

## The Relation Between Lower Spinal Congenital anomalies And Chronic Low Back Pain



Dr. Raof. R. Mirza  
College of Medicine University of Sulaimani  
Kurdistan Region - Iraq

### Abstract

To find out the relation of lower spinal congenital anomalies (CA) to chronic low back pain (LBP) "back pain for more than 3 months". One hundred patients with chronic LBP in whom the cause had not been identified were studied, found that 30 patients (30%) had some kind of CA in their lower spines, while in the other 70 (70%) there were no any identified CA. At the same time 66 normal people who had not suffered from back pain studied on the same standards, found that 19 (28.87%) of them had some kind of CA in their lower spines, while in the rest 47 (71.21%) there were not any evident CA. These figures give a conclusion that there is no definite relation between lower spinal CA and chronic LBP ( $p\text{-value} > 0.8$ ).

**Keywords :-** Low back pain (LBP) and congenital anomalies (CA).

### Introduction

The principal CA of the lower spine include:

1. Sacralization: of the 5th lumbar vertebra; complete or incomplete i.e. "bilateral or unilateral"
2. Lumbarization: of the 1<sup>st</sup> sacral vertebra; complete or incomplete i.e. "bilateral or unilateral"
3. Accessory Sacro-iliac & Lumbar-iliac joint: from broad spinal transverse process articulating with iliac bone <sup>(1,2)</sup>
4. Spina bifida: failure of embryonic neural plate to fold over to form a closed neural tube or of mesodermal tissue fully to invest the

isthmus type neural tube, spina bifida is of 4 varieties (a) spina bifida occulta (b) meningocele. (c) myelomeningocele. (d) rachischisis. In spina bifida occulta the skin and soft tissues are intact <sup>(2)</sup>.

5. Spondylolysis: Fibrous union of the laminar arches.
6. Spondylolisthesis: When the fibrous union in spondylolysis is stretched or torn allowing subluxation of the vertebra. This type of spondylolisthesis is known as

constitute about 50% of all cases, L5 is most commonly affected and occasionally L4 is affected.

Spondylolisthesis may result from 3 other cause:

A: Congenital malformation of superior sacral facets ,known as dysplastic type constitute 20% of cases, it is the most serious type and may associate with spina bifida occulta.

B: Osteoarthritis of the facets joints known as degenerative type :less sever one.

C: Post traumatic <sup>(1,2)</sup>.

7. Congenital spinal stenosis <sup>(2,3)</sup>: As rule of thumb it is recommended that any lumbar sagital diameter below 15 mm. Or above 25 mm be regarded with suspicion. <sup>(4)</sup>

8. Defect in the transverse process of a lumbar vertebra.

9. Hemivertebra: may give rise to sever spinal deformity <sup>(2,3)</sup>.

### **Subjects & Method**

One hundred patients suffering from chronic LBP with out evident cause were collected among those who attended the rheumatology & rehabilitation clinic in sulaimany from 21 st of June to 23rd of November 1997, and 66 normal controls who had not complained of LBP for more than 3 days or taken any medication for LBP in there life were collected from hospital staff, general population & patients who attende.l the

rheumatology & rehabilitation clinic for simple complaints with no any LBP.

All patients and control group have been seen by a consultant physician in Rheumatology & Rehabilitation medicine, patients suffering from LBP for more than 3 months been included in this study with the following exceptions:

1. Patients with sciatic pain or past history of sciatica.
2. Patients gave history of precipitating cause for their LBP like: lifting a heavy weights , undue exercise, trauma, etc.....
3. Evidence of any systemic disease.
4. Presence of any probable underlying causes.

History has been taken from all the patients in detail regarding site, type, radiation, aggravating, relieving factors of pain & for features of neurological claudication.

Clinical examination has been done for spinal deformities, local tenderness, and neurological deficits in lowers limbs and straight leg raising test.

Pathological conditions of the hip were excluded clinically.

All the patients investigated for ESR, WBC & Hb. to exclude systemic rheumatological disease.

The CA was proved by plain X-ray of the lumbosacral spine, both AP and lateral views were taken for the patients & control group.

All the X-ray films had been seen in conjunction with a consultant radiologist in the radiology department of sulaimany general hospital who wrote a report on each film.

(30) Patients (30%) had some sort of CA in the lumbar or sacral region demonstrated on the plain radiographs.

While the rest 70 (70%) of the patients had no evident CA, the following tables [1,2,3&4] show the data collected in details.

Table(1) shows the clinical features, evidence of high ESR (\*) and radiological Lumbar Spondylotic changes in the 100 patients with LBP "Table 1:A" & in the 30 patients with CA "Table 1:B":

Table 1:A

	Features Of Patients With LBP	No.: 100	%
1	Pain aggravated by exertion	92	92%
2	Pain relieved by rest	89	89%
3	Local tenderness	79	79%
4	Radiation of pain to lower limbs	58	58%
5	Positive straight leg raising test	11	
6	Neurological claudication	14	14%
7	High ESR	14	14%
8	Lumbar spondylosis	15	15%
9	Neurological deficit	4	4%
10	Deformities	1	1%
11	Other features	8	8%

1	Pain aggravated by exertion	26	86.6%
2	Pain relieved by rest	29	96.6%
3	Local tenderness	26	86.6%
4	Radiation of pain to lower limbs	22	73.3%
5	Positive straight leg raising test	6	20%
6	Neurological claudication	4	13.3%
7	High ESR	3	10.0%
8	Lumbar spondylosis	2	6.6%
9	Neurological deficit	0	0.0%
10	Deformities	0	0.0%
11	Other features	8	26.6%

(\*) ESR: Erythrocytes Sedimentation Rate.

Table (2) Shows type of CA found , their frequencies & percentages:

	Types of CA	No.	%	Age	F	M	Notes
1	Unilateral sacralization of L5	8	26.66%	25-62	7	1	Rt : Lt =4:4
2	Bilateral sacralization of L5	4	13.33%	18-31	2	2	
3	Unilateral lumbarization of S1	1	03.33%	37	1	0	Rt : Lt =1:0
4	Bilateral lumbarization of S1	2	06.66%	25,36	2	0	
5	Spondylolysis	7	23.33%	22-47	6	1	L5:L4=6:1,5of them combined with spondylolisthesis
6	Spondylolisthesis	5	16.66%	30-47	5	0	L5/S1=4 L4/L5=1
7	Spina bifida occulta	6	20.00%	19-50	5	1	S1=5,L5=1 1 patient with lysis and listhesis
8	Limbus vertebra	2	06.66%	28,45	1	1	L4: 2
9	Spinal stenosis	1	03.33%	56	0	1	
	<b>Total</b>	<b>30</b>	<b>100%</b>	<b>18-62</b>	<b>23</b>	<b>7</b>	

\* Only 8 patients were above 40y and 2 patients were above 50y but more than 2/3 of the patients were below 40y.

\* 5 Patients had spondylolysis with spondylolisthesis.

\*Only 1 patient had spondylolysis with spondylolisthesis combined with spina bifida.

\*F= Female; M= male.

**Table (3) Shows types of CA found, their frequencies and percentages in control group:**

	Types of CA	No.	%	Age	F	M	Notes
1	Unilateral sacralization of L5	1	05.26%	23	0	1	Lt.
2	Bilateral sacralization of L5	5	26.31%	23-46	4	1	
3	Unilateral lumbarazation of S1	1	10.52%	42	1	0	Lt.
4	Bilateral lumbarazation of S1	2	15.87%	23,30	1	1	
5	Spondylolysis	3	15.87%	21-48	1	2	of L5
6	Spondylolisthesis	3	26.31%	30-58	3	0	No spondylolysis
7	Spina bifida occulta	5	00.00%	17-58	2	3	
8	Defect in transverse process of L1	1	05.26%	24	1	0	
	<b>Total</b>	<b>19</b>	<b>100%</b>	<b>17-58</b>	<b>12</b>	<b>7</b>	

\* No spondylolysis that associated with Spondylolisthesis found in the control group.

\* Combined CA found in two persons, spina bifida with spondylolysis in one person and spinabifida with Spondylolisthesis in the other person.

\* 28.87 % of the normal controls had evident CA.

**Table (4) : Combined table shows types of CA, their frequencies and percentages to the CA found and to the total No. of patients and controls.**

	Types of CA	Patients Group			Control group		
		No.	% to CA found	% to total patients	No.	% to CA found	% to total controls
1	Unilateral sacralization of L5	8	26.66%	8 %	1	05.26%	01.51 %
2	Bilateral sacralization of L5	4	13.33%	4%	5	26.31%	07.57 %
3	Unilateral lumbarazation of S1	1	03.33%	1%	1	10.52%	01.51 %
4	Bilateral lumbarazation of S1	2	06.66%	2%	2	15.87%	03.03 %
5	Spondylolysis	7	23.33%	7%	3	15.87%	04.54 %
6	Spondylolisthesis	5	16.66%	5%	3	26.31%	04.54 %
7	Spina bifida occulta	6	20.00%	6%	5	00.00%	07.57 %
8	Limbus vertebra	2	06.66%	2%	0	00.00%	00.00 %
9	Spinal stenosis	1	03.33%	1%	0	00.00%	00.00 %
10	Defect in transverse process of L1	0	0	0%	1	05.26%	01.51 %
	<b>Total</b>	<b>30</b>	<b>100.00%</b>	<b>30 %</b>	<b>19</b>	<b>100.00 %</b>	<b>28.87 %</b>

### Discussion

Back or neck pains are the chief complaint of considerable percentage of the patients presenting in a primary care setting. They are a major challenge for occupational medicine and rheumatology (5). LBP which affect nearly every one of us some stage of our active adult life, is one of the most common ailments afflicting mankind (6). Non-specific LBP of mechanical origin is second only to common cold as a cause of self-limiting symptoms and disability in the community. Chronic nonspecific LBP of more than 3 months duration accounts for less than 3% of all cases (7). Finding the cause might be a key for management. The aim of this study is to find out the relation of CA in the lower spinal vertebrae to the chronic LBP, to achieve this we studied two groups, the 1<sup>st</sup> consists of 100 patients with chronic LBP without evident cause, while the 2<sup>nd</sup> group consists of 66 normal people who have not suffered from a significant LBP. The comparison of the results from both groups showed that the difference in percentage of CA found between the two groups was 1.13% (as it was 30% in patients group and 28.87% in control group) and P(probability)-value was ( $> 0.8$ ) which is statistically not significant (See table 5).

**Table (5): P value for total CA in patients and control group:**

Patients and Controls	No C.A.	With C.A.	P. value
Chronic LBP patients	70	30	> 0.8
Control persons	47	19	
Total	117	49	

Although unilateral sacralization of L5 and spondylolysis (whether associated with spondylolisthesis or not) constitute 50% (26,66% and 23.33% respectively) of all CA in chronic LBP patient but in comparison with the controls they were statistically not significant to be regarded as definite causes of chronic LBP (P-value for unilateral sacralization was  $> 0.05$  and for spondylolysis was  $> 0.5$ ). (see table 6 and 7).

**Table (6): P-Value of Unilateral Sacralization in patients and control group:**

Patients and controls	No Unilat. Sacralization	With Unilat. Sacralization	P-Value
Chronic LBP patients	92	8	> 0.05
Control persons	65	1	
Totals	157	9	

**Table: (7) P-Value of Spondylolysis in patients and control group:**

Patients and controls	No Spondylysis	With Spondylo-ysis	P-Value
Chronic LBP patients	93	7	> 0.5
Control persons	63	3	
Totals	156	10	

During our review of literature about lower spinal CA and their relation to LBP we found that little attention been paid to this topic:

M. Thompson (1982)<sup>(1)</sup> considered these anomalies to cause LBP by the following mechanisms:

- 1- Resultant postural strains.
- 2- Ligamentous deficiency.
- 3- Accessory joints which are particularly subject to degenerative changes.
- 4- Increased predisposition to disc lesion.
- 5- Restricted or excessive spinal mobility.

Adams J.C. and Hamblen D.L. (1995)<sup>(2)</sup> regarded that minor variations in the bony anatomy are common in the lumbar and sacral region, but they are not practically important, at the same time they mentioned that the following CA might cause LBP:

- 1- A false joint between the ilium and hypertrophied transverse process may be source of pain.
- 2- Spondylolysis is often symptomless but it is believed that it is sometimes a cause of LBP.
- 3- Spondylolisthesis: in some cases the deformity is interestingly symptomless, when symptoms occur take form of chronic backache with or without sciatica, the back pain is worse on standing.
- 4- Spinal stenosis: produce pain on walking for 10-15 minutes in the gluteal region and in the lower limbs, relieved by sitting or lying with flexed hips, or during prolonged standing.
- 5- Hemivertebra: this anomaly is a rare cause of scoliosis.

In this series the false joint between ilium and hypertrophied transverse process or hemivertebra had not been found, while there was only one case of spinal stenosis in the patients group, which are regarded by some authors<sup>(2,3)</sup> to be causes of LBP.

Apley (1993)<sup>(3)</sup> giving a figure of 5% of population having defects in the pars inter articularis which is usually present by the age of 7 year, this figure is close to ours as it was 7% in patients and 4.55% in control groups.

Apley (1993)<sup>(3)</sup> also mentioned that the slip may only appear some years later, it is difficult to exclude a genetic factor because spondylolisthesis often runs in families and is more common in certain races, notably Eskimos but the incidence increases with age so an acquired factor probably supervenes to produce what is essentially a stress fracture. The condition is more common than usual in those whose spines are subjected to extraordinary stress e.g.: competitive gymnastics. In our patients group presentation was at age range of (22-47)years (and non of them gave a history of trauma or any precipitating factors), this was quite close to control group; age range was (21-48) years.

Schniderman G.A. (1995)<sup>(8)</sup> considered that the presence of neural elements including free nerve endings within the pars defect tissue may be a source of back pain in some patients with symptomatic spondylolysis

A case report (Lucy S.D. 1995)<sup>(9)</sup> considered spondylolisthesis as a cause of LBP in (2)years old girl who presented with LBP and subsequently diagnosed as spondylolisthesis.

Further more in another case report (Heim M. 1995)<sup>(10)</sup> x-ray revealed a hemivertebra at the level of the last sacral segment in 18 years soldier with intact and functioning lumbar and sacral nerves; this finding been regarded as a very rare cause of LBP by the authors<sup>(10)</sup>.

Analysis of the clinical features of our patients showed that the majority of those with CA had a pain aggravated by exertion (86.6 %) relieved by rest (96.6 %) this justify

the postulation that mechanical problem might be a cause of their back pain.

20% of our patients with CA had a limited straight leg raising test without having any precipitating factor for sciatica, this suggests that the root irritation might be caused by the abnormal anatomy resulted from CA, this also mentioned by M. Thomson (1982)<sup>(1)</sup>.

On the some basis mentioned above the neurological claudication of those 4 patients complaining of it could be explained.

In 3 patients we found moderate elevation of ESR that could not be explained by systemic causes.

8 patients were complaining of other features like headache, knee pain, flu-like illness, gynecological problems were not related to the back problem and were not significant enough to think about systemic causes.

During the whole period, the back problem was not interfering with daily activities of any one of the patients and all the patients attended the center by themselves without assistance this indicate non severity of the LBP in all patients group.

### **Conclusion**

According to the figures came out the greater difference between patients group and

control group was in unilateral sacralization of L5 which was 6.5% (8% in the patients and 1.5% in the controls) and the lowest difference was in unilateral lambarization of S1 which was 0.5% (1% in patients and 1.5% in controls). Although the figures looks to be high in the unilateral sacralization of L5 but still it is not reaching the significant statistical level (P. value > 0.05), therefore a conclusion came out that there is no definite relation between CA and chronic LBP.

More detailed studies of each type of CA suggested by a larger sample sizes to find the true relation of each type with chronic LBP independently.

### **Acknowledgment**

We would like to thank Dr. S. J. Ismael for his help in collecting patients, analyzing data and typing this paper.

I also thank consultant radiologists Dr. A.A.M. Ali and Dr. H. Baban for their help in reporting on X-ray films for the patients and control groups.

I also thank Dr. H.A. Rasheed a head of D.O.H. warehouse for supplying X-ray films for the study in spite of economic embargo.

Many thanks will go to technicians in X-ray department for their help and cooperation.

I also thank all patients and control group who share in this study

## References

1. M. Thompson M.D. , F.R.C.P (1993) : Rheumatic diseases collected reports 1954-1983, 3<sup>rd</sup> edition by: The Arthritis and Rheumatism council for research in Great Britain and Commonwealth P 111.
2. Adams J.C. and Hamblen D.L. (1995) : outlines of orthopedics 12<sup>th</sup> edition, Churchill Livingstone, p 171,172,139,169,198-200, 199.
3. Apely, A.G. and Solomonm L. (1993): A system of orthopedic and fracture, 7th edition, London: Butter worth, P375-376, 374,351.
4. Hinck, Hopkin and Clark -1965 (Isadore Meschan: An Atlas of Anatomy Basic to Radiology 1st E. 1975).
5. H. Ralph Schumacher & John H. Klippel & William J. Koopman (1993) : Primer on the Rheumatic Diseases, 10th E, P 276.
6. Robin Mckenzie(1985): Treat Your Own Back, 3rd E, P1.
7. G. Nuki, S.H. Ralston, R. Luqmani: Diseases of the connective tissues, joints and bones in the Davidson's principles and practice of medicine 18th edition, (1999) P 815-16
8. Schniderman G.A., Melain R.F., Hably M.F. , Neilson S.L., Northern California spine and Rehabilitation, Sacramento USA: *Spine* Aug 1995. 15;20 (16) 17614.
9. . Lucey S.D., Grose R. , Department of surgery Brown Grey School of medicine: *J-Pediatr-Orthop*. Mar 1995 Apr; 15(2): 199-201.
10. Heim-M; Marcovich-C: A rare cause of low back pain. *Am-J-Orthop* Mar. 1995; 24(3): 273-5.

## پەيوەندى نيوان شيواوويه زگماكيه كانى بربره كانى خواروهوى پشت له گەن

نازارى دريژخايه نى خواروى پشت

د. رووف رحيم ميرزا كۆليزى پزىشكى

زانكۆي سلیمانى / هەريه كوردستان - عيراق

### پوخته

بۆ دۆزینه وهى پەيوەندى نيوان شيواوويه زگماكيه كانى بربره كانى خواروهوى پشت و نازارى دريژخايى خواروى پشت ( پشت ئيشه بو زياتر له ماوهى سێ مانگ ) ، ئەم تويزينهويه ئەنجام درا له سەر سەد نهخوش كه نازارى دريژخايى خواروى پشتيان هه بوو به بێ ههچ هۆيه كى ديارىكراو ، دەرکەوت كه سى ( ۳۰٪ ) له نهخوشه كان شيواووى زگماكيان هه بوو له بربره كانى خواروى پشتيان ، به لام له ههفتا ( ۷۰٪ ) كهى تردا ههچ شيواوويه كى زگماكى نه دۆزرايه وه . له هه مان كاتدا شهست وشهش كه سى ئاسايى ، كه ههچ پشت ئيشه يان نه بوو ، تويزينه ويان له سهركرا ، له سەر هه مان بنه ما ، دەرکەوت كه نوزده ( ۲۸,۸۷٪ ) يان شيواووى زگماكيان له بربره كانى خواروهوى پشتيان هه بوو ، به لام له چل و چهوت ( ۷۱,۲۱٪ ) كهى تردا ههچ جوړه شيواوويه ك نه دۆزرايه وه . له ئەنجامه ندا ئه وه دەرکەوت كه ههچ پەيوەندىيه كى به رجه سته نيه له نيوان شيواوويه زگماكيه كانى بربره كانى خواروهوى پشت و نازارى دريژخايه نى خواروى پشت (  $p\text{-value} > 0.8$  ).

## العلاقة بين التشوهات الولادية في الفقرات السفلى و ألم أسفل الظهر المزمن

د. رووف رحيم ميرزا

كلية الطب / جامعة السليمانية / اقليم كوردستان العراق

### الخلاصة

لإيجاد العلاقة بين التشوهات الولادية في الفقرات السفلى و ألم أسفل الظهر المزمن (ألم الظهر لمدة تزيد على ثلاثة أشهر)، أجريت هذه الدراسة على مائة مريض ممن يعانون من ألم مزمن في أسفل الظهر بدون وجود أية أسباب معينة ، فوجد أن ثلاثين ( ۳۰٪ ) من المرضى يعانون من التشوهات الولادية في الفقرات السفلى ، بينما في السبعين ( ۷۰٪ ) الآخرين لم يتم العثور على أية تشوهات ولادية . في نفس الوقت أجريت الدراسة على ست وستين شخص طبيعى ممن لم يعانون من أية آلام في أسفل الظهر ، وبنفس الضوابط ، فوجد أن تسعة عشر ( ۲۸,۸۷٪ ) منهم يعانون من التشوهات الولادية ، بينما في الأربعة والأربعين ( ۷۱,۲۱٪ ) الآخرين لم يتم العثور على أية تشوهات ولادية . من هذه النتائج يتبين عدم وجود أية علاقة وثيقة بين التشوهات الولادية في الفقرات السفلى و ألم أسفل الظهر المزمن .