصفاق كأنسجة مطعمة ذاتيا في الكلاب

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الخلاصة

تم تصميم هذا البحث للمقارنة بين كفاءة لفافة العضلة الصدغية والصفاق كأنسجة ذاتية الطعم مع غراء الفيبرين لإصلاح أذى الجافية المستحدث في الكلاب. تم استخدام ثمانية عشر كلبا بالغا وتم تقسيمهم عشوائيا إلى مجموعتين متساويتين. في جميع الحيوانات تم عمل أذى بقطر حوالي (١ × ١ سم) في الأم الجافية الدماغية. في المجموعة الأولى تم إغلاق الأذى بلفافة العضلة الصدغية، بينما في المجموعة الثانية تم الأذى بقطعة من الصفاق. تم تقييم النتائج من خلال مراقبة العلامات السريرية بعد العملية الجراحة، التغيرات العيانية والنسجية المرضية وقياس ضغط السائل الشوكي الدماغي مع تحليل العد الكلي للخلايا ومستوى الجلوكوز والبروتين للسائل الشوكي الدماغي في اليوم ٦٠،٣٠،١٥ بعد العملية ولكلا المجموعتين. بقيت جميع الحيوانات على قيد الحياة بعد إجراء العملية دون حدوث أي مضاعفات خاصة المتعلقة بظهور العلامات العصبية غير الطبيعية. عيانيا تم انسداد موقع العملية في كلا المجموعتين تماما ولم يلاحظ أي تسرب للسائل الشوكي الدماغي بالإضافة إلى ذلك تم انسداد أذى الجافية داخليا حيث تم اتصلت الأنسجة المطعمة تماما مع الأم الجافية للمضيف. تمثلت التغيرات النسيجية المرضية في تكوين النسيج الضام مع ارتشاح الخلايا وحيدة النواة الالتهابية وتكوين الأوعية الدموية وظهر ضغط السائل الشوكي الدماغي، العد الكلي للخلايا، مستوى الجلوكوز والبروتين ضمن المعدل الطبيعي خلال جميع فترات الدراسة في كلا المجموعتين. بالاستنتاج يمكن استخدام كلا الأنسجة المطعمة ذاتيا مع غراء الفيبرين لإصلاح أذى الجافية في الكلاب.