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Original Articles

DISCITIS – PEDIATRIC

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History:

A 6-year old male was presented by his father for treatment of neck pain of insidious onset.

There was no history of trauma.

Examination Findings

On evaluation of this young fellow, he was noted to have a markedly stiff cervical spine with a loss of range in all six directions. He had a low grade fever.

There was a positive dural root stretch test in the cervical spine.

Imaging Selection

Despite the protestations of the patient's father, who insisted that all I needed to do was "crack his neck," I finally obtained permission to have plain film x-rays taken of the cervical spine.

Imaging Report:

Images of the cervical spine demonstrated end plate destruction at C5. There was complete loss of IVD space at C4/5 with an equivocal increase in retrolaryngeal space.

Impression:

Impression was discitis.

Management:

The patient was referred to his family doctor for management.

Clinical Presentation:

The peak incidence of discitis is about age 6.[1,2]

No sex or race predilections are known.

Interestingly enough, the lumbar spine is involved more frequently than the cervical or thoracic spine.[2]

A low grade fever is usually present.

Common complaints on presentation are: neck pain, and for mid or low back involvement: abdominal pain, hip pain, difficulty standing or walking.[2]

Abdominal complaints were more common in the 3- to 9-year old group.[2]

Etiology:

Spinal sepsis must be divided into 3 categories: vertebral osteomyelitis (pyogenic and nonpyogenic), epidural abscess, and discitis.[1,2,4]

Discitis is the more self-limiting of the three.

It is probably an inflammatory process involving the pediatric intervertebral disc and the adjoining vertebral end plates.[2]

The etiology of discitis is not known, but an infectious process is assumed.[2]

Pathophysiology

In the pediatric disc, abundant anastomosis of interosseous arterials that are lacking in the adult disc, are present.[1,2]

In adults, these anastomoses are diminished, but the vascular supply of the vertebral endplates shows a prominence of interosseous end arteries.[1,2]

The vascularity of a child's disc is such that channels to the disc exist, and these provide a pathway for disc infection.

Laboratory Findings:

The ESR is consistently positive.[2]

The white blood cell count is often normal, but may be elevated in CSF.[1,2]

Tuberculin skin tests should be performed in all patients when discitis is suspected.[2]

Biopsy:

Bacteria can be demonstrated in some patients. The most commonly cultured organisms are Staphylococcus aureus.[1,2]

Differential Diagnosis:

The chief differential diagnosis of inflammatory changes of the IVD are: developmental anomalies and vertebral osteomyelitis.[1,2,3,4]

Imaging:

The images lag behind the clinical symptoms by approximately 2 to 3 weeks.[2]

The radiographic changes remain the sine qua non of the diagnosis.[2,3]

The irregularities occur on both sides of the vertebral end plate.[2]

In the child, the disc is involved early with eventual vertebral end plate involvement.[1,2]

In the adult, the focus is initially at the vertebral end plate. The vertebral endplate is injured early. Because the disc is avascular, the discs are involved secondarily.[1]

The Technetium diphosphonate bone scan remains the procedure of choice and is the gold standard for screening of suspicious or early cases of discitis.[3]

Management:

Management begins with early and prompt diagnosis in young patients presenting with neck or back stiffness.[2]

White blood cell count and tuberculin tests are indicated in all patients. ESR and oral temperature are also important.[2,3]

Antibiotics are only recommended in cases with positive bacteriologic results.[2]

Prognosis:

In most cases, the prognosis for discitis is for complete resolution of symptoms in 9 to 22 weeks without recurrence.[2]

Radiologic changes may persist.[1,2,3,4]

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[2]Rothman, Richard H., Simeone, Frederick A. **The Spine**. W.B. Saunders Company, Philadelphia, Third Edition 1992, pages 365-371.

[3]Resnick, Donald, Niwayama, Jen. **Bone and Joint Imaging**. W. B. Saunders Company, Philadelphia 1989, pages 756-762.

[4]Juhl, John H., Crummy, Andrew B. **Essentials of Radiologic Imaging**. J. B. Lippencott Company, Philadelphia 1987, pages 91-95.

Wilkinson's Syndrome (sclerotic pedicle)

By Jerrold R. Wildenauer, DC, FACO
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A 52-year-old female presented to our clinic with the complaint of lower back pain and pain that radiated down the outside of both legs. The patient is a flight attendant and was lifting a large coffee urn when she felt immediate pain in the lower back. Prolonged standing or staying in one position intensifies her pain. Stretching of the lower back does provide some relief as does 800 milligrams of ibuprofen two to three times per day.

Her examination revealed moderate to considerable tenderness to palpation over L4, L5. There was moderate to considerable tenderness and hypertonicity of the lumbar erector spinae, multifidus and gluteal musculature bilaterally. The dorsal lumbar range motion was within normal limits. The lower extremity deep tendon reflexes were normal as was great toe strength. Kemps sign was positive when performed to the right.

The x-rays reveal minimal right pelvic unleveling. There is left lateral flexion of L4 on L5 with a minimal to mild left lateral convexity of the thoracolumbar spine. Noted is a hyperlordosis of the lumbosacral spine with an increased sacral base angle. Five lumbar-type vertebrae are noted with the intercristal line at the lower aspect of the L4 vertebral body.

There is marked hypoplasia of the right sacro articular process and the right L5 inferior articular process (figure 1) (<). The right L5 lamina and pedicle are also hypoplastic (figure 2) (arrow). Moderately advanced apophyseal joint degenerative changes are visualized at the left L5-S1 level (figure 1) (arrow). There is a 5 percent anterolisthesis at the L5 level. Minimal anterolateral vertebral margins spurring is noted at the L1-2, L2-3, and L3-4 levels with mild disc space narrowing at the L1-2 levels. Noted is a minimal retrolisthesis of L4 on L5 with facet imbrication bilaterally at the L4-5 level. There is a Schmorl's node involving the anterior aspect of the superior endplate at the L1 level. Figure 3 shows a sclerotic left facets.

It is the combination of the right hypoplastic articular processes and the hypoplasia of the right lamina and pedicle causing segmental aberrant motion that results in the contralateral sclerotic changes.

Unilateral spondylolysis with contralateral sclerosis of the pedicle is referred to as *Wilkinson's Syndrome.* Compensating stress hypertrophy appears radiographically as a dense, sclerotic and enlarged pedicle and pars region. It seems to occur more frequently in the mid to lower lumbar spine. Pedicle agenesis may also result in stress hypertrophy of the contralateral pedicle and pars region. Wilkinson syndrome is frequently found in highly motivated athletes who are relatively young and continually perform repetitive hyperextension activities. Unilateral spondylolysis involving one pars region may allow a small degree of anterolisthesis. In addition to the AP and lateral lumbosacral views, oblique radiographs and a tip view of the lumbosacral junction can help provide a more complete assessment of the pedicle in question. Figure 4 reveals a larger portion of the APLS film.

You must use caution in your differential diagnosis because there are other causes of the sclerotic pedicle including:

1. Benign tumors including osteoblastoma, osteoid osteoma and a bone island. The malignant tumors would include lymphoma, myeloma and Ewing's sarcoma.
2. Other bone producing conditions such as sarcoidosis, Paget's disease and fibrous dysplasia.
3. Infection
4. Laminectomy (iatrogenic)
5. Congenital causes, on the contralateral side, including asymmetric facets, hypoplastic pedicle or hypoplastic facet.

The patient is responding favorably to distraction manipulation, lumbar traction and interferential current. She is instructed to avoid extension of the lumbar spine and given specific lumbar pelvic exercises and

stretches. It is my opinion of the sclerotic pedicle is only incidental finding and not contributing significantly to her acute lumbar pain.

Yochum TR, Rowe LJ: *Essentials of Skeletal Radiology*, second edition. Williams and Wilkins, Baltimore, 1996 (pp.356-358)

Figure 1:

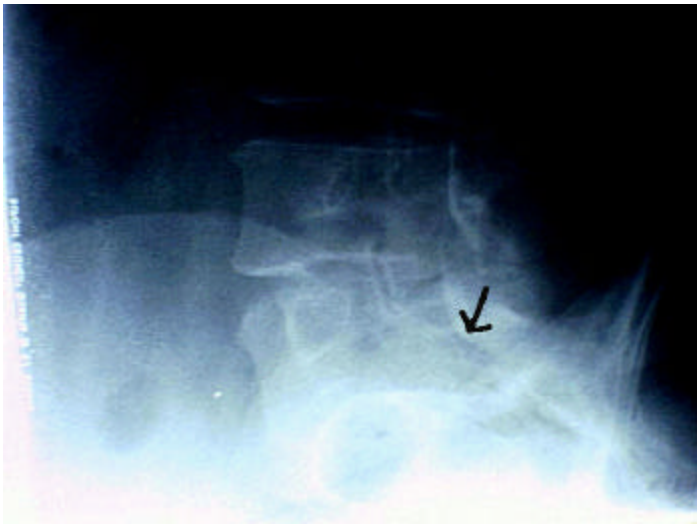

Figure 2:

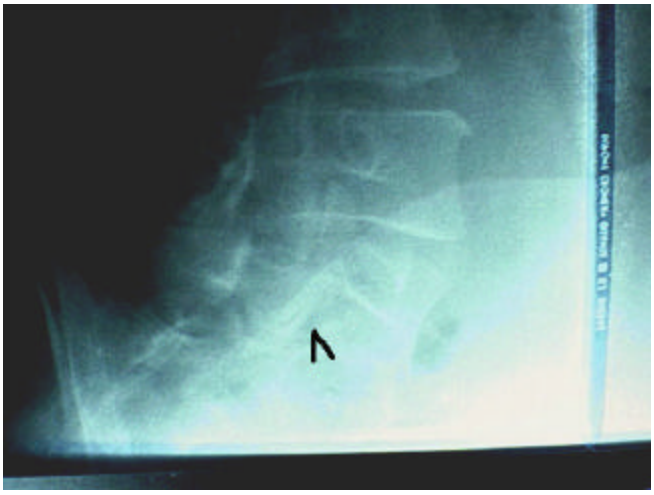

Figure 3:

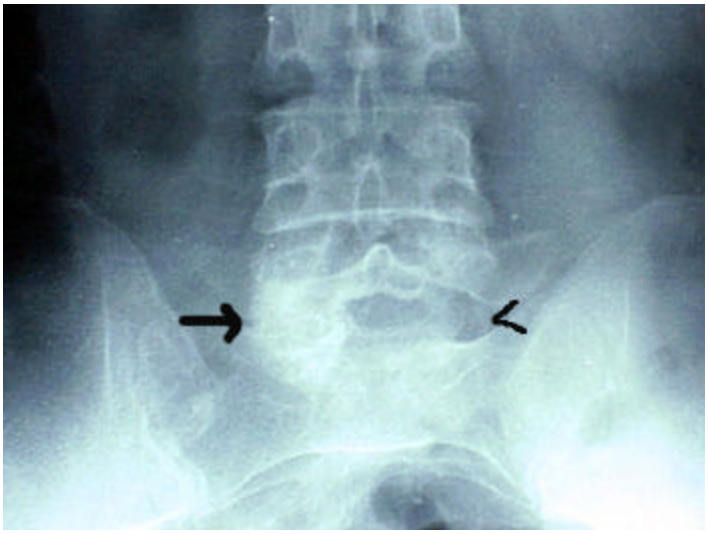

Figure 4:

Figure 4

Reprints

Case History

Clinical Pearl

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Wexler's 5 Point Scale Is One Of The Commonly Used Methods Of Grading The Response During Muscle Stretch Deep Tendon Reflex Testing.

How Well Do You Apply Wexler's 5 Point Scale In Grading Muscle Stretch Deep Tendon Reflexes?

Take This Self Test:

1st Scenario:
You tap on the patellar tendon of one of your patients.
The response is a discernable, low amplitude, non-brisk knee extension.
Question: What do you do next?

a. Record the reflex as 5
b. Record the reflex as 4
c. Record the reflex as 3
d. Record the reflex as 2
e. Record the reflex as 1
f. Record the reflex as 0
g. Perform a Jendrassik's manoeuvre, repeat the test
h. Test for clonus
i. Other

2nd Scenario:
You tap on the patellar tendon of one of your patients.
The response is none: i.e. no discernable knee extension.
Question: What do you do next?

a. Record the reflex as 5
b. Record the reflex as 4
c. Record the reflex as 3
d. Record the reflex as 2
e. Record the reflex as 1
f. Record the reflex as 0
g. Perform a Jendrassik's manoeuvre, repeat the test
h. Test for clonus
i. Other

3rd Scenario:
You tap on the patellar tendon of one of your patients.
The response is none: i.e. no discernable knee extension.
You perform a Jendrassik's manoeuvre, and repeat the test.
The result is a discernable, low amplitude, non-brisk knee extension.
Question: What do you do next?

a. Record the reflex as 5
b. Record the reflex as 4
c. Record the reflex as 3
d. Record the reflex as 2
e. Record the reflex as 1
f. Record the reflex as 0
g. Perform a Jendrassik's manoeuvre, repeat the test
h. Test for clonus
i. Other

4th Scenario:
You tap on the patellar tendon of one of your patients.
The response is none: i.e. no discernable knee extension.
You perform a Jendrassik's manoeuvre, and repeat the test.
The result is none: i.e. no discernable knee extension.
Question: What do you do next?

a. Record the reflex as 5
b. Record the reflex as 4
c. Record the reflex as 3
d. Record the reflex as 2
e. Record the reflex as 1
f. Record the reflex as 0
g. Perform a Jendrassik's manoeuvre, repeat the test
h. Test for clonus
i. Other

5th Scenario:
You tap on the patellar tendon of one of your patients.
The response is a discernable, moderate to high amplitude, brisk knee extension.
Question: What do you do next?

a. Record the reflex as 5
b. Record the reflex as 4
c. Record the reflex as 3
d. Record the reflex as 2
e. Record the reflex as 1
f. Record the reflex as 0
g. Perform a Jendrassik's manoeuvre, repeat the test
h. Test for clonus
i. Other

6th Scenario:
You tap on the patellar tendon of one of your patients.
The response is a discernable, mode rate to high amplitude, brisk knee extension.
You test for clonus, and the result is no clonus.
Question: What do you do next?

a. Record the reflex as 5
b. Record the reflex as 4
c. Record the reflex as 3
d. Record the reflex as 2
e. Record the reflex as 1
f. Record the reflex as 0
g. Perform a Jendrassik's manoeuvre, repeat the test
h. Test for clonus
i. Other

7th Scenario:
You tap on the patellar tendon of one of your patients.
The response is a discernable, moderate to high amplitude, brisk knee extension.
You test for clonus, and the result is: 2 beats of clonus (i.e. transient clonus).
Question: What do you do next?

a. Record the reflex as 5
b. Record the reflex as 4
c. Record the reflex as 3
d. Record the reflex as 2
e. Record the reflex as 1
f. Record the reflex as 0
g. Perform a Jendrassik's manoeuvre, repeat the test
h. Test for clonus
i. Other

8th Scenario:
You tap on the patellar tendon of one of your patients.
The response is a discernable, moderate to high amplitude, brisk knee extension.
You test for clonus, and the result is: 10 beats of clonus (i.e. sustained clonus).
Question: What do you do next?

a. Record the reflex as 5
b. Record the reflex as 4
c. Record the reflex as 3
d. Record the reflex as 2
e. Record the reflex as 1
f. Record the reflex as 0
g. Perform a Jendrassik's manoeuvre, repeat the test
h. Test for clonus
i. Other

Answers:
1. d.
2. g.
3. e.
4. f.
5. h.
6. c.
7. b.
8. a.

And of course, we would always perform the reflexes bilaterally, and compare the results.

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McNamara, John, Neurological Evaluation Notes: *Reflex Testing*, Neuroanatomy/Neurophysiology Laboratory, Jefferson College of Health Sciences, Department of Biomedical Sciences BIO 330L, p.1
http://www.adjustyourself.com/chs/handouts/bio330lreflexnoteshandout.html

0 = no response with Jendrassik maneuver, 1 = hyporeflexia, present but diminished,
2 = normal, 3 = hyperreflexia (with no clonus), 4 = hyperreflexia with transient clonus,
5 = hyperreflexia with sustained clonus

Review of the Literature

GUIDELINES FOR MEDICO-LEGAL REPORTING OF WHIPLASH INJURY

By Dr. R. Mathews (M.Tech.Chiropractic)

These guidelines are taken from the following text:

S. M. Foreman, A.C. Croft 1995 . WHIPLASH INJURIES The cervical acceleration/deceleration syndrome. Williams & Wilkins Baltimore USA. 2nd Edition. 500p. ISBN 0-683-03315-8

Introduction:

The whiplash phenomenon results from severe forces that accelerate and decelerate the head at greater rates than the vehicle. The following phases are described in the existing scientific literature.

Four phases of whiplash:

Phase 1

Impact from the rear accelerates the vehicle and occupant/s, this causes:

- Compression of the torso into the back of the seat, which straightens the spine through an upward compressive force. This compressive force has the potential to injure pelvic and spinal joint structures. At the point of maximum straightening the head and neck start to whip backward.

- Inertia during the sudden acceleration pulls the legs away from the brake pedal, allowing the impact force to increase the overall acceleration.

Phase 2

- The seat back starts to rebound against the compression of the torso, and the torso is accelerated forward while the head is still being whipped backwards. This acceleration of the body forwards and the head backwards imparts a significant force on the neck. The speed at which the head moves has been calculated in test crashes to be on average2.5 times greater than that of the car.

- The forward movement of the torso is then usually restrained by the seatbelt. The diagonal strap holds back one shoulder while the other continues to move forward. This causes rotational forces which also have the potential to injure the neck.

Phase 3

- Once the head has moved backward to the maximum limit, the forward momentum of the torso causes the neck to start whipping forward.

- Slack in the seatbelt is totally taken up and the deceleration of the torso starts.

- The forward movement and deceleration of the body allows the driver to apply brakes, which causes a more rapid deceleration. The deceleration may however be extremely rapid if a front-on impact with another vehicle occurs.

Phase 4

- Even once the car and torso are fully decelerated the head and neck continue to decelerate.

- This deceleration occurs in a forward arc due to the inertia of the head and the stationary attitude of the torso. The related forces cause a severe forward whipping of the head and injury to the neck.

<u>The forces generated during whiplash:</u>

The convention for measuring forces during acceleration / deceleration studies is the unit ”G”. One G is equivalent to the earth's gravity (9.8 m/sec^2). Studies exposing volunteers to a one G acceleration found the head to move in and opposite direction as much as

4.78 G, and on average at 2 to 2.5 G.

- Forces delivered to the human head during a 5 G acceleration have been found to cause cerebral concussion in 50% of test subjects. This 5 G acceleration is achieved by a relatively low impact speed of 10.8 mph. The severe extremes of forward and backward motion have been found to cause most damage to the lower areas of the neck (C4 to C7). The nature of the injuries include fractures, joint sprains, tears of muscles and ligaments, intervertebral disc damage, nerve root injury and vascular tears.

- Injury of the lower back is relatively common and studies have found an incidence of 57% overall. The incidence is far higher for broadside collisions where an incidence of 71% was found. A long-term follow up study of low back pain related to whiplash found that after 10.8 years 34% still complained of low back pain. The forces calculated to act upon the torso during an 8.5 mph collision for a 175 lb man are roughly the equivalent of applying half a ton to the feet, buttocks and back over a period of 50 milliseconds.

Regardless of the area injured, it is clear that significant injuries can result from seemingly minor trauma.

<u>Prognosis for treatment of whiplash injuries:</u>

One of the biggest problems faced by the physician, the patient and the attorney is the length of time that passes before the patient is discharged from treatment. The lack of a widely used system for establishing an accepted

prognosis often results in prolonging the treatment for months in an attempt to relieve symptoms that will probably never resolve. This has allowed some practitioners to abuse the system and has caused the industry to view most whiplash injuries with suspicion.

It is therefore important that physicians are able to recognize symptoms in patient's injuries that will not resolve, failure to do so results in:

- excessive medical charges
- large settlement requests without objective proof of the extreme chronicity of the condition
- insurance carriers deny payment for seemingly unnecessary medical bills and force the claim to litigation

The benefits of using a defendable scale in determining a prognosis include:

- future problems can be predicted, such as the development of neurological deficits or failure to respond to conservative treatment
- the patients progress can be monitored
- insurance companies can establish accurate settlement reserves and decrease the number of litigated cases
- the patient has a better understanding of what future problems may develop from the injury.

The Foreman and Croft classification system:

These authors have developed a numerical rating scale to classify whiplash injuries objectively in an effort to detect those few patients who will pose a realistic chance of developing long-term dependence on medicine, manual therapy, or surgery.

The findings during the initial examination of the patient provide the information for classification into a major injury category (MIC). X-ray examination and a patient questioning provide the data for the clinical modifiers to this system.

Categories:

MIC 1: is used for patients who complain of symptoms directly related to their injuries but on examination there are no objective findings of loss of motion or neurological deficit.

MIC 2: applies to patients who have restricted movement of the neck in addition to MIC 1. These patients do not have neurological deficit.

MIC 3: this group includes MIC 1 and MIC 2 symptoms plus objective neurological deficit. This deficit may include a decrease or loss of sensation, muscle strength or tendon reflex. A loss of this nature can be assessed further by widely accepted medical scales for diminished reflexes and muscle strength. A study using this classification system on 61 whiplash patients found that the scale correlated with a significant presence of residual pain (pain after 2 years). The incidence of residual pain per group were:

- 56 % of MIC 1
- 81% of MIC 2
- 90% of MIC 3.

Typically these symptoms were neck pain, headaches and sensations of numbness or tingling.

The study also found that 41% of the MIC 1 group had a total resolution of their symptoms within two years.

Modifiers to the prognosis:

A variety of problems can influence the patient's recovery. Studies of radiographs in whiplash injury patients have identified the following factors as the most significant modifiers to the prognosis.

- **Canal size of 10 to 12 mm**. A narrower spinal canal has been found to be associated with a higher incidence of neurological damage at the time of injury that may require surgery.

- **Canal size of 13 to 15mm**. Here neurological deficit is not usually produced directly by the whiplash injury but by the effect of joint degeneration at a later stage in a slightly narrower than normal canal.

- **Straight cervical spine**. The loss of the normal neck curve or lordosis suggests muscle spasm and/or ligament damage.

- **Kyphotic cervical curve**. This refers to a complete reversal of the normal curve and is associated with ligamentous tears. These patients have a significantly higher incidence of arthritic changes due to the increased weight bearing loads placed on the intervertebral discs by this alignment.

- **Fixated segments**. These pre-existing fusions (congenital, developmental eg. arthritic or surgical) or segments with limited motion on flexion and extension x-rays are focal stiff areas which cause magnification of injury forces directly above and below the fixated segments.

- **Pre-existing degenerative changes**. Studies have found that in nearly all cases with pre-existing arthritis the recovery prospects are poorer.

- **Loss of consciousness**. By striking the head a second injury is sustained. Studies found that patients who sustained loss of consciousness had a high likely-hood of residual symptoms, with 64% developing degenerative changes.

Point values for the Foreman and Croft Classification System:

MIC groups: according to the symptoms and signs patients are categorized into one of the following with the correlating point value:

MIC 1. 10 points

MIC 2. 50 points

MIC 3. 90 points

Modifiers: the points assigned to modifiers are accumulative except for canal size and spinal curvature modifiers where only one of each will be applicable per patient.

Canal size of 10 to 12 mm. 20 points

Canal size of 13 to 15mm. 15 points

Straight cervical spine. 10 points

Kyphotic cervical curve. 15 points

Fixated segments. 15 points

Pre-existing degeneration. 10 points

Loss of consciousness. 15 points

Interpretation:

Based on the point total the patient is placed into one of five categories.

Prognosis group 1 : 10 -30 points

These patients are MIC 1 who may have one high scoring or two low scoring modifiers. The prognosis for these patients is excellent because most will recover fully. Few will have residual pain, but those that do will complain of relatively minor problems as occasional muscle pain. There is no reasonable expectation of long-term dependence on medication or the need for surgery.

Prognosis group 2 : 35 - 75 points

These are either MIC 1 or MIC 2 patients with associated modifiers. The prognosis here is good and long-term dependence on medication or the need for surgery is unlikely. Most of these patients are expected to have residual pain in the form of intermittent, moderate neck pain which may require treatment.

Prognosis group 3 : 75 - 100 points

This group is mainly made up of MIC 2 patients with several modifiers or MIC 3 patients. Here the prognosis is fair, and a number of these patients develop neurological deficits. Residual symptoms in this group include areas of numbness or muscle weakness. Long-term dependence on medication or the need for surgery is still considered unlikely, but 2^{nd} or 3^{rd} specialist opinions are suggested.

Prognosis group 4 : 100 - 125 points

Here MIC 2 patients with many modifiers and MIC 3 patients with few modifiers are found. The probability of future neurological deficits is likely and the prognosis is consequently poor. There is a fair probability that surgery will be required in the future. Further opinions from surgeons are suggested in these cases.

Prognosis group 5 : 130 - 165 points

This group has an unstable prognosis. Future surgery is likely and they will in all likely-hood become dependent on medication.

Example prognosis:

Mr. X presented with post-traumatic changes after his car was hit from behind a few hours previously. Examination revealed pain on palpation of the anterior neck muscles. Range of motion was restricted on extension and within normal limits for all other directions. Radiographic examination revealed a straightening of the cervical curve. There were no other radiographic findings. The patient's history was unremarkable. No loss of consciousness was reported.

Prognosis rating: Examination revealed soft tissue injury and restricted motion - these place him in MIC 2 with a score of 50.

X-rays showed one modifier, a straight cervical spine - adding 10 points.

With a combined total of 60 points Mr. X is in prognosis group 2. A good prognosis is indicated and there is little chance of long-term dependence medications, neurological deficit or possible surgery.

How long will symptoms persist?

A retrospective study of 266 medico-legal cases found that 45% continued to have symptoms 2 years after settlement of court actions. This strongly dispels the suspicion that the majority of whiplash injury cases resolve with monetary settlement.

Other studies report the presence of headaches in 60% of cases 6 months after injury, 33% within 1 year and 20% after 3 years. One study found 31% to still experience symptoms after 5 years.

These findings suggest that residual symptoms do occur in a significant proportion of the whiplash injury population. This should be considered when determining future care cost estimates, but it must also be remembered that the severity of the symptoms will determine the extent of such costs, if any.

Mild muscle aches in the neck and headaches are usually of a temporary nature and tend to resolve spontaneously for most cases in a short period (hours to days). In these cases conservative therapy with medication and manual therapy only plays a palliative role and does not reduce the likely-hood of future bouts. Cost incurred by these milder complaints are likely but not always necessary. Most of these cases will have a MIC 1 classification. The high incidence of these mild complaints in the general population also makes it difficult to continue relating the symptoms to the whiplash injury year after year.

Moderate residual symptoms of pain and neck stiffness are likely in most MIC 2 and MIC 3 whiplash cases, but treatment over a long period is necessary in order to prevent excessive degenerative changes.

Symptoms of severe pain, stiffness and neurological symptoms will often develop residual pain and further complications which may require high cost interventions such as surgery. This applies mainly to MIC 3 cases and in some rare cases also to MIC 2 cases.

Financial estimates for chiropractic treatment:

Most prognosis group 1 patients should recover in 3 to 6 visits over 2 weeks at an estimated expense of R600.00 (6 x R100.00).

Prognosis group 2 and some group 1 patients will have a similar initial care requirement of 6 treatments over 2 weeks but future treatment may be needed for residual symptoms for 2 years at a conservative estimate of 10 treatments per year. This results in a total minimum cost of R 2600.00 (26 x R 100.00).

Prognosis group 3 patients will generally require more intensive initial treatment and an estimate of 10 to 12 visits is suggested for the initial four weeks after onset of the injury. Thereafter future treatments at the same conservative estimate of 10 per year for the first 2 years can be expected, but for a significant number of these patients treatment will continue beyond 2 years. A minimum estimated cost for 30 treatments in the first 2 years is R 3000.00, but R 6000.00 may well be incurred over a 5 year period.

These estimates do not take into account treatment rate inflationary increase and additional costs for x-rays, medications, self treatment modalities (heat packs, ice packs, home electro-stimulation devices, medication, orthopedic pillows, home traction unit and cervical collars).

Prognosis groups 4 and 5 may have initial chiropractic treatment in the range of 10 to 12 visits (R 1000.00 to R 1200.00). However, the nature of residual symptoms in these patients is usually of a surgical nature and future chiropractic treatment is unlikely to be of benefit.

Current Events

MUA Coding Issues

Question: How would you code manipulation of the spine under anesthesia for the specific areas of the spine (ie, cervical, thoracic, and lumbar)? Source: CPT Assistant, v7n3, March 1997

CPT answers: From a CPT coding perspective, code 22505, Manipulation of the spine requiring anesthesia, any region, should be reported only once, for any and all regions manipulated on that date.

Question: What type of anesthesia must be used to report CPT codes 22505 and 23700?

CPT answers: The code descriptors do not state the type of anesthesia that must be given.

From a CPT coding perspective, codes having the descriptor "requiring anesthesia" means requiring general anesthesia. Therefore, CPT codes 22505, Manipulation of spine requiring anesthesia, any region, and 23700, Manipulation under anesthesia, shoulder joint, including application of fixation apparatus (dislocation excluded) are both intended to report these services performed under general anesthesia. Source: CPT Assistant, v9n1, January 1999

American College of Chiropractic Consultants Grant Advanced Standing to Chiropractic Orthopedists

Did you know the American Board of Chiropractic Consultants (ABCC) grants advanced standing to chiropractic orthopedists? Much of the course work needed to complete the orthopedic program is counted by the ABCC toward a diplomate in consulting (DABCC). Diplomates of the American Board of Chiropractic Consultants are trained to provide expert consulting services in the area of quality assurance, utilization review and forensics. More information is available at the American College of Chiropractic Consultants' website (www.ACCC-chiro.com) or by e-mailing accc@essex1.com.

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