KKU Res.j. 2015; 20(4) : 465-479
http://resjournal.kku.ac.th

Clinical Lumbar Instability and Core Stabilization Exercise: A Literature Review

Pattanasin Areeudomwong[1*]

[1]*Department of Physical Therapy, School of Health Science, Mae Fah Luang University, Chiang Rai, Thailand 57100*

Abstract

Clinical lumbar instability is increasingly recognized as one of the significant causes of chronic low back pain. The patients with clinical lumbar instability require the effective exercise intervention to improve the stability of their lumbar spine. This article reveals the etiology and clinical diagnosis of clinical lumbar instability, the effective treatment as core stabilization exercise (CSE) and also provides some relevant researches on CSE for improving outcome measurements in patients with clinical lumbar instability. Commonly, clinical signs and the specific clinical tests including instability catch sign, apprehension sign, painful catch test and prone instability test are considered as clinical diagnostic criteria due to absence of radiological findings in patients with clinical lumbar instability. Moderate to high impacted evidences on the effectiveness of CSE to improve pain-related outcomes and deep trunk muscle activation have been reported in patients with clinical lumbar instability but further studies regarding long-term effectiveness of CSE and the use of precise instrument to detect deep trunk muscle activation are still required.

Keywords: *clinical lumbar instability, core stabilization exercise, diagnostic criteria, stability*

1. Introduction

Low back pain (LBP) is a major health and socioeconomic problems worldwide (1). LBP can be classified into heterogeneous subgroups and one significant subgroup is clinical lumbar instability (2-5). In Thailand, the prevalence of clinical lumbar instability was 13% (1). Pain, functional disability and altered trunk muscle activations resulted from excessive lumbar intersegmental motion (3,5-6). Nevertheless, clinical lumbar instability is often absent of radiological findings (7) and clinical criteria using the specific signs or symptoms are beneficial for diagnosing clinical lumbar instability (7-11).

Although exercise therapy is an acceptable approach to eliminate problems of LBP, it is difficult to conclude that a specific type of exercise is better than another. As new treatment approaches are emerging, a better realization of the effect of each technique on patient problems is considered as worthwhile for clinical

researches (12). Exercise therapy which emphasizes the spinal stabilization is hypothesized to be effective for clinical lumbar instability to improve abnormal excessive movement of the lumbar spine. Recently, core stabilization exercise (CSE) has been designed to improve control skill of neuromuscular system and increase segmental stability of lumbar spine (12-13). Thus, CSE could be a choice of treatment for clinical lumbar instability.

2. Lumbar stability and lumbar instability

Panjabi (14) suggested a model of a spinal stabilizing system including three stabilizing subsystems: the passive, active and neural control. All subsystems have a proper function and interaction to provide spinal stability. The motion segment is capable to support a load and maintain normal pattern of displacement within its physiologic limits, it is considered stable (3,14).

The passive stabilizing subsystem composes of vertebral body, intervertebral disc, facet joint, facet joint capsule, spinal ligaments and passive mechanical tension from the spinal muscles (15). Although this subsystem provides stabilizing role throughout the range of spinal motion, especially in end-range of spinal motion to restrain excessive motion of the spine, it can support a limited load approximately 100 Newton that is less than body weight (14,15). Thus, the second subsystem as an active stabilizing subsystem is required to support body weight and additional loads, especially during trunk movements (3-4). The active stabilizing subsystem comprises of the spinal muscles and their tendons surrounding the spinal column. This subsystem provides mechanical stability for loads exceeding 1,500 Newton (14-15). It is a major dynamic stabilizer to generate forces to support the lumbar motion segments. There are two systems of trunk muscles: global and local muscles which may influence human stability and movement. The global muscle system (superficial trunk muscles) consists of rectus abdominis, external oblique, iliocostalis lumborum pars thoracis and lattisimus dorsi muscles. These muscles are mobilizing muscles which demonstrate discontinuous activation to produce general gross trunk stabilization and generate large torque for trunk movement as well as spinal compression (2). Moreover, they are also important for shock absorption of the loads and balancing external loads (2). However, these muscles control spinal orientation and movement by their activations in a directional specific response (2). On the contrary, the local muscle system (deep trunk muscles) consists of transversus abdominis, lumbar multifidus, semispinalis, rotatores, interspinalis, intertransversarii, inferior fibers of internal oblique, quadratus lumborum and diaphragm muscles. They directly attach to the lumbar vertebrae that provide the lumbar segmental stability and directly control each lumbar motion segment. They provide the local stabilizing effect that control intersegmental motion, and maintain mechanical stiffness of the spine by increasing their stiffness. Furthermore, the local muscle system activation is independent of directional movement and continuously actives through the movement (2). Therefore, it plays an important role in providing both static and dynamic stability to the lumbar motion segments. The last subsystem is neural control subsystem. It has the complex task

of receiving the proprioceptive afferent from mechanoreceptors that present in the passive stabilizing subsystem (spinal ligaments, intervertebral discs and facet joint capsules) and active stabilizing subsystem (muscle spindles and Golgi tendon organs) to determine the specific requirements for maintaining spinal stability and lastly, adjusting and generating the coordination and activation of the stabilizing muscles depending on the mechanical spinal stability needed (2,14). Inappropriate interaction of the three subsystems or deficit of one or more subsystems can lead to lumbar instability (14).

Lumbar instability has been introduced by Panjabi (4) who conducted the biomechanical study of lumbar instability. He suggested that lumbar instability is abnormal excessive movement beyond the normal movement of the lumbar motion segment that may presents due to the damage of the constrained structures establishing the spinal stability. This state causes a painful sensation and progressive deformity, and neural structures may be at risk eventually.

Lumbar instability can be classified into two types: radiological and clinical (7). Radiological lumbar instability is commonly diagnosed by using lumbar flexion-extension radiographs (16-17). Flexion-extension radiographs can demonstrate slippage or subluxation of the affected vertebrae on the adjacent one (17). Leone et al (16) described the etiology of radiological lumbar instability that intervertebral disc degeneration results in laxity of the interbody and facet joints caused by decreased tensile stress on the facet joint capsules and laxity of ligament responsibility for binding the adjacent vertebrae together. It leads to subluxation of the facet joints during lumbar flexion and extension. Radiological lumbar instability may show facet joint capsules or vertebral ligament damage. It may cause spondylolisthesis. Radiological lumbar instability may result in mechanical deformation of the lumbar spine, intraspinal nerve tissue pain and/or neurologic deficit (9).

The threshold of radiological lumbar instability has been defined by White and Panjabi (17) as sagittal translation larger than 4.5 mm or larger than 15% of the vertebral body width when comparing with other, or sagittal rotation of larger than 15° at L1-L2, L2-L3 or L3-L4, 20° at L4-L5 or 25° at L5-S1. Radiological lumbar instability can be treated with surgical management. On the other hand, clinical lumbar instability should be distinguished because the obvious abnormal translation and rotation cannot be observed in lumbar flexion-extension radiographs, so specific clinical assessments are required to diagnose for this condition.

3. Clinical lumbar instability

The definition of clinical lumbar instability proposed by White and Panjabi (17) has been clinically used that is the inability of the spine to maintain its normal patterns of displacement under physiologic loads so that there is no initial or additional neurologic deficit, no major deformity, and no incapacitating pain. Clinical signs and symptoms should be used to diagnose rather than radiological findings (4,7) because flexion-extension radiographs focus on end range of motion while abnormally excessive movement of clinical lumbar instability may be relevant with specific clinical symptoms of clinical

lumbar instability in mid-range range of motion (4,7). It is noted that radiographs are frequently failed to detect the spinal problem in patients with clinical lumbar instability (7).

It is not only degeneration of passive stabilizing subsystem which causes clinical lumbar instability but also dysfunction of active stabilizing and improper neural control subsystems may be significant causes of this condition (14). Clinical lumbar instability may have a similar pathomechanism with radiological lumbar instability (3); however, early degree of lumbar instability causing only compromise of mechanical properties of the motion segment may cause clinical lumbar instability (3). Interestingly, the study of Silfies et al (6) reported patients with clinical lumbar instability showed a reduction of local muscle activation, especially internal oblique and lumbar multifidus muscles in mid-range of motion of the trunk flexion. It is suggested that protective function of local muscle system decreases in stabilizing lumbar motion segment during movement under physiologic load. Therefore, stability of the lumbar spine may be compromised. A deficit in one of three spinal stabilizing systems caused by degenerative process, injury, dysfunction and/or surgery can result in clinical lumbar instability (14,16). The neural control subsystem receives these deficits, which compensates by initiating changes in the active stabilizing subsystem. Even though the necessary spinal stability could be reestablished, deleterious consequences of the compensation may occur to the components of the spinal stabilization system such as accelerated degeneration of the lumbar spine, muscle spasm and spinal injury (14).

Nowadays, several authors suggested that the deficit of neuromuscular system leaves the lumbar spine potentially vulnerable to instability (6,18-19). Silfies et al (6) investigated trunk muscle recruitment patterns during a standing reach test between patients with clinical lumbar instability and healthy controls. They reported that patients with clinical lumbar instability demonstrated significantly greater activity levels of the global abdominal muscles including external oblique and rectus abdominis muscles and lower abdominal synergist ratios: internal oblique relative to rectus abdominis, and external oblique relative to rectus abdominis than the healthy controls. It could be interpreted that it is the presence of changes in the pattern of abdominal recruitment patterns in patients with clinical lumbar instability. These patterns have been described as substitution activation of global muscle system (superficial abdominal muscles) to the local muscle system (deep abdominal muscles) (6,20). This appears to be the neural control subsystem attempting to maintain the stability demands of the spine in the presence of local muscle system dysfunction (12). The muscle substitution may provide more spinal compression (12). Marshell and Murphy (18) measured the muscle onset time of transversus abdominis muscle during rapid unilateral shoulder flexion, extension and abduction using a surface electromyography (sEMG) in chronic LBP patients. They reported that 75% of patients showed lacking anticipatory activation because transversus abdominis muscle was delayed in its activation to perturbation from arm movements. Furthermore, delayed transversus abdominis activation was related with ipsilateral arm flexion and extension. Silfies

et al (19) investigated changes in trunk muscle onset time between patients with clinical lumbar instability and healthy controls during self-arm perturbation task. They suggested that the healthy controls demonstrated patterns of anticipatory activations of transversus abdominis, lumbar multifidus, rectus abdominis, external oblique and erector spinae muscles and these muscles activated significantly earlier than other trunk muscles when compared with patients with clinical lumbar instability.

4. Diagnosis of clinical lumbar instability

Although some medical doctors often use flexion-extension radiographs which concentrate on end range of motion for their diagnosis of lumbar instability, clinical lumbar instability may present abnormally excessive motion in mid-range of motion where is emerged the clinical symptoms (7). Despite of arguable findings for diagnosing clinical lumbar instability, numerous authors have suggested that specific clinical signs and symptoms of this illness as well as specific clinical tests could be used in identifying clinical lumbar instability (5,7, 9-10). These included patients reports about their back giving way, catching or locking of the back, pain during transitional activities or sustained postures, chronic back pain, shaking during movement, instability catch sign, Gower's sign, apprehension sign, and hypermobility during springing test. Interestingly, instability catch sign, apprehension sign, painful catch sign, and prone instability test is clinically used for diagnostic criteria to identify clinical lumbar instability because other signs, symptoms and specific clinical tests may be not clear to differentiate clinical lumbar instability from other spinal diseases. Instability catch sign has been widely employed as a clinical determination for diagnosing clinical lumbar instability and is the unique symptom of clinical lumbar instability (2,11,13,20) with high specificity (75%) and sensitivity (89%) (21). Instability catch sign is defined as a sudden painful snap when patients extend their back from the trunk forward bending position into the upright position resulting in reflexive painful muscular spasm (9,11,13,21) (Figure 1). Although, apprehension sign is not reported regarding clinical sensitivity and specificity yet, it often occurs during trunk movement and can be one of the specific indications of clinical lumbar instability (7-9). Apprehension sign is explained that patients feel a sudden sensation of lower back collapse while movement (7-9). Painful catch sign and prone instability test were included to be diagnosing criteria for clinical lumbar instability (5,9-10). Painful catch sign is defined as legs suddenly drop because of a sharp pain in the lower back after perform the double leg raises (5,9) (Figure 2). For prone instability test, it is a provocation test and the patient is in prone lying position with the trunk on the plinth, anterior superior iliac spines (ASISs) at an edge of the plinth and both feet rest on the floor. While the patient rests in this position, the investigator performs posteroanterior (PA) glide at each segment of the lumbar spine. If a painful segment is identified, the patient then lifts the legs from the floor and hand holding to the plinth is used to maintain position, and PA glide is reapplied to that painful segment. The prone instability test is positive when pain is provoked in the resting position and eliminated during active muscle contraction to lift the legs from the floor (8-10) (Figure 3).

Figure 1. Instability catch sign

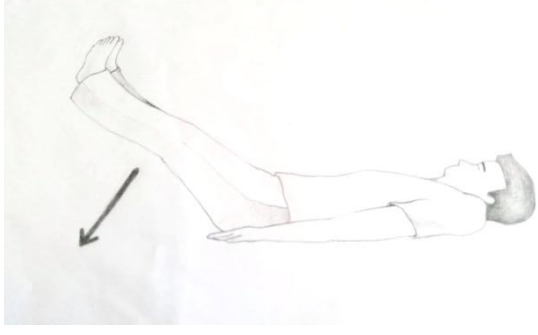

Figure 2. Painful catch test

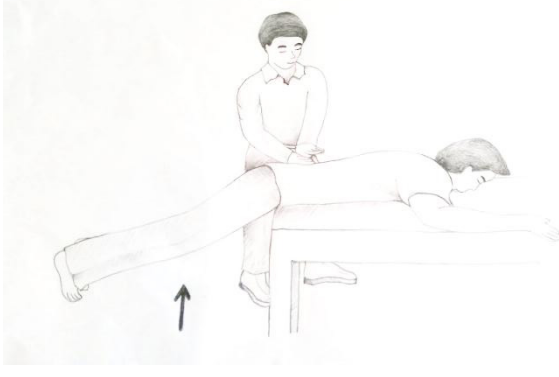

Figure 3. Prone instability test

5. Considering of core stabilization exercise as the treatment of clinical lumbar instability

Some therapists may prescribe non-steroidal anti-inflammatory drugs, short courses of narcotic analgesic, a muscle relaxant, resting during exacerbation period (17), lumbosacral corsets (17), educational program focusing on avoiding end range of movements of the lumbar spine (20), and general exercise of trunk flexors and extensors to patients with clinical lumbar instability (17), even though a standard approach to address the problems of this illness has not been suggested. It has been hypothesized that the treatment which can improve lumbar stability could be beneficial for this illness. Recently, CSE has become popular therapeutic treatment for LBP, and it is based on principle about how spinal stability can be provided in daily works by the trunk muscles (17). Dysfunction of the trunk muscles especially local trunk muscles, such as transversus abdominis (TrA) and lumbar multifidus (LM) muscles could minimize lumbar stability (6,14,19,22). It is speculated that CSE is an approach to improve performance of these muscles that may decrease the problems of clinical lumbar instability.

Richardson et al (12) described a specific exercise intervention which has been well known as CSE to train co-activation of the TrA and LM muscles for enhancing static and dynamic lumbar stability. Reduction of pain and disability are emerged after performing CSE (22-26). Isometric co-activation of TrA and LM muscles while maintaining the neutral lumbar spine should induce to relearning of stabilization role of these muscles (12-13). The CSE aims to improve activations of deep trunk muscles including TrA and LM muscles to enhance spinal stability (12). Hodges et al (27) reported that intra-abdominal pressure (IAP) during functional tasks can be enhanced by the TrA muscle that did not produce a significant flexion moment of the lumbar spine, and suitable amount of IAP can maintain stability of the lumbar spine during trunk movement or loading. Nevertheless, significantly delayed contraction of the TrA muscle could be seen in patients with LBP and it indicates a decrease of lumbar stability and a fundamental problem with motor control (18). Several authors were focusing on the importance of LM muscle because it provides lumbar intersegmental control during static posture and movements and it is so-called neutral lumbar lordosor (2). Besides, it contributes two thirds of all active forces at L4-L5 and provides 66% of the segmental stiffness at this level (2). Therefore, any dysfunction or deficit of LM muscle may compromise the lumbar stability (12).

6. How to train with core stabilization exercise?

To enhance static and dynamic stability of the lumbar spine, the CSE for training the TrA and LM muscles is suggested. This exercise is based on the principles of motor learning theory and skill acquisition that described by Fitts and Posner who stated three stages in motor learning skill (25).

The first stage in the early exercise training period is to train the patients how to perform isolated contraction of TrA and LM muscles cognitively to enhance the skill and precision of their contractions. Consequently, an isometric co-activation of

the TrA and LM muscles without lumbo-pelvic movement in low-load position should be received. Additional pelvic floor muscle contraction with controlled normal respiration can provide effective isometric co-activation of the two muscles and inhibit muscle substitution such as rectus abdominis or erector spinae muscle. The procedure for training the TrA and LM muscles is that the patients to gently draw the lower abdomen in and up towards their spine without lumbo-pelvic movement and emphasize the TrA and LM muscle activations by using pelvic floor contraction with controlled normal respiration to maintain a static neutral spine position (Figure 4). In this stage, patients are required more awareness in order to achieve co-activation of the TrA and LM muscles. Increase of holding time of contraction is the progression in the first stage and it can be provided the effectiveness by using biofeedback or facilitation technique (12). Once the patient achieves the first stage, the second stage of CSE is prescribed. It aims at increasing precision and number of holding time of co-activation of the TrA and LM muscles, and decreasing the use of feedback. This stage is not only emphasizing training of co-activation of the two muscles but also training the integration of local and global trunk muscles. The muscle performances are challenged with heavier loading position such as bridging position and 4-point kneeling position with limb raise. Throughout practice, the concept of sustainable co-activation of the TrA and LM muscles with controlled neutral spine and normal respiration is maintained (Figure 5). Patients will achieve this stage if they are able to control functionally with postural awareness without or minimal pain (12). The last stage of CSE requires a low degree of attention for the correct performance of the tasks. Furthermore, the patients should tell physical therapists for their situations or positions that they feel "unstable" experience or anticipated pain. In this stage, the patients are able to automatically do co-activation of the TrA and LM muscles while performing the unstable position and during functional demands of daily living in various environments and contexts (12). However, number of achievement in each stage is depended on individual's performance.

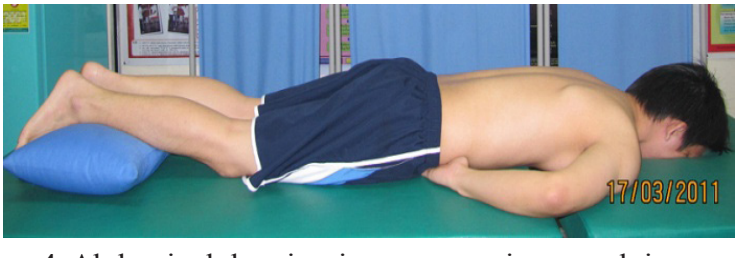

Figure 4. Abdominal drawing-in maneuver in prone lying position

Figure 5. Abdominal drawing-in maneuver in four-point kneeling position with one leg raise

7. Benefits of core stabilization exercise in patients with clinical lumbar instability

To the best author's knowledge, there are several studies to investigate the effectiveness of CSE on pain-related outcomes and electromyographic responses of the trunk muscles (22-26) (Table 1). The high quality study (PEDro score = 7/10) of O'Sullivan et al (25) compared the effects of 10-week CSE versus control group treated with protocol of general practitioner in patients with isthmic lumbar spondylolysis or spondylolisthesis. They reported that significant reductions in pain intensity and functional disability measured were shown in the CSE, and the results were maintained up to 30-month follow-up. However, there was no significant improvement in any outcome measures in control group. Between-group comparisons showed that there were significant reductions in pain intensity (mean difference = 15, 32, 36 and 35-mm of visual analogue scale for 10 weeks, and 3-, 6- and 30-month follow-up, respectively) ($p< 0.001$) and functional disability (mean difference = 13, 10, 15 and 18-mm for 10 weeks, and 3-, 6- and 30-month follow-up, respectively) ($p<0.001$) in favor of CSE throughout follow-up period. O'Sullivan et al (28) conducted further high quality study (PEDro score = 7/10) to assess the abdominal muscle response after receiving 10-week CSE in patients with isthmic lumbar spondylolysis or spondylolisthesis. The findings showed a significant increase in activation ratio of internal oblique relative to rectus abdominis muscles (mean difference = 2.47, $p<0.001$). Therefore, O'Sullivan et al (25,28) concluded that 10-weeks CSE can alter the consciousness and automatic patterns of abdominal muscle activation, and also decrease pain intensity and functional disability in patients with isthmic spondylolysis or spondylolisthesis. Although, the patients with isthmic spondylolysis or spondylolisthesis are claimed that they had spinal instability, it is not clear whether these findings could be generalized to clinical lumbar instability because important clinical signs and symptoms such as instability catch sign (11,20-21), prone instability test (10), painful catch (5,9) or apprehension symptom (7-9) were not used for diagnosis. Moreover, Ganju (29) stated that young isthmic spondylolisthesis may be in stable equilibrium and the patients in studies O'Sullivan et al (25,28) had a mean age of 33 years. Additionally, the control group of study of O'Sullivan et al (25,28) treated

with the treatments prescribed by the individual's general practitioner, so there was no standardized treatment for the control group in that study. Hence, the findings of O'Sullivan et al (25,28) should be clinically considered with caution.

The study (PEDro score = 5/10) of Kumar (24) investigated the acute effects of 15-min CSE in patients with clinical lumbar instability. The researcher reported that there were significant acute improvements in pain, joint stiffness and pain pressure threshold but placebo which was received prone lying position was seen only the improvement in pain. However, the conclusion could not be made about the superior effects of CSE when compared with placebo because the researcher did not compare the effects between the groups. Other study (PEDro score = 4/10) of Javadian et al (23) compared the effects of 8-week CSE combined with general exercise and general exercise only in patients with clinical lumbar instability. The findings showed that both interventions provided significant improvement from baseline at 8-week intervention and 3-month follow-up in pain intensity, functional disability, trunk flexion range of motion, trunk flexors endurance, trunk extensors endurance, right trunk lateral flexors endurance and left lateral trunk flexors endurance. The researchers claimed that pain intensity, functional disability, trunk flexion range of motion and trunk muscle endurance in CSE had greater improvement than another; however, the statistical analysis was not done to measure the magnitude of group differences. It is difficult to draw a conclusion about the superiority of CSE.

According to the pilot study of Areeudomwong et al (22) (PEDro score = 6/10), the researchers examined the effects of core stabilization exercise on pain intensity of the instability catch sign (ICS), functional disability and trunk muscle activity in patients with clinical lumbar instability. This study used the clinical signs and tests for included the patients that are ICS, apprehension sign, painful catch test and prone instability test. The findings showed a significant reduction in pain intensity of ICS and functional disability after 10 weeks of intervention but CSE showed a significant increase in the activation ratio of the TrA muscle relative to the rectus abdominis muscle only. In addition, significantly greater improvements in all outcome measures are in favor of CSE after 10 weeks of intervention (mean difference = 3.09, 4.07, 7.89 of numerical rating scale, Roland-Morris Disability Questionnaire and ratio activation of transversus abdominis relative to rectus abdominis, respectively) ($p<$ 0.001). The recent high quality study (PEDro score = 8/10) of Puntumetakul et al (26) was conducted to investigate the long-term effectiveness of 10-week CSE on pain-related outcomes, health-related quality of life, patient satisfaction to the intervention and trunk muscle activation in patients with clinical lumbar instability at one and three months follow-ups. It is an extended study from the study of Areeudomwong et al (22). The patients were randomly assigned to 10-week CSE or control receiving trunk stretching exercises and hot pack. Their findings showed that CSE provided significantly greater improvements in pain (mean difference = 3.29 and 2.86 of numerical rating scale for one- and three-month follow-ups, respectively; $p<$ 0.001), functional disability (mean difference = 5

and 2.76 of Roland-Morris disability questionnaire for one- and three-month follow-ups, respectively; $p< 0.01$), patient satisfaction (mean difference = 1.86 and 1.67 of Global perceived effect for one- and three-month follow-ups, respectively; $p< 0.001$), and physical component of short form-36 (mean difference = 8.4 and 7.99 for one- and three-month follow-ups, respectively; $p< 0.01$) than those observed in the control group. The CSE could facilitate TrA activation greater than in the control group; however, deterioration of LM muscle activation has been shown in the control group when compared with the CSE group. From the studies of Areeudomwong et al (22), and Puntumetakul et al (26), they used the surface electromyography (sEMG) to detect the trunk muscle activation. Although the two studies believed that sEMG is reliable to detect the LM muscle activation, the EMG signal from LM muscle could be interfered with the cross-talk of other muscles such as erector spinae muscles. Stokes et al (30) suggested that LM activation should be measured using an indwelling EMG rather than a sEMG to obtain the reliable data. Therefore, the results regarding LM activation form the two studies (22,26) should be considered with caution.

Table 1. Summary of included researches

Author	Characteristics of participants	Interventions	Outcome measures	PEDro score
O'Sullivan et al (1997) and (1998)	● 42 patients with isthmic spondylolysis or spondylolisthesis ● Age 16-49 years old ● Duration of low back pain >3 months	● 10 weeks of core stabilization exercise (CSE) versus usual general practitioner care for one a week ● Follow-up: 10 weeks, 3, 6, and 30 months	● Pain (short-form McGill visual analogue scale) ● Functional disability (Oswestry Disability Index, ODI) ● EMG abdominal muscle recruitment patterns	7/10
Kumar (2011)	● 18 patients with clinical lumbar instability, mean age 23 years old and duration of low back pain > 3 months ● Had painful arc during spinal movement, positive prone instability test and had ● score of Delphi criteria for 7/13 and 8/14 in subjective and objective aspects	● A single session of CSE or placebo with cross-over design	● Pain (visual analogue scale, VAS) ● Joint play grading scale (0-6 score) ● Pain pressure threshold	5/10
Javadian et al (2012)	● 30 patients with clinical lumbar instability ● Age 18-45 years old ● Duration of low back pain > 3 months ● Positive one of the trunk aberrant movement patterns (painful arc during flexion and return from flexion, Gower's sign and instability catch) ● Positive prone instability test ● Negative straight leg raising	● 8 weeks of CSE combined general exercise versus general exercise only ● Follow-up: 8 weeks and 3-month follow-ups	● Pain (VAS) ● Lumbar flexion and extension range of motion (modified-modified Schober's test) ● Endurance of trunk flexor and extensor muscles (second) Endurance of trunk lateral flexor muscles (side support test, second) ● Functional disability (Modified ODI)	4/10
Areeudomwong et al (2012)	● 20 patients with clinical lumbar instability ● Age 20-60 years old ● Duration of low back pain > 3 months ● Positive instability catch sign ● Positive one of the prone instability test, painful catch sign and apprehension sign	● 10 weeks of CSE versus hot pack and trunk stretching exercises	● Pain (numerical rating scale, NRS) Functional disability (Roland-Morris Disability Questionnaire, RMDQ) ● Ratio activation of trunk muscles	6/10

Author	Characteristics of participants	Interventions	Outcome measures	PEDro score
Puntumetakul et al (2013)	● 42 patients with clinical lumbar instability ● Age 20-60 years old ● Duration of low back pain > 3 months Positive instability catch sign ● Positive one of the prone instability test, painful catch sign and apprehension sign	● 10 weeks of CSE versus hot pack and trunk stretching exercises ● Follow-up: 1- and 3-month follow-up*s*	● Pain (NRS) ● Functional disability (RMDQ) ● Patient satisfaction (Global perceived effect, GPE) ● Physical and and mental component summary of Short Form-36 (SF-36)	8/10

8. Summary

Nowadays, instability catch sign, apprehension sign, painful catch test and prone instability test are increasing used as clinical diagnostic criteria for patients with clinical lumbar instability. In comparison to treatments of general practitioner as well as the superficial heat therapy and trunk stretching exercises, CSE supposed to provide more effective for eliminating pain intensity and improving functional ability, health-related quality of life, global perceived effect and deep trunk muscle activation for patients with clinical lumbar instability. Most of the studies are acceptable in quality based on PEDro consideration but eventually further studies with outstanding methodology are remained to attest the effectiveness of CSE on pain-related outcomes and trunk muscle activations in patients with clinical lumbar instability. Balance measurement, muscle endurance and indwelling EMG or ultrasound imaging for measuring more precise deep trunk muscle activations to provide insight into the potential effects of this exercise would also be included in the future studies. In addition, the other concerning point is long-term effect of CSE and critical theories regarding the mechanisms of CSE to improve pain-related outcomes would be investigated.

9. References

(1) Puntumetakul R, Yodchaisarn W, Emasithi A, Keawduangdee P, Chatchawan U, Yamauchi J. Prevalence and individual risk factors associated clinical lumbar instability in rice farmers with low back pain. Patient Prefer Adherence. 2015; 9: 1.

(2) Izzo R, Gianluigi G, Giuseppe G, Mario M. Biomechanics of the spine. Part I: spinal stability. Eur J Radiology. 2013;8(1):118-26.

(3) Boyling JD, Jull G. Grieve's modern manual therapy. 4th ed. London: Churchill Livingstone; 2004.

(4) Panjabi MM. The stabilizing system of spine. Part II neutral zone and instability hypothesis. J Spinal Disord. 1992;5(4):390-7.

(5) Wong DA, Transfeldt E. Macnab's backache. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2007.

(6) Silfies SP, Squillante D, Maurer P, Westcott S, Karduna AR. Trunk muscle recruitment patterns in specific chronic low back pain populations. Clin Biomech. 2005;20(5):465-73.

(7) Cook C, Brismel JM, Sizer JSP. Subjective and objective descriptors of clinical lumbar instability: a Delphi study. Man Ther. 2006;11(1):11-21.

(8) Hicks GE, Fritz JM, Delitto A, McGill SM. Preliminary development of a clinical prediction rule for determining which patients with low back pain will response to a stabilization exercise program. Arch Phys Med Rehabil. 2005;86(9):1753-62.

(9) Kotilainen E, Valtonen S. Clinical instability of the lumbar spine after microdiscectomy. Acta Neurochir (Wien). 1993;125(1-4):120-6.

(10) Magee DJ. Orthopaedic physical assessment. 4th ed. Philadelphia: WB Saunders; 2006.

(11) Mascarenhas AA, Thomas I, Sharma G, Cherian JJ. Clinical and radiological instability following standard fenestration discectomy. Indian J Orthop. 2009;43(4):347-51.

(12) Richardson CA, Hodges P, Hides J. Therapeutic exercise for lumbipelvic stabilization: a motor control approach for the treatment and prevention of low back pain. 2nd ed. Edinburgh: Churchill Livingstone; 2004.

(13) Demoulin C, Distrée V, Tomassella M, Crielaard J-M, Vanderthommen M. Lumbar functional instability: a critical appraisal of the literature. Ann Readapt Med Phys. 2007; 50(8):677-84.

(14) Panjabi MM: The stabilizing system of the spine. Part I Function, dysfunction, adaptation, and enhancement. J Spinal Disord. 1992;5(4):383-9.

(15) Dagenais S, Haldeman S. Evidence-based management of low back pain. St. Louis Missouri: Elsevier Mosby; 2012.

(16) Leone A, Guglielmi G, Cassar-Pullicino VN, Bonomo L. Lumbar intervertebral instability: a review. Radiology. 2007;245(1):62-77.

(17) White AA, Panjabi MM. Clinical biomechanics of the spine. 2nd ed. Philadelphia: JB Lippincott; 1990.

(18) Marshell PW, Murphy BA. Delayed abdominal muscle onsets and self-report measures of pain and disability in chronic low back pain. J Electromyogr Kinesiol. 2010;20(5): 833-9.

(19) Silfies SP, Mehta R, Smith SS, Karduna AR. Differences in feedforward trunk muscle activity in subgroups of patients with mechanical low back pain. Arch Phys Med Rehabil. 2009;90(7): 1159-69.

(20) Fritz JM, Erhard RE, Hagen BF. Segmental instability of the lumbar spine. Phys Ther. 1998;78(8): 889-96.

(21) Demoulin C, Cany-Serres S, Distrée V, Grosdent S, Depaifve A, Vanderthommen M, et al. Specificity and sensitivity of identifiers specific to clinical lumbar spine instability. Paper presented at the Conference on the 7th Interdisciplinary World Congress on Low Back and Pelvic Pain; 2010 Nov 9-12; Los Angeles, America.

(22) Areeudomwong P, Puntumetakul R, Jirarattanaphochai K, Wanpen S, Kanpittaya J, Chatchawan U, et al. Core stabilization exercise improves pain intensity, functional disability and trunk muscle activity of patients with clinical lumbar instability: a pilot randomized controlled study. J Phys Ther Sci. 2012;24(10):1007-12.

(23) Javadian Y, Behtash H, Akbari M, Taghipour-Darzi M, Zekavat H. The effects of stabilizing exercises on pain and disability of patients with lumbar segmental instability. J Back Musculoskelet Rehabil. 2012;25(3):149-55.

(24) Kumar SP. Efficacy of segmental stabilization exercise for lumbar segmental instability in patients with mechanical low back pain: A randomized placebo controlled crossover study. North Am J Med Sci. 2011;3(10):456-61.

(25) O'Sullivan PB, Phyty GD, Twomey LT, Allison GT. Evaluation of specific stabilizing exercise in the treatment of chronic low back pain with radiologic diagnosis of spondylolysis or spondylolisthesis. Spine**.** 1997;22(24):2959-67.

(26) Puntumetakul R, Areeudomwong P, Emasithi A, Yamauchi J. Effect of 10-week core stabilization exercise training and detraining on pain-related outcomes in patients with clinical lumbar instability. Patient Prefer Adherence. 2013;7:1189.

(27) Hodges PW, Martin Eriksson AE, Shirley D, Gandevia SC. Intra-abdominal pressure increases stiffness of the lumbar spine. J Biomech. 2005;38(9):1873-80.

(28) O'Sullivan PB, Twomey L, Allison GT. Altered abdominal muscle recruitment in patients with chronic back pain following a specific exercise intervention. J Orthop Sports Phys Ther. 1998;27(2):114-24.

(29) Ganju A. Isthmic spondylolisthesis. Neurosurg Focus. 2002; 13(1): 1-6.

(30) Stokes IAF, Henry SM, Single RM. Surface EMG electrodes do not accurately record from lumbar multifidus muscles. Clin Biomech. 2003;18(1):9-13.