DEGENERATIVE LUMBAR SPINE DISEASE: ASSESSMENT OF CHANGES IN EPIDURAL INFUSION PRESSURE AND CLINICAL OUTCOME AFTER EPIDURAL STEROID INJECTION

AL-SAYYED HASAN FAHMY AL-SAYYAD, MOHAMMAD HASSAN ALI AND ESSAM MOHAMMAD HEMMAT ABDUL-HAMIED***

Rheumatology & Rehabilitation, Anesthesiology* and Radiodiagnosis** Departments, Zagazig University Faculty of Medicine

KEY WORDS: DEGENERATIVE LUMBAR SPINE DISEASE, EPIDURAL STEROID INJECTION, EPIDURAL INFUSION PRESSURE, CLINICAL OUTCOME.

ABSTRACT

Objective: *Assessment of the effect of epidural steroid injection on epidural infusion pressure and clinical outcome variables in low back pain patients due to degenerative lumbar spine disease.*

Methodology: *This study was conducted on 40 patients, 29 males and 11 females complaining of radicular low back pain with radiological evidence of degenerative lumbar spine disease referred from Rheumatology & Rehabilitation Department to the Pain Clinic of the Anesthesiology Department of Zagazig University Hospitals. They were classified into two groups:* ***Group I:*** *patients who exhibited lumbar degenerative spine disease (DSD) for less than 1 year (early DSD).* ***Group II:*** *patients who exhibited lumbar degenerative spine disease (DSD) for more than 1 year (advanced DSD). At starting therapeutic intervention and after 6 weeks of treatment, low back pain intensity, tension sign (straight leg raising test), functional disability, lumbar mobility and infusion epidural pressure were assessed for both groups.*

Results: *group I (early DSD): A significant decrease in epidural infusion pressure from baseline level was observed after epidural steroid injections ($p<0.05$); significant*

improvements in pain, disability, lumbar mobility and tension sign (Straight leg raising test) were observed when comparing baseline values with that at the end of treatment. Group II (advanced DSD): insignificant decrease in epidural infusion pressure from baseline level was observed after epidural steroid injections ($p>0.05$). Also, insignificant improvements in pain, disability, lumbar mobility and tension sign (Straight leg raising test) were observed when comparing baseline values with that at the end of treatment ($p>0.05$).

Conclusion: *Epidural steroid injections may be of benefit to patients with early DSD whose symptoms do not improve with more conservative management. They may serve as a low-risk alternative to surgery in patients with degenerative lumbar spine disease. But the role of steroid injections not to replace physical activity and therapy; rather, steroids serve to control pain so that patients can work with therapists on their biomechanics and improve their functional outcomes. Further researches must be done to clarify more information about the mechanism of action of epidural steroids in treatment of spine disease, it's effect on epidural infusion pressure and to define accurately the relative indications and clinical features that predict success with epidural steroid injections (ESI) therapy.*

INTRODUCTION

The classic symptoms of a herniated nucleus pulposus are described as back pain followed by pain and paresthesias radiating to the leg. Various structures in and around the spine have been found to be responsible for pain. Specifically, pain generators exist in the outer third of the annulus fibrosis, the facet synovium, anterior longitudinal ligament, posterior longitudinal ligament, nerve roots, nerves and muscles. Disc herniation may cause pain by mechanical irritation of these structures. Additionally, pain may be caused by inflammatory components that occur with disc herniation. Disruption of the annulus fibrosis causes leaking of the nucleus pulposus into the spinal canal, which contains various irritants to tissue, including glucoproteins, phospholipase A2, and nitric oxide, which in turn cause an inflammatory response in and around the pain sensitive nerve tissues *(Shahbandar & Press, 2005).*

One common treatment for low back pain is the epidural steroid injection. The purpose of an epidural steroid injection is to deliver medication directly to the affected nerve roots *(Cluff et al., 2002)*.

Corticosteroids delivered into the epidural space is able to attain high local concentrations *(Singh & Manchikanti, 2002)*. Yet the mechanism of action of epidural steroids in the treatment of pain associated with spine disease is poorly understood *(Dunbar et al., 2002)*.

Several studies of epidural infusion pressure have been performed in normal, obese and pregnant patients *(Hodgkinson & Husain, 1981 and Messih, 1989)*.

Recent studies demonstrate outflow obstruction of the epidural space with increase infusion epidural pressure in patients with degenerative spine disease *(Dunbar et al., 2002)*.

The therapeutic effects of epidural steroid injections are attributed to an inhibition of the synthesis or release of pro-inflammatory substances *(Manchikanti, 2002)*.

Aim of the Work:

The aim of this study was to assess the effect of epidural steroid injection on epidural infusion pressure and clinical outcome variables in patients with low back pain due to degenerative lumbar spine disease.

PATIENTS AND METHODS

This study was conducted on 40 patients; 29 males and 11 females complaining of radicular low back pain with radiological evidence of degenerative lumbar spine disease referred from Rheumatology & Rehabilitation department to pain Clinic of anesthesiology department Zagazig University Hospitals. Degenerative spine disease was defined as that described by the radiologist as spinal stenosis attributable to disc herniation with or without facet hypertrophy significant enough to occlude a neural foramen at any lumbar level.

The patients of this study were classified into two groups:

Group I: 20 patients (15 males and 5 females with a mean age of 45.9 ± 5 years) who exhibited lumbar degenerative spine disease for less than 1 year (early DSD)

Group II: 20 patients (14 males and 6 females with a mean age of 46.2 ± 4 years) who exhibited lumbar degenerative spine disease for more than 1 year (advanced DSD).

Exclusions from Study:

1) Symptoms requiring early surgical treatment (severe motor weakness).
2) Structural spinal deformities (scoliosis, spondylolisthesis).
3) Symptoms from causes other than a herniated degenerative nucleus pulposus.
4) Undergone low back surgery or chemonucleolysis.
5) Received any spinal injection.
6) Patients older than 60 years.

All patients were subjected to:

1) Complete history taking.
2) Thorough clinical examination.
3) Radiological diagnosis by MRI or CT.
4) Evaluation of clinical outcome measures:

a- Pain intensity: was assessed using the visual analogue scale (VAS), the total score vary from 0 (no pain) to 100 mL (severe pain).

b- Disability: according to *Oswestry* low back pain disability score (Wolfe, 1995). The Oswestry questionnaire included 10 questions regarding back pain that induced disability in daily functions and life activities (Personal care) such as dressing, washing, lifting, walking, sitting, standing, sleeping, sex life, social life and traveling. For each question, the subjects selected one number from the scale of 0 to 5, which best described their disabilities. In scale, 0 indicated no disability and 5 indicated total disability. After finishing the 10 questions, the score % is calculated.

c- Tension sign (straight leg raising test) was measured by degrees.

d- Lumbar mobility: According to Schober test in cm *(McRae, 1992).*

(5) Epidural steroid injection and measurement of epidural infusion pressure:

- Sterilize the back of the patients.

- Epidural needle placement (the level of insertion to be as close as possible to the level of pathology).

- Identification of epidural space by loss of resistance to 2 ml saline using 17 gauge Tuohy needle.

- Pressure measurement and infusion apparatus was attached to epidural needle by stopcock side arm.

- Infusion carried out by infusion pump syringe and pressure measurement carried by invasive pressure transducer *(Capto SP*

840 attached to a monitor, Siemens ST 8000, Germany).

- The patients receiving 3 epidural injections of 10 mL bolus of mixture of 8 mL of bupivacaine 0.125% and 2 mL of methyl prednisolone (40mg/mL) at 6 ml/minute every 3 days and baseline epidural infusion pressure was measured during first epidural injection.

- After 6 weeks from last epidural steroid injection, all patients are re-evaluated for the clinical outcome measures and the epidural infusion pressure was measured again during patients receiving one epidural injection of the same above mixture volume of local anesthetic and steroid agents by the same infusion rate.

Statistical analysis:

The results of the study were statistically analyzed on a standard computer program using the student's "t" test for paired and unpaired data.

RESULTS

The first group included 20 early DSD patients (less than 1 year); 15 males and 5 females with mean duration of symptoms 6.7 ± 2.2 months. The second group included 20 advanced DSD patients (more than 1 year); 14 males and 6 females with mean duration of symptoms 25 ± 8 months. Both groups were similar in age. The most common radiculopathy in the two groups was at L4-5 (75% versus 65% of patients in group 1 and group 2 respectively) and the sensory neurological deficit was the most common finding in the two groups (50% versus 60% of patients in group 1 and group 2 respectively) table (1).

Table (1): Demographic information and baseline clinical findings of 40 patients with DSD.

Data		Group I	Group II
Age	(mean ± SD years)	45.9 ± 5	46.2 ± 4
Sex	Males/ Females	15/5	14/6
Symptoms duration	(mean ± SD, range/months)	6.7 ± 2.2, 3-9	25 ± 8, 15-36
Radicular level affected	- L4 – 5 (%) - L5 – S1 (%)	15 (75) 5 (25)	13 (65) 7 (35)
Neurological deficits	- Motor % - Sensory % - Reflex %	2 (10) 10 (50) 6 (30)	6 (30) 12 (60) 10 (50)

Table (2): Epidural pressure and clinical findings at the entry and at the end of

study among 20 patients with early DSD.

Data	At entry of study Mean ± SD	At the end of study Mean ± SD	t	p value
Epidural pressure (mmHg).	27.6 ± 5.2	18.5 ± 3.2	2.58	< 0.05 S
Pain score VAS (0- 100mm).	41.2 ± 6.2	25.4 ± 6	8.2	< 0.001 HS
Disability score (% score).	45.7 ± 12	25.5 ± 9	6.02	< 0.001 HS
Lumbar mobility (Schober test in cm).	11.2 ± 0.32	14.2 ± 3	- 2.1	< 0.001 HS
Tension sign (SLR test by degree).	44.5 ± 7.2	65.5 ± 7	- 9.4	< 0.001 HS

and tension sign; marked improvement were observed at the end of the study and the difference was statistically significant in group 1 (table 2).

Insignificant differences were observed at the end of the study regarding patients epidural infusion pressure, pain, disability, lumbar mobility and tension sign in group 2 (table 3).

Regarding epidural infusion pressure; a decrease in epidural infusion pressure after epidural steroid injection (6 weeks from the last steroid injection) was found in patients with early DSD (group 1) and the difference was statistically significant (baseline epidural pressure was 27.6 ± 5.2 and after steroid therapy was 18.5 ± 3.2, $p<0.05$). Regarding patients pain, disability, lumbar mobility

Table (3): Epidural pressure and clinical findings at the entry and at the end of study among 20 patients with advanced DSD.

Data	At entry of study Mean ± SD	At the end of study Mean ± SD	t	p value
Epidural pressure (mmHg).	32.8 ± 7.1	31.7 ± 6.2	0.5	> 0.05
Pain score VAS (0- 100mm).	50.7 ± 3.2	49.6 ± 2.1	1.3	> 0.05
Disability score (% score).	52 ± 7	49.7 ± 3	1.2	> 0.05
Lumbar mobility (Schober test by cm).	14.8 ± 0.41	15.2 ± 1	- 1.3	> 0.05
Tension sign (SLR test by degree).	39.5 ± 8	40.4 ± 6	- 0.4	> 0.05

the second group had marked improvement, 6 (30%) of patients in the first group versus 4 (20%) of patients in the second group had moderate improvement and 7 (35%) of patients in the first group versus 15 (75%) of patients in the second group had no

This study demonstrated that epidural steroid injection were beneficial for 65% of DSD patients in the first group and for 25% of DSD patients in the second group as following: 7 (35%) of patients in the first group versus 1 (5%) of patients in

difference was statistically significant (baseline epidural pressure was 27.6 ± 5.2, 32.8 ± 7.1 and after steroid therapy was 18.5 ± 3.2, p< 0.05, 31.7 ± 6.2 P > 0.05 in group one and group two respectively (figure 2).

response (figure 1).

Regarding epidural infusion pressure, a decrease in epidural infusion pressure after epidural steroid injections (6 weeks from last injection), was found in patients with early DSD and the

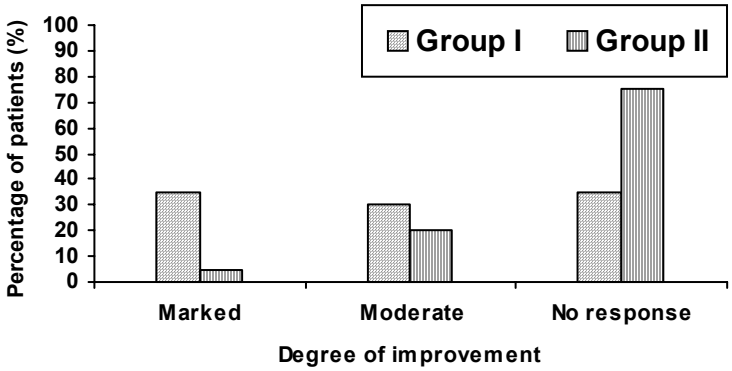

Fig. (1): Degree of improvement of 40 patients at end of study.

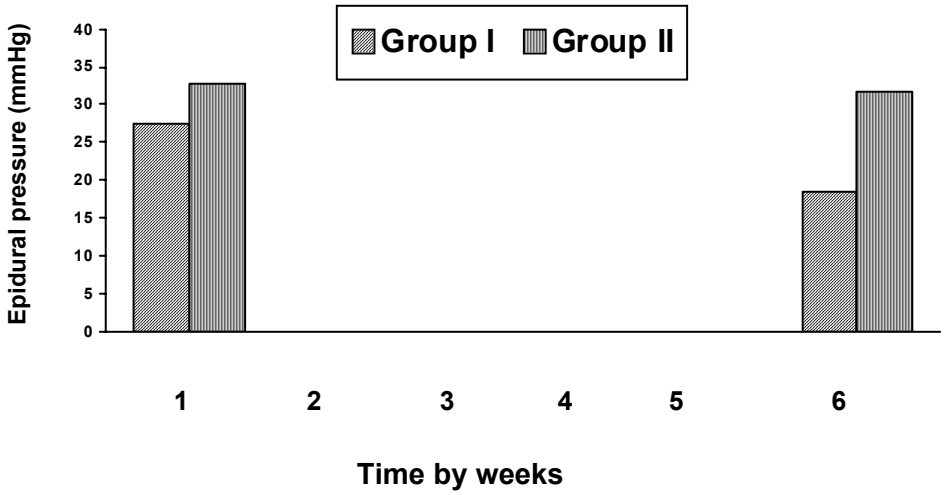

Fig. (2): Show value of epidural pressure at the entry and at the end of study in both groups.

DISCUSSION

The effects of epidurally administered corticosteroids may be due to their ability to inhibit the synthesis of prostaglandins, their anti-inflammatory effects and their ability to inhibit ectopic discharges from injured sensory nerves. Local anesthetics exert their analgesic effects by blocking the conduction in nerves and suppressing the ectopic signal generation in injured nerves. In addition to providing temporary pain relief, local anesthetics may provide prolonged benefit by interrupting the cycle of pain *(Cluff et al., 2002).*

In the present study we aimed to evaluate the effect of epidural steroid injection on epidural infusion pressure and clinical outcome variables in patients with low back pain due to early and advanced degenerative lumbar spine disease.

One and half month after last epidural steroid injection; great difference in improvement regarding patients pain, disability, tension sign (SLR test) and lumbar mobility was found in group I (early DSD) than in group II (advanced DSD) tables (2, 3).

Also, our study demonstrated that epidural steroid injection were beneficial for 65% of DSD patients in the first group and for 25% of DSD patients in the second group (figure 1).

So, our results demonstrated the importance of patients selection, reasonable expectations and starting the epidural steroid injection very early as possible. In agreement of our results *Boulu & Benoist (1996)* who advised to start epidural steroid injection very early to prevent persistent nerve root pain due to peripheral and central sensitization.

Our results is also consistent with the findings of *Rocco et al. (1997),* who reported that the spread of liquids in the epidural space would depend on the state of the epidural space, the extent of scarring and the degree of stenosis, both centrally in the canal and laterally in the foramina. Furthermore, the location of the obstruction is paramount; if the obstruction is at the lateral edge of the foramen, the posterior ganglion can be bathed in anesthetic and good anesthesia will result. However, if scarring involves the whole lateral recess and part of the epidural space, there would be no anesthesia on that side.

Curve Crest & Stillwater, (1999) found that spinal steroid injections both epidural spinal injection (ESI) and intrathecal spinal injection (ISI) are beneficial for a small number of

patients with low back pain resulting from advanced lumbar degenerative disc disease.

Buttermann (2004), in study about the effect of spinal steroid injections for DSD (symptoms for more than one year); he found that spinal steroid injections were beneficial for 25% to 35% of patients with advanced DSD.

Clinical studies done by *Mclain et al. (2005)*, supported the efficacy of epidural steroid injection in the treatment of patients with back and leg pain.

Bush et al. (1992) showed that 76% of disc herniation showed partial or complete resolution over 12 months with aggressive conservative management including up to 3 epidural steroid injections.

Saravanakumar et al. (2006) reported that; 49% of patients with radiculopathy benefited from the epidural steroid injection and 31.37% had pain relief lasting for more than 6 weeks.

In contrast *Carette et al. (1997)* found in their study no significant functional benefit of epidural steroid injections.

Also, *Valat et al. (2003)* found in their study that the efficacy of isotonic saline administered epidurally for sciatica can not be excluded, and the epidural steroid injections provide no additional improvement.

The epidural space can be modeled as a collapsible, distensible, leaky reservoir, which resists inflow. The leak from the space is related to the patency of foramina and the ease with which liquid can move to successive compartments. The normal epidural space is seen to be filled primarily with loose adipose tissues and veins. Once the space is filled with liquid, undamped transmission is possible *(Rocco et al., 1997)*.

Pressure change in the epidural space is a complex relationship between subarachnoid pressure, intra-abdominal pressure and epidural venous plexus pressure *(Hodgkinson & Husain 1981)*.

Rocco et al. (1997) concluded that both epidural infusion pressure and resistance are elevated in the diseased epidural space. Thus to measure the dynamic response of the system it is necessary to use a system that offers little resistance to injection as compared with the resistance in the space itself.

Regarding epidural infusion pressure, a decrease in epidural infusion pressure after epidural steroid injections (6 weeks from last injection), was found in patients with early DSD and the

difference was statistically significant. (Baseline epidural pressure was 27.6 ± 5.2, 32.8 ± 7.1 and after steroid therapy was 18.5 ± 3.2, $p< 0.05$, 31.7 ± 6.2 $p> 0.05$ in group one and group two respectively (figure 2).

In agreement, *Dunbar et al. (2002)* who found in their study that DSD patients had significantly increased infusion pressure over normal, which mean outflow resistance or obstruction and also found a significant decrease in epidural infusion pressure after epidural steroid treatment which may reflect the efficacy from epidural steroid injections.

The increase in baseline epidural infusion pressures recorded in patients with DSD may be due to reduction in the elasticity of the epidural space and spinal stenosis, and this lead to increase resistance to inflow and the decreased in the epidural infusion pressure after epidural steroid injections may be secondary to either local anesthetic or steroid or to simply due to infusion a volume of fluid via epidural space.

In our study, the more possible cause of the increase in baseline epidural infusion pressure is spinal stenosis which leads to increase resistance to inflow. In agreement of our study *Usubiaga et al. (1967)* who reported that higher epidural anesthetic levels and higher infusion pressure were evident due to spinal stenosis in patients with DSD when age was not a factor.

On the basis of above considerations, its possible that epidural steroids by its effect in reducing edema and decompressing the spinal canal can lead to decrease the degree of stenosis and this lead to pain relief and decrease in epidural infusion pressure.

Conclusions:

Epidural steroid injections may be of benefit to patients with early DSD whose symptoms do not improve with more conservative management, they may serve as a low-risk alternative to surgery in patients with degenerative lumbar spine disease. But the role of steroid injections not to replace physical activity and therapy; rather, steroids serve to control pain so that patients can work with therapists on their biomechanics and improve their functional outcomes. Further researches must be done to clarify more information about the mechanism of action of epidural steroids in treatment of spine disease and its effect on epidural infusion pressure and to define accurately

the relative indications and clinical features that predict success with epidural steroid injection (ESI) therapy.

REFERENCES

Boulu P and Benoist M (1996): Recent data on the pathophysiology of nerve root compression and pain. Rev. Rhum Eng J; 63.

Bush K, Cowan N, Katz et al. (1992): The natural history of sciatica associated with disc pathology. A prospective study with clinical and independent radiological follow up. Spine17 (10).

Buttermann GR (2004): The effect of spinal steroid injections for degenerative disc disease, The spine journal (4): 495-505.

Carette S, Leclaire R, Marcoux S et al. (1997): Epidural corticosteroid injections for sciatica due to herniated nucleus pulposus. N. Eng 1 Med; 40.

Cluff R, Mohio AK, Cohen SP et al. (2002): The technical aspects of epidural steroid injections: A national Survey. Anesth Analg 8.

Curve Crest B and Stillwater MN (1999): The effect of spinal steroid injections for degenerative disc disease. Mid West Spine Institute.

Dunbar SA, Manijanton P and Philip J (2002): Epidural infusion pressure in degenerative spinal disease before and after epidural steroid therapy. Anesth Analg 20.

Hodgkinson R and Husain L (1981): Obesity, gravity and Spread of epidural anesthesia. Anes Analog 4.

Manchikanti L (2002): Role of neuro-axial steroids in interventional pain management. Pain physician 5.

Mclain RF, Kapural L and Mekhail NA (2005): Epidural steroid therapy for back and leg pain: mechanisms of action and efficacy. The spine Journal (5): 191-201.

McRae R (1992): The thoracic and lumbar spine, In: Clinical orthopaedic examination, 3rd ed., Chap. 8: P. 121. Churchill Livingstone.

Messih M, (1989): Epidural space pressures during pregnancy. Anesthesia 44.

Rocco AG, Philip JH, Boas RA et al. (1997): Epidural space as a starling resistor and elevation of inflow resistance in a diseased epidural space. Reg. Anesth 22.

Saravanakumar K, Plant C and Kabeer A (2006): Epidural steroid injection therapy: Prospective analysis of factors influencing the efficacy. Eur. J. of Pain, Vol (10).

Shahbandar L and Press J (2005): Diagnosis and non operative management of lumbar disc Herniation. Oper Tech Sports Med 13: 114-121.

Singh V and Manchikanti L (2002): Role of Caudal epidural injections in the management of chronic low back Pain. Pain physician 5.

Usubiaga LE, Wilkinski JA and Usubiaga LE (1967): Epidural pressure and it's relationship to spread of local anesthetic solutions in the epidural space. Anesth Analg; 46: 440-446. Quoted from Dunbar et al., 2002.

Valat JP, Giraudeau B, Rozenberg S et al. (2003): Epidural corticosteroid injections for sciatica: a randomized, double blinding controlled clinical trial. Annals of the Rheumatic diseases; 62.

Wolfe F (1995): Health status questionnaires. Rheum Dis Clin North Am; 21.

تحديد التغيرات فى ضغط سريان المحلول العلاجى وبعض الظواهر الإكلينيكية التى تحدث لمرضى تآكل غضاريف الفقرات القطنية بعد الحقن بالكورتيزون من خلال الأم الجافية

السيد أحمدحسن فهمي الصياد - محمد حسان على*- عصام محمد همت عبد الحميد**

أقسام الروماتيزم والتأهيل -التخدير * -الأشعة التشخيصية** - كلية الطب – جامعة الزقازيق

وقد أجريت هذه الدراسة على أربعون مريضا من ألم أسفل الظهر المزمن الناتج عن تآكل غضاريف الفقرات القطنية وقد تم تقسيم هؤلاء المرضى إلى مجموعتين متماثلتين فى العمر.

المجموعة الأولى: وشملت عشرون مريضا بمرض تآكل غضاريف الفقرات القطنية المبكر.

المجموعة الثانية: وشملت عشرون مريضا بمرض تآكل غضاريف الفقرات القطنية المتقدم.

وتم قياس ضغط سريان المحلول العلاجى قبل العلاج الموضعى بالكورتيزون فى فراغ الأم الجافية وتقييم هؤلاء المرضى إكلينيكيا بالنسبة لشدة الألم والإعاقة وحركة أسفل الظهر وبعض الظواهر الأخرى.

وتم حقن هؤلاء المرضى بالكورتيزون لفراغ الأم الجافية 3 مرات بواقع حقنه كل ثلاث أيام وتقييم هؤلاء المرضى مرة أخرى بعد شهر ونصف من نهاية العلاج بالنسبة للظواهر الإكلينيكية وضغط سريان المحلول العلاجى وأوضحت نتائج الدراسة تحسن بدلالة إحصائية فى الظواهر الإكلينيكية كما أوضحت انخفاض ملحوظ فى ضغط سريان المحلول العلاجى من خلال فراغ الأم الجافية وذلك بعد شهر ونصف من خلال العلاج بالكورتيزون عبر فراغ الأم الجافية فى المجموعة الأولى عنه فى المجموعة الثانية. ويتضح من هذه الدراسة أن العلاج الموضعى للكورتيزون من خلال فراغ الأم الجافية فى الحالات المبكرة ذو أهمية إكلينيكية فى تخفيف الألم وتقليل ضغط سريان المحلول العلاجى من خلال الأم الجافية ويكون بديلا أكثر أمنا من الجراحة لمرضى تآكل غضاريف الفقرات القطنية.