

THE ACADEMY OF CHIROPRACTIC ORTHOPEDISTS



Editorial Board

Bruce Gundersen, D.C., F.A.C.O.
Editor-In-Chief

Dave Leone, D.C., F.A.C.O.
Original Articles Editor

Steve Yeomans, D.C., F.A.C.O.
Reprints Editor

Rick Corbett, D.C., F.A.C.O.
Case History Editor

Loren Miller, D.C., F.A.C.O.
Clinical Pearls Editor

Michael Smithers, D.C.
*Abstracts Editor &
Literature Review Editor*

James R. Brandt, D.C. F.A.C.O.
Current Events Editor

Editorial Review Board

A. Michael Henrie, D.O.
Robert E. Morrow, M.D.
Jeffrey R. Cates, DC, FACO
Ronald C. Evans, DC, FACO
B. Timothy Harcourt, DC, FACO
John F. Hayes III, DC, FACO
Martin Von Iderstine, DC, FACO
Joseph G. Irwin, DC, FACO
Charmaine Korporaal, DC,
Joyce Miller, DC, FACO
Gregory C. Priest, DC, FACO
Jeffrey M. Wilder, DC, FACO
Warren Jahn, DC, FACO
Joni Owen, DC, FACO
James Demetrius, DC, FACO

e-Journal

Quarterly Journal of ACO – September 2007 – Volume 4; Issue 3

Original Articles

Abstracts & Literature Review

Role of anticyclic citrullinated peptide 2 assay in long-standing rheumatoid arthritis.

ORIGINAL ARTICLE

APLAR Journal of Rheumatology. 9(3):211-215, September 2006.
NG, Kristine P. 1; AUSTIN, Paul 2; AMERATUNGA, Rohan 3; McQUEEN, Fiona 4

Abstract:

Background: The anti-cyclic citrullinated peptide (anti-CCP) antibody test is a new serological marker with high specificity for rheumatoid arthritis (RA).

Aims: This study evaluated the frequency of anti-CCP antibodies in comparison to rheumatoid factor (RF) in a group of patients with inflammatory polyarthritis.

Methods: Samples were obtained from 106 patients with established RA and non-RA inflammatory polyarthritis. Anti-CCP (second generation assay - INOVA) and RF antibodies were measured by enzyme-linked

immunosorbent assay. Patient demographics, disease duration and clinical diagnosis were obtained.

Results: Fifty-five patients had established RA and the remaining 51 had non-RA inflammatory polyarthritis. The sensitivity of the anti-CCP2 assay for a diagnosis of RA was 73% compared with 56% for RF. In the RA group, half the RF negative patients (12/24) tested positive for anti-CCP. The specificity of anti-CCP was extremely high at 98% compared with RF at 94%. In the non-RA group, only one patient with lupus tested positive for anti-CCP compared with three who were RF positive (all of whom also had lupus).

Conclusion: The second generation anti-CCP assay was more sensitive and specific than RF for a diagnosis of RA in this population. This test may improve accuracy of diagnosis in patients with long-standing polyarthritis.

Concussion – a review

Compiled by: Warren T. Jahn, DC, MPS, DABCO, DACBSP, DABFP
Leanne N. Cupon, DC, DABFP, DACRB

With fall contact sports under way, this review document provides an overview of clinical facts (bulleted) that are important to chiropractic orthopedists who, at an event, must differentially consider concussion as a diagnosis in blunt force trauma. This document is only a guide, is of a general nature, does not include return to play or treatment recommendations, is consistent with the reasonable, objective practice of a healthcare professional and is not intended as a standard of care.

Definition

Concussion or mild traumatic brain injury (MTBI) is a pathophysiological process affecting the brain induced by direct or indirect biomechanical forces.

Common features

- Rapid onset of usually short-lived neurological impairment, which typically resolves spontaneously
- Acute clinical symptoms that usually reflect a functional disturbance rather than structural injury
- A range of clinical symptoms that may or may not involve loss of consciousness (LOC)
- Neuroimaging studies are typically normal

Epidemiology

- Concussions occur commonly in helmeted and non-helmeted sports, and account for a significant number of time loss injuries
- Published reports indicate concussion injuries occur at a rate of:
 - 0.14–3.66 injuries per 100 player seasons at the high school level, accounting for 3–5% of injuries in all sports
 - 0.5–3.0 injuries per 1,000 athlete exposures at the collegiate level
 - Self-report data suggests significantly higher incidence of concussion.
 - Because of under recognition and/or under reporting, the incidence of concussion and its sequelae is unknown

Causes

- A previous concussion is a significant risk factor for sustaining a concussion
- One study reported that risk of sustaining a concussion was 4-5 times higher in patients who had at least 1 concussion in the past
- Other risk factors for sustaining a concussion that have been suggested but not proven include not wearing mouth guards, poor fitting helmets, and genetic predisposition. Research in all of these areas continues today

Sport Specific Biomechanics

Mechanisms of injury may differ between sports. Possible mechanisms of injury include compressive forces, which may directly injure the brain at the point of contact (coup). Tensile forces produce injury at the point opposite the injury (contrecoup), as the axons and nerves are stretched. Finally, rotational forces may result in a shearing of axons. Therefore, the direct force at the point of contact may not solely be responsible for the

severity of injury if a high rotational component with significant shear effect occurs.

All of the different mechanisms may result in biochemical changes related to perfusion, energy demand and utilization at the site of injury that is not well understood. At this time, it is unclear as to whether any experimental animal model or human studies on more severe brain injured patients accurately reflect the pathophysiology of the typical mild traumatic alteration in brain function.

On-Field/Floor

Evaluate the injured athlete on-the-field/floor in a systematic fashion:

- Assess for adequate airway, breathing, and circulation (ABC's)
- Followed by focused neurological assessment emphasizing mental status, neurological deficit, and cervical spine status
- Determine initial disposition (emergency transport vs sideline evaluation)

Sideline

- Obtain a more detailed history and perform a more detailed physical examination
- Delineate the mechanism of injury
- Assess for cognitive, somatic, and affective signs and symptoms of acute concussion, with particular attention to retrograde amnesia (RGA), posttraumatic amnesia (PTA), and more than brief LOC (minutes, not seconds), because of their prognostic significance
- Perform a more detailed assessment of cognitive function (e.g., memory, calculations, attention span, concentration, speed of information processing)
- Do not leave the player unsupervised
- Perform serial neurological assessments

Post event

- Determine disposition for symptomatic and non-symptomatic players, including post-injury follow-up (options include return-to-play, home with observation, or transport to hospital)
- Provide post-event instructions to the athlete and others (e.g., regarding alcohol, medications, physical exertion and medical follow-up)
- Coordinate the care and follow-up of the athlete
- Discuss status of athlete with parents, caregivers, certified athletic trainers and coaching staff within disclosure regulations

Assessment tools

1. A Glasgow Coma Scale (GCS) is routinely used to assess head injuries in an emergency department. This 15-point scale is used to assess eye (spontaneous opening = 4 to no response = 1), motor (obeys commands = 6 to no response = 1), and verbal responses (oriented = 5 to no response = 1) in an attempt to quantify level of consciousness. This tool is not sensitive enough to evaluate more mild injuries and should not be used on the field as it relates to playability.
2. McCrea has developed a sideline evaluation that may help the chiropractic orthopedist evaluate the more subtle injured brain. A 30-point scale is used to look at orientation, concentration, immediate memory, and delayed recall. Preseason testing must be done if a chiropractic orthopedist is hoping to use this tool as a supplement to their neurological and mental status exam; if the baseline status of an

individual is not known, assessment for change after a head injury is useless. The sideline evaluation uses months of the year in reverse after a study by Young showed lack of reliability of serial 7s in the baseline evaluation even in non-head-injured athletes.

3. Sport Concussion Assessment Tool (SCAT) is another standardized tool. This instrument combines multiple different assessments into a single assessment device. This combined tool was produced as a part of the Summary and Agreement Statement of the Second International Symposium on Concussion in Sport (Prague, 2004).

Classification

Many different classification schemes have been proposed over the last 15 years. No one classification system is necessarily better than another classification system. In addition, no scientific basis for any of the classification systems exists. Cantu's guidelines, Omayya and Generalli's guidelines, the Colorado guidelines, and the 1997 Neurology consensus guidelines all have been proposed to aid in the evaluation of a concussion.

The CDC Tool Kit on Concussion for High School Coaches is available on-line at http://www.cdc.gov/ncipc/tbi/Coaches_Tool_Kit.htm and uses the 1997 Neurology guidelines in supporting a classification scheme.

Recent studies suggest that loss of consciousness may not be a great predictor of short-term or long-term neurological functioning, which makes the guidelines more controversial.

All classification schemes concur with the ultimate recommendation: Do not allow return to play until the patient is completely asymptomatic. The athlete must be free of headache, dizziness, amnesia, blunted affect, and delayed verbal or ocular responses and all cognitive functioning must have returned to normal.

With four years of college scholarship Division I competition and 30+ years of sports healthcare experience, Dr. Jahn prefers to characterize concussions for simplicity as follows:

1. Simple is injury that progressively resolves after 7-10 days without complication.
2. Complex is persistent symptoms that may include symptoms that recur with exertion, specific sequelae such as seizure associated with the injury, prolonged loss of consciousness (>1 min), or prolonged impairment of cognitive function.

Complications of Concussion

- Cumulative concussions may increase risk for subsequent concussions
- Convulsive motor phenomena
- Tonic posturing or convulsive movements within seconds of the concussion
- Post traumatic seizures
- Postconcussion syndrome
- Reported second impact syndrome (occurs within minutes of concussion in athlete still symptomatic from prior brain injury)

Forensic Issues

Most of the complications listed below probably already existed when the athlete sustained the initial head injury; in other words, they are not caused by a MTBI. These conditions may be associated with what was thought of as a MTBI. Therefore, the chiropractic orthopedist should not think of these conditions as a

complication of a mild traumatic head injury but rather other conditions they must consider when evaluating an athlete with a head injury.

- Subdural hematoma is a rare injury in the athlete presenting with a presumed concussion. Classic presentation is acute and persistent LOC associated with the initial injury.
- No association between epidural hematoma and brain injury exists. This condition classically presents with a brief period of unconsciousness, followed by a lucid period and subsequent deterioration over 15-30 minutes. Tearing of the middle meningeal artery secondary to an associated temporal skull fracture is the usual cause of epidural hematoma.
- Subarachnoid bleeding also may occur with a head injury of any sort. Worsening headache and other signs of increasing intracranial pressure will gradually increase after the initial event.
- Second impact syndrome has been described in many review articles. In this condition, fatal brain swelling occurs after minor head trauma in individuals who still have symptoms following minor head trauma from a prior injury. Thus far, all cases of second impact syndrome have been described in relatively young patients (younger than 20 years old). Significant controversy exists over the etiology of this condition, although it is thought to be secondary to loss of autoregulation of cerebral blood flow in an already injured brain. More recently, authors have questioned the validity of this condition due to problems with documentation of the initial event, documentation of persistent symptoms, and documentation of severity of second impact. Despite these problems, chiropractic orthopedists should be aware of this possible complication, especially when evaluating the relatively immature brain of a young athlete. Treatment of second impact syndrome requires immediate recognition and immediate treatment with hyperventilation and osmotics. Surgical treatment for this condition is ineffective. The overall prognosis is usually grim.
- Postconcussive syndrome consists of prolonged symptoms related to the initial head injury. Unfortunately, severity of the concussion does not necessarily predict who will experience prolonged symptoms. Similarly, the number of concussions is not necessarily predictive of future problems. Symptoms usually consist of persistent recurrent headaches, dizziness, memory impairment, loss of libido, ataxia, sensitivity to light and noise, concentration and attention problems, depression, and anxiety.
- Most patients with MTBI recover in 48-72 hours, even with detailed neuropsychological testing, and are headache free within 2-4 weeks of the injury. Obtain a more detailed history of emotional, concentration, and associated symptoms for patients with persistent symptoms lasting more than 1 week. As you may be aware, a recent study of retired professional football players (average age 53.8 +/- 3.4 years) reports significant memory changes in those players with a history of recurrent concussions.

Prevention

- Concussions cannot be completely prevented
- Helmet use decreases the incidence of skull fracture and major head trauma, but does not prevent, and may actually increase, the incidence of concussion. Controversy regarding possible helmet wearing in soccer recently has been proposed. Although helmets have been shown to clearly reduce the risk of head injury in recreational bicycle riding, no clear evidence exists that the type of headgear proposed for youth soccer will prevent acute or chronic head injury among soccer players. Most concussions in soccer are the result of direct contact rather than heading of the ball.
- Improper use of the head and improper fit of helmet or protective equipment may increase the risk of concussion. Studies of football helmet use in high school have demonstrated that only 15% of the helmets fit

properly. Further documentation of the possible increase in risk of head injury associated with poor helmet fit has not been completed.

- There are rules to limit concussion (e.g., spearing, head-to-head contact, leading with the head). These rules have significantly reduced the frequency of severe head injuries in American football.
- During the pre-participation evaluation, obtain a concussion history.
- Discuss the enforcement of rules to limit concussion with coaching staff and officials before practice and competition
- Discuss with players and coaches techniques which may increase the risk of concussion
- Support anchoring soccer goals to the ground because many deaths secondary to head injury in soccer have been the direct result of a goal tipping over on a player
- Support the use of mouth guards to decrease the risk of dental and facial injury; although the protection they provide to concussion risk is unclear. No controlled study has proven their usefulness in concussion prevention
- Educate athletes, parents, and coaches on the importance of reporting symptoms of concussion to limit complications
- Educate athletes, parents, and coaches regarding the escalation of violence in sports

References

1. American College of Sports Medicine. Sideline preparedness for the team physician: a consensus statement. *Med. Sci. Sports Exerc.* 33:846–849, 2001
2. American College of Sports Medicine. The team physician and return-to-play issues: a consensus statement. *Med. Sci. Sports Exerc.* 34:1212–1214, 2002
3. Boden BP, Kirkendall DT, Garrett WE Jr. Concussion incidence in elite college soccer players. *Am J Sports Med.* Mar-Apr 1998;26(2):238-241. [\[Medline\]](#)
4. Cantu RC. *Second Impact Syndrome.* 20. 1992:55-66
5. Capruso DX, Levin HS. Cognitive impairment following closed head injury. *Neurol Clin.* Nov 1992;10(4):879-93. [\[Medline\]](#)
6. Guskiewicz KM, Marshall SW, Bailes J, McCrea M, Cantu RC, Randolph C, et al. Association between recurrent concussion and late-life cognitive impairment in retired professional football players. *Neurosurgery.* Oct 2005;57(4):719-26; discussion 719-26. [\[Medline\]](#)
7. Guskiewicz KM, McCrea M, Marshall SW, Cantu RC, Randolph C, Barr W, et al. Cumulative effects associated with recurrent concussion in collegiate football players: the NCAA Concussion Study. *JAMA.* Nov 19 2003;290(19):2549-55. [\[Medline\]](#)
8. Kelly JP, Nichols JS, Filley CM, et al. Concussion in sports. Guidelines for the prevention of catastrophic outcome. *JAMA.* Nov 27 1991;266(20):2867-9. [\[Medline\]](#)
10. McCrea M, Guskiewicz KM, Marshall SW, Barr W, Randolph C, Cantu RC, et al. Acute effects and recovery time following concussion in collegiate football players: the NCAA Concussion Study. *JAMA.* Nov 19 2003;290(19):2556-63. [\[Medline\]](#)
11. McCrea M, Kelly JP, Kluge J, et al. Standardized assessment of concussion in football players. *Neurology.* Mar 1997;48(3):586-8. [\[Medline\]](#)
12. McCrea M, Kelly JP, Randolph C, et al. Standardized assessment of concussion (SAC): on-site mental status evaluation of the athlete. *J Head Trauma Rehabil.* Apr 1998;13(2):27-35. [\[Medline\]](#)
13. McCrory P, Johnston K, Meeuwisse W, Aubry M, Cantu R, Dvorak J, et al. Summary and agreement statement of the 2nd International Conference on Concussion in Sport, Prague 2004. *Br J Sports Med.* Apr 2005;39 (4):196-204. [\[Medline\]](#)
14. McCrory PR, Berkovic SF. Second impact syndrome. *Neurology.* Mar 1998;50(3):677-83. [\[Medline\]](#)
15. McGuine T, Nass S. *Football Helmet Fitting Errors in Wisconsin High School Players.* American Society for Testing and Materials; 1996:83-88

16. Pieter W, Zemper ED. Head and neck injuries in young taekwondo athletes. *J Sports Med Phys Fitness*. Jun 1999;39(2):147-53. [\[Medline\]](#)
17. Powell JW, Barber-Foss KD. Traumatic brain injury in high school athletes. *JAMA*. Sep 8 1999;282(10):958-63. [\[Medline\]](#)
18. Wojtys EM, Hovda D, Landry G, et al. Current concepts. Concussion in sports. *Am J Sports Med*. Sep-Oct 1999;27(5):676-87. [\[Medline\]](#)
19. Young CC, Jacobs BA, Clavette K, et al. Serial sevens: not the most effective test of mental status in high school athletes. *Clin J Sport Med*. Jul 1997;7(3):196-8. [\[Medline\]](#)

Prevalence of Lumbar Facet Arthrosis and Its Relationship to Age, Sex, and Race: An Anatomic Study of Cadaveric Specimens

Study Design. An anatomic, epidemiologic study of facet arthrosis in cadaveric lumbar spines.

Objective. To define the prevalence of lumbar facet arthrosis in a large population sample and to examine its association with age, sex, and race.

Summary of Background Data. Arthrosis of lumbar facet joints is a common radiographic finding and has been linked to low back pain. However, no population studies have specifically defined the prevalence of facet arthrosis in the lumbar spine in relation to age, sex, and race.

Methods. A total of 647 cadaveric lumbar spines were examined by a single examiner for evidence of lumbar facet arthrosis. Information on race, age, and sex were collected. Arthrosis at each facet was graded from 0 to 4 on a continuum from no arthritis to complete ankylosis.

Results. Facet arthrosis was present in 53% (L1-L2), 66% (L2-L3), 72% (L3-L4), 79% (L4-L5), and 59% (L5-S1). By decade, facet arthrosis was present in 57% of 20- to 29-year-olds, 82% of 30- to 39-year-olds, 93% of 40- to 49-year-olds, 97% in 50- to 59-year-olds, and 100% in those >60 years old. Fisher exact test and t test demonstrated that men had a greater prevalence and degree of facet arthrosis than women at all lumbar levels ($P < 0.001$). The lumbar level with the highest prevalence and degree of arthrosis was the L4-L5 level, as compared with each of the other levels ($P < 0.001$). There was no difference in arthrosis between right versus left facet joints ($P > 0.5$).

Conclusion. Facet arthrosis is a universal finding in the human lumbar spine. Evidence of arthrosis begins early, with more than one half of adults younger than 30 years demonstrating arthritic changes in the facets. The most common arthritic level appears to be L4-L5. Men have a higher prevalence and degree of facet arthrosis than women.

Eubanks JD, et al. *Spine*. September 1, 2007; Vol. 32, Iss. 19, pp. 2058-2062.

Editor Comment (Jahn): Since facet arthrosis is so universal then it would make sense that prevention of weightbearing shifting posteriorly be included within the long term goals of the treatment plan. Additionally, facet arthrosis should be part of the differential diagnosis for patients presenting with LBP

--

Case History

Two Cases submitted by Warren Jahn.

Etiological Factors in Thoracic Outlet Syndrome

ORTHOPEDICS 2007; 30:425

June 2007

To the Editor:

A 22-year-old woman presented with numbness and tingling in her right arm 2 months after falling on her extended left arm with her right arm holding a door handle concomitantly. She felt numbness, tingling and fatigue with her right arm in the resting position. She also reported that prior to the accident (though significantly less than after the accident) she experienced weakness and fatigue from hyperhydrosis in her hands when she used her arms overhead or when she carried heavy objects.

Neck and upper extremity motions were normal on physical examination, and neurological examination showed no pathological findings except for palmar hyperhydrosis in both hands. Adson, hyperabduction, and Roos tests were positive on the left side with decreased radial pulse during the former 2 tests and with weakness and paleness during the latter test. Cervical radiographs revealed bilateral long transverse processes of C7 and decreased lordosis (Figure 1). Magnetic resonance angiography was suggestive of mild compression in the subclavian arteries (Figure 2). The patient was diagnosed with vascular thoracic outlet syndrome and a traumatic brachial plexus traction injury (neuropraxia). She was prescribed a home-based exercise regimen (for strengthening the cervical muscles, especially the shoulder elevators) and daily life modifications with regard to thoracic outlet syndrome. At one-month follow-up all symptoms, including palmar hyperhydrosis, had improved.



Figure 1: Cervical AP (A) and lateral (B) radiographs demonstrating bilateral long transverse processes of the C7 and decreased lordosis, respectively. **Figure 2:** MRA of the patient with arms in abduction. White arrows depict a mild hypointensity, especially on the right side.

Thoracic outlet syndrome is caused by compression of the neurovascular structures (eg, subclavian vessels, brachial plexus, cervical ganglia, and vertebral arteries) in the cervicoaxillary region.¹ The underlying etiology encompasses congenital and acquired factors. The former comprise bony pathologies like long transverse process of the seventh cervical vertebra, cervical rib, and anomalous first rib;²⁻⁴ and soft-tissue problems like congenital bands, ligaments, or scalene muscle problems.⁵ The latter consists of bony problems like fractures of the clavicle and the first rib; or soft-tissue problems like mass lesions, cervical strain, and postural problems.⁶

The patient was demonstrative as it rendered a combination of etiologies—bilateral underlying congenital bony factors and bilateral cervicoaxillary trauma. Both causative factors contributed alike (ie, the presence of underlying bony factors may have increased the risk of a plexus injury as the trauma provoked the thoracic outlet syndrome symptoms that would have otherwise been due only to congenital bony factors). Finally, since thoracic outlet syndrome may masquerade as many other clinical conditions,⁷⁻⁹ it is noteworthy that our patient's pre-existing scenario of thoracic outlet syndrome would have been overlooked had she not suffered from this recent traumatic episode.

Fevziye Ünsal Malas, MD
Levent Özçakar, MD
Bayram Kaymak, MD
Fuat Özkan, MD
Ankara, Turkey

References

1. Özçakar L, Inanici F, Kaymak B, Abali G, Çetin A, Hasçelik Z. Quantification of the weakness and fatigue in thoracic outlet syndrome with isokinetic measurements. *Br J Sports Med.* 2005; 39:178-181.
2. Korkmaz N, Özçakar L. Long cervical costae articulating with the first ribs: a salient case of thoracic outlet syndrome. *Am J Phys Med Rehabil.* 2006; 85:104.
3. Çağlı K, Özçakar L, Beyazit M, Sirmali M. Thoracic outlet syndrome in an adolescent with bilateral bifid ribs. *Clin Anat.* 2006; 19:558-560.
4. Vanderstraeten G, Özçakar L, Verstraete K. Thoracic outlet syndrome portending Klippel-Feil syndrome. *Joint Bone Spine.* 2006; 73:763-764.
5. Konuskan B, Bozkurt MC, Tagil SM, Özçakar L. Cadaveric observation of an aberrant left subclavian artery: a possible cause of thoracic outlet syndrome. *Clin Anat.* 2005; 18:215-216.
6. Özgüçlü E, Özçakar L. Supraclavicular mass disguising as thoracic outlet syndrome. *Rheumatol Int.* 2006; 26:777-778.
7. Malas FU, Özçakar L. Legends of thoracic outlet syndrome. *Rheumatol Int.* 2006; 27:109-110.
8. Özdemir O, Özçakar L. Thoracic outlet syndrome: another cause for unilateral palmar hyperhidrosis. *Clin Rheumatol.* 2006 Aug 29; [Epub ahead of print].
9. Kaymak B, Özçakar L, Oguz AK, Arsava M, Özdöl Ç. A novel finding in thoracic outlet syndrome: tachycardia. *Joint Bone Spine.* 2004; 71:430-432.

EDITOR'S COMMENTS: Every chiropractic orthopedist recognizes, that there is a need to differentiate between cervical radiculopathy, thoracic outlet and brachial plexus injury with this type of presentation. Cervical radiculopathy is usually manifested in the C5 and/or C6 (nerve root and/or upper cervical plexus) dermatome where thoracic outlet usually involves the C8 and/or T1 dermatome (lower cervical plexus). Brachial plexus traction injury can involve both upper and lower aspects of the plexus. Electrodiagnosis may be warranted to confirm the site and location of involvement for the neurological component; MRA for the vascular component as performed evaluating this case.

Low-back Pain and Unrecognized Cobb Syndrome in a Child Resulting in Paraplegia

By Kristofer S. Matullo, MD; Amer Samdani, MD; Randal Betz, MD
ORTHOPEDICS 2007; 30:237

March 2007

Cobb syndrome was originally described in 1915 and consists of cutaneous vascular markings associated with spinal vascular malformation within the same metameric distribution.¹ While a literature review demonstrates <40 reported cases of Cobb syndrome, the repercussions of a missed diagnosis are severe. The cutaneous markings can appear as traditional port wine stains or even as verrucous malformations.² The underlying spinal malformation can range in size, and in our patient occupied the lower thoracic and most of the lumbar spine. Tethering of the spinal cord with resultant neurologic deficits also can occur.³ Once recognized, treatment options include excision or endovascular embolization.⁴

Case Report

An 11-year-old boy presented with severe, 10/10, sharp pain in his stomach that radiated to his back. Approximately 2 weeks prior, he reported low-back pain, which occurred mostly at night and was partially relieved by massage, localized under his birthmark after participating in a competitive soccer game. He initially presented to his pediatrician, who diagnosed him with muscle strain or myalgias secondary to a viral illness and sent him home.

Medical history revealed an 11-year-old boy who was the product of a normal, spontaneous, vaginal delivery at full gestation with no perinatal complications. At birth, he had a large red birthmark on his back overlying the left paralumbar musculature (Figure 1), and smaller similar marks on his left third finger and right arm (Figures 2 and 3). These birthmarks are red during periods of warmth and excitement and turn bluish in a colder environment. Originally, they were diagnosed as port wine stains.

The patient reached all of his developmental milestones, and was active in soccer and basketball until he presented for treatment.

Bowel obstruction and kidney stones were ruled. When the patient stood to urinate, he immediately lost feeling and strength in his legs. Radiographs of the spine and lower extremities and a computed tomography scan of the spine were negative. Neurologic examination demonstrated 0/5 motor strength in his lower extremities with no sensation. He was diagnosed with Guillan Barre syndrome.

Magnetic resonance imaging (MRI) demonstrated an artereovenous malformation at T8, with a large leaking aneurysm. The artereovenous malformation extended down to the sacrum. He underwent an arteriogram and embolization of the aneurysm and artereovenous malformation.

One year after this ischemic spinal cord injury, the patient has demonstrated partial recovery. He has proprioception, vibration, and extreme temperature sensation to his bilateral lower extremities, however he lacks pain and pressure. Motor examination reveals 3/5 hip flexion, adduction, abduction, knee flexion, knee extension, toe flexion, and toe extension bilaterally. He is wheelchair dependent, and wears bilateral molded ankle foot orthoses for ankle support. He has no bowel or bladder control.

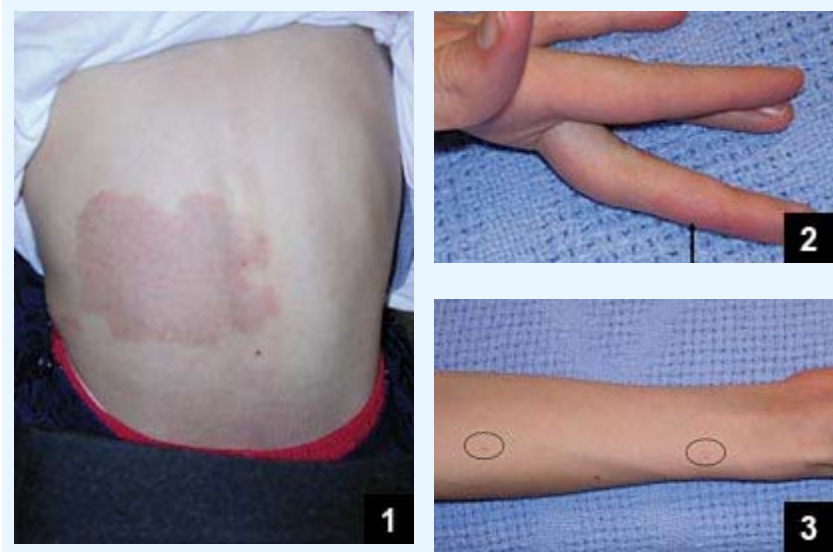


Figure 1: The large arteriovenous malformation on the left thoracolumbar back. **Figure 2:** Left third finger demonstrating a discolored birthmark on the radial aspect. **Figure 3:** Two other arteriovenous malformations on the radial aspect of the right arm.

Discussion

Guillan Barre is normally a progressive process, working from distal to proximal throughout the nervous system and not usually an acute episode of paralysis. It tends to cause symmetric motor loss and lack true sensation loss. Treatment is supportive and tends to resolve.

The patient was given the diagnosis of viral myalgias, back strain, and then Guillan Barre Syndrome. Viral myalgia was entertained, although the patient reported previous illness, fevers, cough, malaise, sore throat, etc.

Other differential diagnoses include herniated nucleus pulposus, although not as common in the thoracic spine, and fibrocartilaginous emboli, which could immediately be ruled out by MRI. Cauda equina syndrome can present with severe back pain with saddle anesthesia accompanied by a disturbance of bowel and bladder function (overflow incontinence and poor rectal tone). The radiation of pain from the back to the stomach must make one think of aneurysm, gastric ulceration, cholecystitis, or appendicitis, knowing that younger patients have difficulty specifically localizing pain. Nephrolithiasis and bowel obstruction were included in this patient's differential diagnosis and were excluded through examination, abdominal radiograph, and urinalysis.

The number of documented cases of Cobb Syndrome is small and the treatment is directed at the arteriovenous malformation and the sequelae of a spinal cord injury if it occurs. Intravascular embolization can be used to decrease the flow through the vascular malformation, or to stop the bleeding of a ruptured malformation. Since blood can compromise neurologic function, by both local irritation and by compressive effects, corticosteroids and a possible decompression may be necessary if neurologic complications occur. Our patient was not decompressed for two reasons: first, the diffuse nature of the hematoma did not have a mass effect on the cord, and second, the paraplegia was thought to result from a stroke. The embolization that occurred was to stop the continued bleeding to prevent a mass effect, and to reduce/eliminate the arteriovenous malformation to prevent further embolic complications.

Birthmarks on infants are common entities, and most can be readily dismissed as benign lesions that may resolve with age. However, large lesions overlying the spinal cord may be a forewarning of underlying spinal

pathology. In a child with Cobb Syndrome, this pathology is a spinal arteriovenous malformation, which is a potentially treatable entity. When a child presents with low back pain accompanied by any neurologic complication (ie, weakness, numbness, radicular pain, bowel/bladder changes), an MRI of the spine should be performed.

References

1. Cobb S. Haemangioma of the spinal cord associated with skin naevi of the same metamere. *Ann Surg.* 1915; 62:641-649.
2. Clinton TS, Cooke LM, Graham BS. Cobb syndrome associated with a verrucous (angiokeratomalike) vascular malformation. *Cutis.* 2003; 71:283-287.
3. Brant AJ, James HE, Tung H. Cutaneomeningospinal angiomatosis (Cobb syndrome) with tethered cord. *Pediatr Neurosurg.* 1999; 30:93-95.
4. Soeda A, Sakai N, Iihara K, Nagata I. Cobb syndrome in an infant: treatment with endovascular embolization and corticosteroid therapy: case report. *Neurosurgery.* 2003; 52:711-715.

EDITOR'S COMMENT: During my orthopedic training and in 30 years of practice, I have never learned about nor have seen Cobb syndrome. The number of documented cases of Cobb syndrome is small but this case emphasizes that any back pain in children should be evaluated thoroughly.

Clinical Pearl

Clinical Imaging Pearls

Posterior Lumbar Interbody Fusion Failure: A Brief Case Presentation

Written by: James Demetrious, DC, FACO

A 48-year old patient was referred to our office by a local Physical Medicine and Rehabilitation physician. The patient presented with unremitting severe lower back and bilateral leg pain, urinary incontinence and sexual dysfunction of six years duration. His past medical history included five prior lumbar surgeries and Posterior Lumbar Interbody Fusion (PLIF).

Upon examination, the patient was afebrile and exhibited normal vital signs. He exhibited significantly reduced and painful global ranges of lumbar motion. Seated and supine straight leg raise produced bilateral leg pain to the calves. Valsalva was positive for localized lower back pain. Neurologic examination revealed 0/5 Achilles muscle stretch reflexes, 4/5 motor strength of bilateral plantar flexion, dorsiflexion, eversion and inversion. Palpation revealed painful trigger points with jump signs affecting the quadratus lumborum and the gluteal musculature. Intervertebral segmental dysfunction was noted affecting L1/2 and the left sacroiliac joint.

Upon our request, the patient underwent CT evaluation with sagittal and coronal reconstruction views (Figures 1-3). The CT images revealed pedicle screw malposition. Pedicle screw malposition represents an early complication that can adversely affect neurologic and vascular structures. Okuda et al. defined pedicle screw malposition as penetration of the medial or lateral pedicle cortex by more than half the diameter of the pedicle screw or penetration of the anterior vertebral cortex by more than 5 mm of the pedicle screw tip, as assessed using postoperative CT studies. (1)

Okuda reported that the most serious complications of lumbar PLIF were postoperative severe neurological complications and adjacent-segment degeneration. (1) Our patient experienced early and late complications, including severe compromise of activities of daily living (ADLs) and total/permanent disability status.

In our office, the patient received flexion-distraction of the adjacent segment superior to the fusion, manual trigger point therapies of paraspinal and gluteal musculature and active rehabilitative core exercises using a Swiss Ball. For the first time since the onset of pain six years prior, the patient reported improvement of pain and ADLs as evidenced by Oswestry pain scales, visual analog and pain drawings.

Despite strong evidence of the first demonstrable clinical improvement since the onset of pain and subsequent surgeries, the patient's worker's compensation carrier decided to curtail chiropractic care to the patient's dismay. Upon last speaking to the patient, he was under pain management care and a sixth spinal surgery was being considered.

Figure 1. Sagittal view revealing penetration of left, lateral cortex of L5 vertebral body.



Figure 2. Axial view revealing penetration of right, lateral cortex of L4 vertebral pedicle and body.

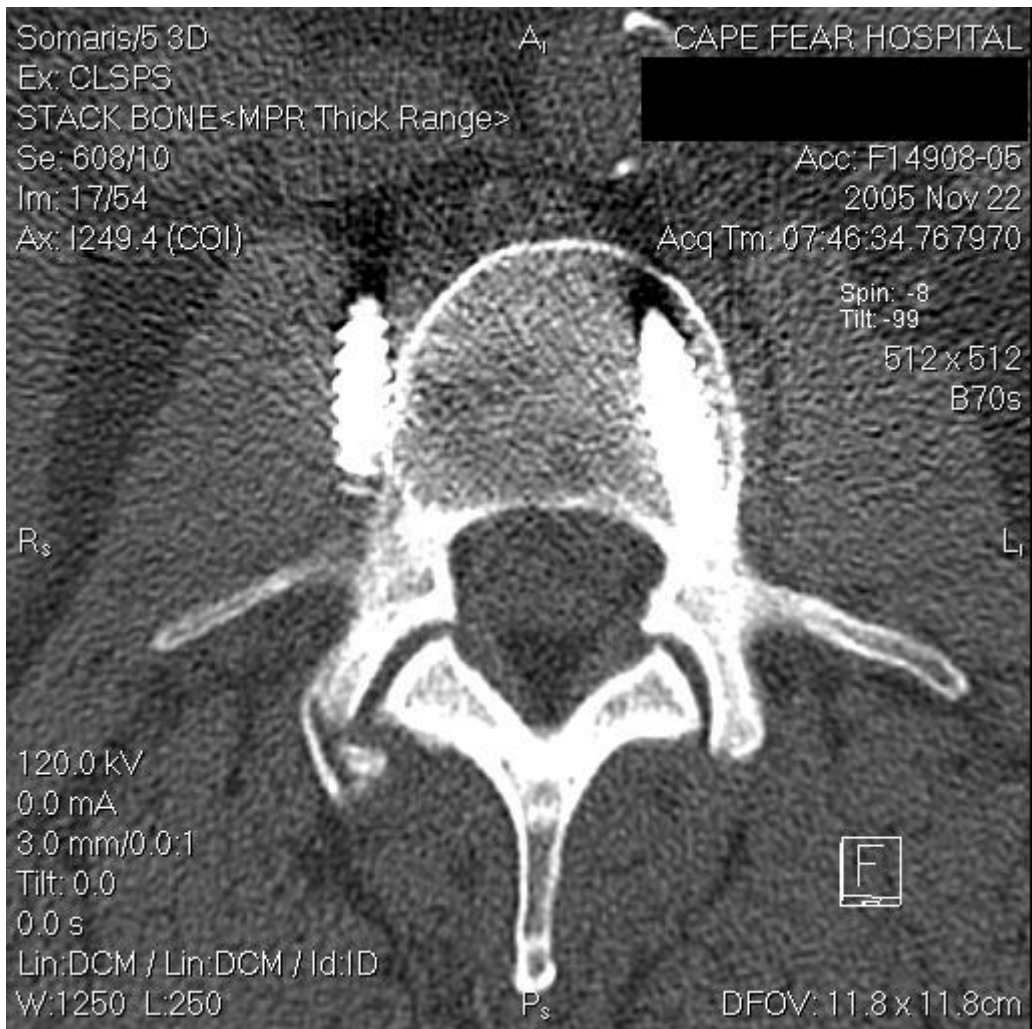
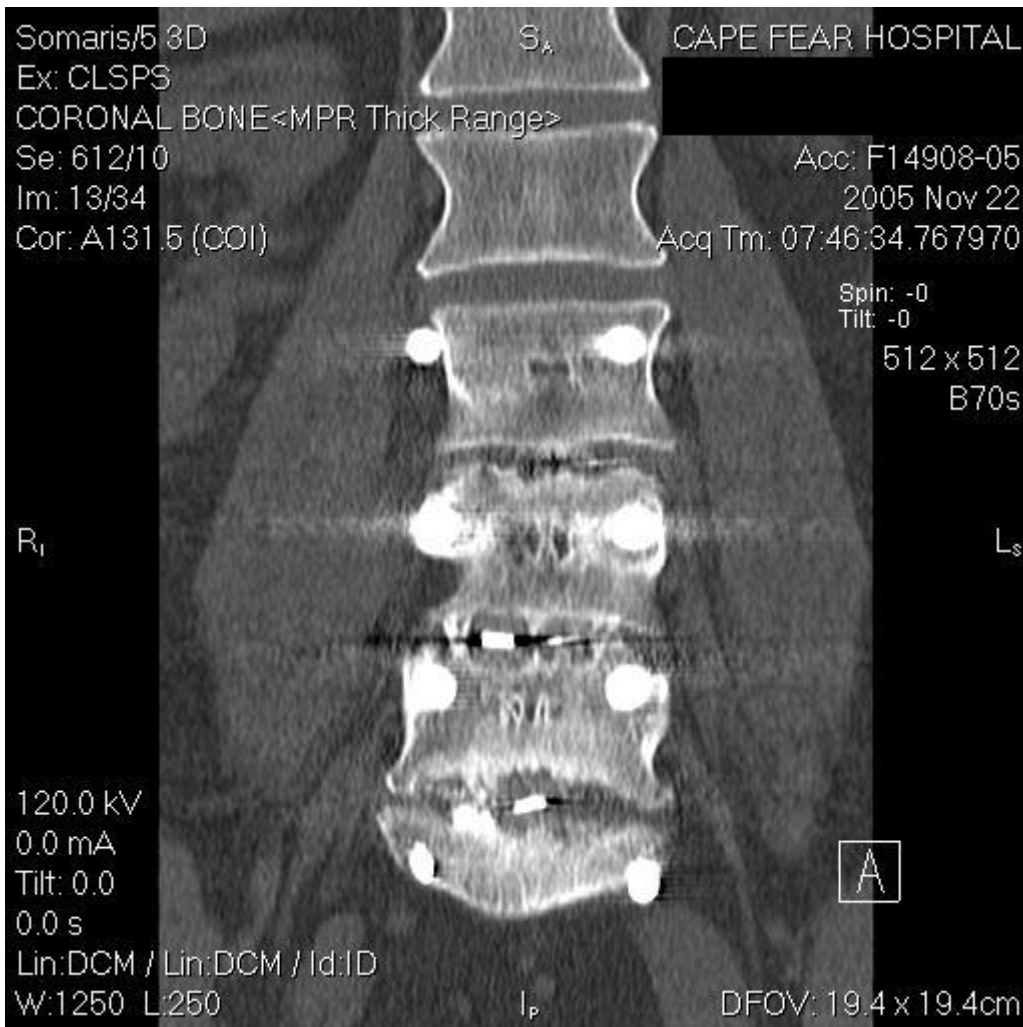


Figure 3. Coronal View reveals pedicle screw malposition external to right lateral aspect of the L3 and L4 vertebral bodies.



References

1. Okuda et al. Surgical complications of posterior lumbar interbody fusion with total facetectomy in 251 patients. *J Neurosurg Spine* 4:304–309, 2006.

Current Events

Spring 2008 Academy Diplomate Examination

The Academy Orthopedic Diplomate Examination will be held May 3, 2008, at Texas College of Chiropractic in Pasadena, Texas.

The candidate who successfully completes the Academy Orthopedic Certifying Examination will receive the designation *Diplomate of the Academy of Chiropractic Orthopedists (DACO)*

Applications are available from the: Academy at www.dorthoacademy.com

If you wish to participate please contact Cheryl at the Academy office at 515-981-9654.

Review class

Review class sponsored by Texas Council of Chiropractic Orthopedists (TCCO) and Texas College of Chiropractic will be held January, 2008. For detail call Texas College of Chiropractic postgraduate department at 800-533-9822

Attribution

Ed Payne, FCER,