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Retrospective Case Analysis of a Patient Experiencing Musculoskeletal Lower Back Pain and Shoulder Pain Who Later Developed Non-musculoskeletal Abdominal Pain

Abstract

Background and Purpose

A young woman with a history of chronic intermittent low back pain is referred to physical therapy by her orthopedic surgeon. During the course of her physical therapy treatment she develops abdominal pain and is referred by the physical therapist to an internal medicine specialist.

Case Description

The patient was a 34 year old woman who reported a 5 year history of intermittent lower back pain. The physical therapy examination revealed findings suggestive of lumbar segmental instability. Her plain films were negative for pathology. She was taking prescription ibuprofen tablets (400mg) twice daily. The patient's systems review was negative for pathology or pregnancy. Approximately 3 weeks into her physical therapy treatment regimen she complained of right upper quadrant abdominal and right trapezius pain. The physical therapist re-examined the patient and determined the abdominal symptoms were most likely visceral in origin, and the patient was referred to internal medicine specialist. Blood tests and an abdominal diagnostic ultrasound, confirmed a diagnosis of drug-induced hepatitis and hepatic cyst as a result of the ibuprofen use.

Outcomes

The patient was immediately taken off the ibuprofen and was cleared to resume physical therapy. This intervention resulted in the abolition of the patient's right upper quadrant abdominal and trapezius pain within 2 weeks of discontinuing the ibuprofen. The patient's lower back pain significantly improved over the course of physical therapy which enabled her to resume a normal activity level.

Discussion

Physical therapists may have an important role in identifying non-musculoskeletal disorders utilizing a differential diagnosis process. Recognizing visceral signs and symptoms is imperative and should be an important part of the physical therapist examination. Appropriate medical referral is necessary to provide proper diagnosis and intervention of visceral disorders.

Background and Purpose

Physical therapy practice is progressing toward an autonomous role in the health care system [1-3]. Physical therapists must possess differential diagnostic skills to enable appropriate clinical decision making. During an initial evaluation or subsequent re-evaluation the physical therapist must recognize the signs and symptoms of both musculoskeletal and nonmusculoskeletal causes of pain and dysfunction. Jette et al. demonstrated that physical therapists with an orthopedic specialization were almost twice as likely to make correct decisions for critical medical and musculoskeletal conditions in a direct-access environment [4]. Davenport et al. stated that physical therapists can act as intermediaries for revising diagnoses by recognizing that certain features of the history and physical examination that are unresponsive to physical therapy intervention, and that referral to an appropriate specialist is necessary for additional testing and treatment [5]. Physical therapists use a diagnostic process to gain enough certainty that initiating physical therapy treatment will not delay a patient's access to more appropriate health services [5].

Visceral structures of the abdominopelvic cavity may refer pain to particular areas of the body that can mimic musculoskeletal pain. The physical therapist must continually screen for medical conditions throughout the treatment process in order to identify potential nonmusculoskeletal conditions that require physician referral. The site of the visceral pain corresponds to dermatomes from which the diseased organ receives its innervations [6]. Visceral disease of the abdomen and pelvis is more likely to refer pain to the back, whereas intrathoracic disease refers pain to the shoulder(s) [6]. Visceral pain rarely occurs without associated signs and symptoms. Careful questioning of the patient will usually elicit a systemic pattern of the symptoms. Visceral pain is not well localized because innervation of the viscera is multisegmental with few

nerve endings. Back and shoulder range of motion is usually full and painless in the presence of visceral pain [6]. Table 1, describes the typical differentiation of systemic and musculoskeletal pain. Figure 1, demonstrates visceral referred pain sites for several organ systems. Table 2, describes signs and symptoms of systemic related back pain.

Drug- induced hepatitis also known as non-viral hepatitis is a condition that can mimic musculoskeletal pain or coexist with a true musculoskeletal pathology that is amenable to physical therapy treatment. This type of hepatitis is a form of liver inflammation that occurs secondary to exposure to certain drugs from which most people recover without serious complications [6]. Drug-induced hepatitis causes liver alterations with liver responses dependent on the chemical nature of the hepatotoxin or the genetic makeup of the person. Several non-steroidal anti-inflammatory (NSAID) agents have been documented to cause hepatic injury in over-the-counter (OTC) and prescription doses [6-9]. Approximately 10% of the total reported drug-induced hepatotoxicity is related to NSAID consumption [7].

Ibuprofen is one of the NSAID medications that has been shown to cause drug-induced hepatitis [7-9]. Studies indicate ibuprofen's potential to create toxicity and cause liver injury [7-12]. Since the introduction of ibuprofen in 1969, this drug has become one of the most prescribed drugs for musculoskeletal pain throughout the world [8]. Doses of this medication range from 400mg/day (analgesic dose) to 2400mg/day (anti-inflammatory dose) the latter is associated with higher GI adverse events [8]. The pharmacokinetic properties of ibuprofen, especially the short plasma half-life of elimination (t1/2 is 2-3 hours) and lack of development of pathologically related metabolites support the view that it has a low toxic potential [7,8].

Clinical signs and symptoms of drug induced hepatitis may include: sense of fullness of the abdomen, right upper quadrant (RUQ) abdominal pain and tenderness, right shoulder pain, fatigue/malaise, nausea/vomiting, jaundice, dark urine, clay-colored stools, abdominal pain not relieved by position change or rest [6]. Figure 2, shows the common sites for referred pain from the liver and gallbladder.

The most common medical tests used to confirm the diagnosis of hepatotoxic disease are blood tests for liver function, diagnostic ultrasound, and CT scan [2,13]. The treatment of this disorder is to cease the NSAID medication, and resolution of symptoms usually occurs within 4 weeks of cessation [7,8]. Although rare, adverse hepatotoxicity from the use of ibuprofen can and does occur. The purpose of this case study is to describe the clinical reasoning process associated with a patient referred to physical therapy for low back and shoulder pain who later developed a drug-induced hepatitis and liver cyst from prescription ibuprofen.

Patient History and Review of Systems

Patient Description

The patient was a 34 year old female who worked as a marketing representative for a local theme park. She complained of a 5 year history of intermittent low back pain (LBP). She reported the initial injury while lifting boxes of home furnishings during a move to a new house. She described her LBP as localized and sharp, with intermittent “catching”. She denied any neurologic or radicular signs or symptoms. The patient was seen by her family physician at that time (5 years ago) who prescribed a pain medication and muscle relaxer. She was seen by a

physical therapist in another city and received passive modalities and stretching for approximately 6-8 weeks. She reports the pain resolved slowly over a 6 to 9 month time period. She went on to report at least 5 episodes of LBP since the initial back injury each lasting 2-12weeks.

The patient described a recent onset of LBP along with intermittent right shoulder pain 2 months prior to initiating physical therapy. She reported initiating an exercise class that involved repetitive bending and overhead barbell lifting as the probable cause of her symptoms. She described the pain as localized and made worse by bending, lifting, and prolonged sitting or standing. The patient was seen by her family physician approximately 4 weeks after the onset of her symptoms. Plain films of her LB and pelvis were performed which were reported as negative by the patient. She was given ibuprofen 800mg/day prescription and instructed to see an orthopedic specialist because of this ongoing history of LBP. Approximately 6 weeks after the onset of her symptoms she was seen by an orthopedic specialist. Another set of plain films were taken of her lumbar spine, pelvis and hips. The new films were interpreted as negative as well, and she was instructed to continue the ibuprofen and referred for physical therapy. She was diagnosed with a lumbar sprain/strain and right shoulder impingement syndrome.

The patient was seen for a physical therapy evaluation approximately 2 months after the most recent exacerbation of her LBP. The patient had stopped her exercise program and continued to use the prescribed ibuprofen, reporting on some days taking twice the normal dosage because of increased LBP. Upon reviewing her health history forms, she was a non-smoker and reported no significant medical history. She reported no gynecological issues, bowel or bladder issues, and was not pregnant. She listed pharmaceutical use including an oral contraceptive and occasionally

a multi-vitamin. Her height was 1.65 m and her weight was 61kg which corresponded to a BMI of 22 kg/m^2 which is in the normal healthy range for females. Based on the information gathered during the interview and systems review the patient's signs and symptoms were determined to be musculoskeletal in nature. Her complaints followed a typical pattern of musculoskeletal impairment that was manageable by physical therapy.

Examination

The patient was experiencing LBP described as 7 out of 10 pain on the numeric pain rating scale (NPRS) and minor right shoulder and upper trapezius pain rated at 3 out of 10 on the NPRS.

Stratford and Spadoni [14] reported the NPRS as a reliable assessment for documenting pain intensity (ICC's .64 to .86). Standing posture was observed in the frontal and sagittal planes. The patient had an accentuated lumbar lordosis and thoracic kyphosis as well as a forward head posture described by McGee [15]. She ambulated independently, but was guarded with transferring sit-to-stand and supine-to-sit.

Spinal exam: Standing trunk active range of motion (AROM) was assessed, as described by Boissonnault [2]. AROM revealed 20% limitation and increased pain with flexion and extension as well as right trunk rotation. Repeated movement testing of her trunk revealed aberrant motion and complaint of "catching" upon returning to an upright stance from the forward flexed position. Hip AROM was recorded as within normal limits (WNL). Sacroiliac assessment consisting of 5 provocative tests described by Laslett [17] was negative. A cervical and thoracic

spine screen involving AROM, quadrant testing and vertebral posterior to anterior spring testing were negative for symptom reproduction or loss of motion. Rib springing was negative and full inhalation and exhalation did not appear difficult or reproduce symptoms. Spinal vertebral posterior to anterior spring testing as described by McGee [15] of the lumbar spinal segments reproduced her LBP at the lower lumbar levels and were assessed as hypermobile at 2 of the 5 lumbar segments. She was tender to palpation over the lumbar paravertebral muscles especially along the right side of her lumbar spine. There was no palpable "step off" which may have indicated a spondolytic lesion [15]. The patient reported increased LBP with extension and side bending quadrant testing of the trunk [15,16]. Lastly, she had a positive lower trunk instability test as described by Hicks [19]. This resulted in a significant reduction in symptoms with manual stabilization of the patient's lumbar spine during prone bilateral leg extension.

Neuromuscular testing included: Manual muscle testing (MMT), reflex testing, and sensory testing as described by McGee [15] and Hoppenfield [16]. Deep tendon reflexes for the upper and lower extremities were WNL. Sensory assessment of light touch along all extremity dermatomes was unremarkable. The MMT of the extremities revealed strength of 5/5 for all major muscle groups except right shoulder external rotation with her arm at her side, and left hip flexion, left hip abduction and left hip internal rotation that were each assessed as 4/5. The patient reported minor increase in right shoulder pain and LB pain with muscle testing across these joints. The supine straight leg raise was negative and measured as 95 degrees on the left and 100 degrees on the right.

Shoulder exam: Shoulder AROM was tested in a seated position as described by McGee [15], and was WNL without complaints of increased symptoms. Right shoulder impingement testing

using the Neer and Hawkins tests described by McGee [15] increased her right shoulder pain along with tenderness over the rotator cuff tendon attachment on the head of the humerus. The shoulder impingement tests have a sensitivity ranging from 75 to 92.1 and specificity ranging from 25 to 47.5 as reported by Cook [18].

Following the physical examination the therapist was convinced the patient's condition was of a musculoskeletal origin and a plan for physical therapy intervention was initiated. The patient's low back signs and symptoms correlated with a clinical prediction rule (CPR) for lumbar instability treatable with stabilization exercise [20]. This CPR developed by Hicks et al. [20] included 4 variables; age < 40 years, average straight leg raise > 91 degrees, aberrant movement present, and a positive prone instability test [20]. The presence of at least 3 of these variables resulted in a specificity of 0.86 (95% CI; 0.71-0.94) and a positive likelihood ratio of 4.0 (95% CI; 1.6-10.0). In light of this patient meeting all 4 criteria for the CPR, a course of lumbar stabilization exercise was planned and implemented for this patient.

Intervention

The original plan of care was to treat the patient for 2 times a week for 4 weeks to address the musculoskeletal impairments. Spinal stabilization exercises have been shown to be effective in the treatment of patients with acute and chronic LBP [21,22]. This type of exercise as well as more functional global pushing, pulling and squat/lunge type exercises described by Porterfield and DeRosa [23] were implemented immediately and progressed over the next 2-3 weeks of treatment. The patient also initiated shoulder girdle exercises for rotator cuff and scapular strength and

stabilization similar to those described by Tate et al. [24]. Biomechanical counseling, postural awareness and body mechanics training were also implemented.

Approximately 3 weeks after initiating treatment the patient reported a significant change in her symptoms. She was now experiencing new pain in the area of her right upper trapiezius and right upper quadrant abdominal area under her right rib cage. The treating physical therapist decided a re-evaluation was warranted in light of the new symptoms.

Re-evaluation

The interview was based on differential diagnostic procedures described by Boissonnault [2]. The patient was interviewed regarding the sudden onset of her new symptoms and she described a constant, dull ache pain in her right upper abdominal quadrant and right upper trapezius, as well as a feeling of fullness under her right rib cage. The pain was unaffected by postural or positional changes and woke her at night when she would lie in a prone position. A review of systems revealed a “slight” increase in abdominal pain after eating “fast food”. She reported no increase in her symptoms when eating other types of food or drink and denied any nausea or vomiting. She reported no other bowel or bladder symptoms and stated she was not pregnant or experiencing any gynecological pain or dysfunction. The patient reported no signs or symptoms of an infection (fever, chills, malaise). She was not experiencing any neurologic signs with loss of sensory or motor function. The patient denied any recent history of trauma or event that may have a direct cause and effect relationship to this new pain. Lastly, she was questioned about her medication use and she reported, prescription oral contraceptive, an OTC multi-vitamin, and prescription ibuprofen. By the time of the re-evaluation she had been taking the ibuprofen for

approximately 7 weeks. Based on the information from this interview, the signs and symptoms did not correlate with a musculoskeletal impairment. The differential diagnostic process concluded a probable non-musculoskeletal origin of the patient's pain. Other medical conditions (Figure 3) would need to be ruled out as potential causes of the patient's new symptoms.

The physical examination included a re-assessment of the patient's neck, trunk and rib cage mobility and bilateral upper and lower quarter screen which were performed on the initial evaluation [2,15]. The patient's trunk mobility had improved and had only a minor complaint of "catching" in the lumbar spine upon extending from the flexed position. She had full mobility of her neck, thoracic spine, and both shoulders in all planes. Cervical and thoracic vertebral segmental posterior to anterior spring testing was negative for any symptom reproduction. Vertebral spring testing over the lumbar spine still reproduced her localized LBP, but reported as diminished compared to her initial examination. She continued to have tenderness over the right rotator cuff tendon attachment site on the humeral head. Manual muscle testing for external rotation of the right shoulder elicited some discomfort at the rotator cuff attachment site on the humerus. Hawkins impingement testing of the right shoulder caused minor discomfort at the rotator cuff attachment site only. The lower quarter screen revealed full pain-free range of motion of bilateral hips, knees, and ankles. Her neurologic scan demonstrated normal reflexes for upper and lower extremities and no sensory changes along any extremity dermatome or over the abdomen. She had no bruising or skin changes over her abdominal area or rib cage. Palpation over the inguinal lymph nodes was unremarkable. The patient's abdominal symptoms were reproduced with palpation over the superior border of the right upper abdominal quadrant. Biossanault [2] describes placing the therapist's left hand under the patients back, around the T10-

T11 region on the right side of the patient, then lift gently. The fingers of the therapist's right hand apply a moderate pressure slightly cephalically and posteriorly. The patient is asked to hold a deep breath; the therapist may note the liver edge as it moves down into the therapists fingers [2]. This maneuver reproduced the patient's pain in the right upper quadrant and right upper trapezius area. The data collected during the physical exam suggested the patients abdominal and right upper trapezius pain was likely originating from the abdominal viscera, in particular, the liver. The patient was instructed to contact her orthopedic physician and seek a referral to an internal medicine specialist.

The patient was seen by an internal medicine specialist approximately 1 week after the physical therapist re-evaluation. She had blood tests ordered that were suggestive of a liver disorder. A diagnostic ultrasound was performed in the physician's office which revealed an "inflamed liver" and a liver cyst. The physician diagnosed the patient with a drug induced hepatitis resulting from the use of the prescription ibuprofen. She was instructed to immediately discontinue the ibuprofen and was cleared to resume physical therapy.

Outcome

The patient returned to physical therapy 3 days after her visit to the internist and resumed her course of physical therapy. The abdominal and right upper trapezius pain were monitored and within 12-14 days after discontinuing the ibuprofen, she had complete abolition of her abdominal and right upper trapezius pain. Approximately 8 weeks after initiating physical therapy treatment, the patient reported an 86% reduction in her LBP which was reported as an intermittent 1/10 on the NPRS. Her shoulder impingement pain had completely resolved, and

she reported no recurrence of her abdominal and upper trapezius pain. She was able to sleep without interruption and returned to full unrestricted activity and was discharged from physical therapy.

Discussion

The patient in this case study presented with signs and symptoms of a musculoskeletal and non-musculoskeletal condition coexisting during the course of her physical therapy treatment. This case analysis emphasizes the importance of using a differential diagnostic approach to hypothesize the potential systemic dysfunction, and the referral to an appropriate medical specialist. Visceral disorders frequently refer pain to the spine and extremities that may be difficult to differentiate from musculoskeletal dysfunction [2]. A thorough physical therapy interview and examination are necessary to identify a potential disease process not appropriate for physical therapy intervention.

The risk of serious gastrointestinal, renal, and hepatic toxicity due to OTC and prescription ibuprofen is well documented [7,8,25-27]. Nonsteroidal anti-inflammatory drugs are among the most common drugs associated with drug induced liver injury, with an estimated incidence of 3 to 23 per 100,000 patient years [12]. The incidences of death, liver failure, vanishing bile duct syndrome, hepatocellular, and cholestatic liver damage have been reported as a result of ibuprofen toxicity [7]. Data also suggests ibuprofen produces liver reactions with concomitant use of certain antibiotics, anti-hypertensive, and statin drugs [8]. The physical therapist needs to be aware of the potential for these types of reactions because of the widespread use of ibuprofen.

Searching the literature for possible interactions between ibuprofen and oral contraceptives revealed no evidence of adverse interaction between these pharmaceuticals and several articles describe a synergistic effect in controlling dysmenorrhea [28,29].

In this case study recognizing the signs and symptoms of a potential coexisting visceral pathology led to a medical referral for further testing and treatment. Vigilant re-examination throughout the physical therapy treatment is important to monitor for possible systemic conditions. The patient in this case study did not complain of several common signs and symptoms associated with hepatobiliary or gallbladder disease. She denied having a history of nausea, vomiting, fatigue, ascites, or jaundice [6]. Given the cluster of other signs and symptoms such as, pain matching the common pattern of referral for liver and gallbladder pathology, constant ache pain unaffected by postural change or movement, a reported feeling of fullness under the right rib cage, night pain, a "slight" increase in symptoms associated with ingestion of "fatty food", and reproduction of abdominal and right upper trapezius pain with palpation of the liver, the treating therapist suspected a visceral origin of pain co-existing with her LBP and right shoulder dysfunction. The physician's examination included blood tests and a diagnostic ultrasound, both of which are standard procedures for diagnosing liver pathology. A definitive diagnosis of drug induced liver injury is based largely on exclusion and the gold standard is expert opinion [30]. The abolition of abdominal symptoms following discontinuation of ibuprofen left no doubt as to the cause of the patient's abdominal pain.

The strength and stabilization intervention for both the trunk and shoulder girdle musculature demonstrated excellent results and are in line with those described by Hicks [20], Tate [24] and McClure [31]. Hicks et al. reported a positive likelihood ratio of 4.0 for successful outcome with

utilizing a stabilization exercise program with LBP patients [20]. McClure and Tate described a positive impact on physical impairments and functional limitations with an exercise program combined with education on patients diagnosed with shoulder impingement syndrome [24,31]. The results obtained in this case study correlated well with the outcomes of these particular studies.

This case study emphasizes that the physical therapist may be the first practitioner to recognize visceral pathology. A thorough examination and screening for musculoskeletal and visceral impairments, along with a process of differential diagnosis will hasten proper intervention, for physical therapy patients.

REFERENCES

1. APTA. Guide to Physical Therapy Practice. Second Edition. Phys Ther. 2001; 81: 9-746.
2. Boissonnault W. Primary Care for the Physical Therapist: Examination and triage. St. Louis, MO: WB Saunders Co; 2005.
3. Donato EB, DuVall RE, Godges JJ, Zimmerman GJ, Greathouse DG. Practice analysis: defining the clinical practice of primary contact physical therpy. J Orthop Sports Phys Ther. 2004; 34: 284-304.
4. Jette DU, Ardleigh K, Chandler K, McShea L. Decision-making ability of physical therapists: physical therapy intervention or medical referral. Phys Ther. 2006; 86: 1619-1629.
5. Davenport TE, Kulig K, Resnik C. Diagnosing pathology to decide the appropriatness of physical therapy: Whats our role? J Orthop Sports Phys Ther. 2006; 36(1):1-2.
6. Goodman C, Snyder T. Differential Diagnosis in Physical Therapy. 3rd ed. Philadelphia, PA: WB Saunders Co; 2000.
7. Bessone F. non-steroidal anti-inflammatory drugs: what is the actual risk of liver damage? World J Gastroenterol. 2010; 16 (45): 5651-5661.
8. Rainsford KD. Ibuprofen: pharmacology, efficacy and safety. Inflammopharmacology. 2009; 17: 275-342.
9. Mayoral W, Lewis JH, Zimmerman H. Drug-induced liver disease. Curr Opin Gastroenterol. 1999; 15 (3): 208-16.

10. Rostom A, Goldkind L, Laine L. Non-steroidal anti-inflammatory drugs and hepatic toxicity: a systematic review of randomized controlled trials in arthritis patients. Clin Gastroenterol Hepatol. 2005; 3 (5): 489-98.
11. O'Connor N, Dargan PI, Jones AL. Hepatocellular damage from non-steroidal anti-inflammatory drugs. QJM. 2003; 96 (11): 787-91.
12. Aithal GP, Day CP. Non-steroidal anti-inflammatory drug-induced hepatotoxicity. Clin Liver Dis. 2007; 3: 763-75.
13. McKinnis L. Fundamentals of Musculoskeletal Imaging, 3rd Ed. FA Davis, Philadelphia, PA. 2010.
14. Stratford PW, Spadoni GF. The reliability, consistency, and clinical application of a numeric pain rating scale. Physiotherapy Canada. 2001; 51 (2): 88-91.
15. Magee DJ. Orthopedic Physical Assessment, 4th Ed. Saunders, Philadelphia, PA. 2002.
16. Hoppenfeld S. Physical Examination of the Spine and Extremities. Appleton-Century-Crofts, Nowalk, Connecticut. 1976.
17. Laslett M, Aprill CN, McDonald B, Young SB. Diagnosis of sacroiliac pain: validity of individual provocation tests and composites of tests. Man Ther. 2005; 10 (3): 207-218.
18. Cook CE. Orthopedic Physical Examination Tests: An Evidence-Based Approach. Pearson Education Inc. Upper Saddle River, NJ. 2008.
19. Hicks GE, Fritz JM, Delitto A, Mishock J. Interrater reliability of clinical examination measures for identification of a lumbar segmental instability. Arch Phys Med Rehabil. 2003; 84: 1858-64.

20. Hicks GE, Fritz JM, Delitto A, McGill SM. Preliminary development of a clinical prediction rule for determining which patients with low back pain will respond to a stabilization exercise program. Arch Phys Med Rehabil. 2005; 86: 1753-1762.
21. 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; 2959-2967.
22. Hides JA, Richardson CA, jull GA. Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. Spine. 1996; 21: 2763-2769.
23. Porterfield JA, DeRosa C. Mechanical Low Back Pain: Perspectives in Functional Anatomy. W.B. Saunders, Philadelphia, PA. 1998.
24. Tate AR, McClure PW, Young IA, Salvatori R, Michener LA. Comprehensive impairment-based exercise and manual therapy intervention for patients with subacromial impingement syndrome: a case series. J Orthop Sports PhysTher. 2010; 40 (8): 474-493.
25. Moore N. Forty years of ibuprofen use. Int J Clin Pract Suppl. 2003; 135: 28-31.
26. Lewis JD, Kimmel SE, Localio AR, Metz DC, Farrar JT, Nessel L, Brensinger C, McGibney K, Strom BL. Risk of serious upper gastrointestinal toxicity with OTC nonaspirin non-steroidal anti-inflammatory drugs. Gastroenterology. 2005; 129 (6): 1865-74.
27. Fendrick AM, Greenberg BP. A review of the benefits and risk of non-steroidal anti-inflammatory drugs in the management of mild-to-moderate osteoarthritis. Osteopath Med Prim Care. 2009; 3: 1-9.

28. Chan WY, Dawood MY, Fuchs F. Prostaglandins in primary dysmenorrhea. Comparison of prophylactic and nonprophylactic treatment with ibuprofen and use of oral contraceptives. Am J Med. 1981; 70 (3): 535-41.

29. Knights KM, McLean CF, Tonkin AL, Miners JO. Lack of effect of gender and oral contraceptive steroids on the pharmacokinetics of ibuprofen in humans. Br J Clin Pharmacol. 1995; 40 (2): 153-6.

30. Hayashi PH. Causality assessment in drug-induced liver injury. Semin Liver Dis. 2009; 29: 348-356.

31. McClure PW, Bralker J, Neff N, Williams G, Karduna A. Shoulder function and 3-dimensional kinematics in people with shoulder impingement syndrome before and after a 6-week exercise program. Phys Ther. 2004; 84:832-8.

Table 1. Systemic vs. Musculoskeletal Pain [6].

Differentiation of Systemic and Musculoskeletal Pain

Systemic
Disturbs sleep
Deep aching or throbbing
Reduced with pressure
Constant or waves of pain and spasm
Is not aggravated by mechanical stress
Associated with:
Jaundice, migratory arthralgias, skin rash, fatigue, weight loss,
low-grade fever, generalized weakness, cyclic and progressive symptoms,
history of infection
Musculoskeletal
Generally lessens at night
Sharp or superficial ache
Usually decreases with cessation of activity
Usually continuous or intermittent
Is aggravated by mechanical stress
Usually associated with nothing specific

Table 2. Signs and symptoms of systemic back pain [6].

CLUES SUGGESTING SYSTEMIC BACK PAIN
■ Age over 45 years
■ Nocturnal back pain
■ Back pain that causes writhing, prompts the client to move about, or curl up in the sitting position
■ Back pain with constitutional symptoms: nausea, fatigue, vomiting, diarrhea, fever
■ Back pain that is insidious in onset and progression
■ Previous history of cancer
■ Back and abdominal pain at the same level (may occur simultaneously or alternately)
■ Sacral pain in the absence of history of trauma or overuse
■ Elevated body temperature, night sweats, febrile chills
■ Back pain that is unrelieved by recumbency
■ Back pain that does not vary with exertion or activity
■ Severe, persistent back pain with full and painless movement of the spine
■ Severe back and lower extremity weakness without pain
■ Back pain associated with meals (increase or decrease in symptoms)

Figure 1. Chart demonstrating typical visceral referred pain sites [2].

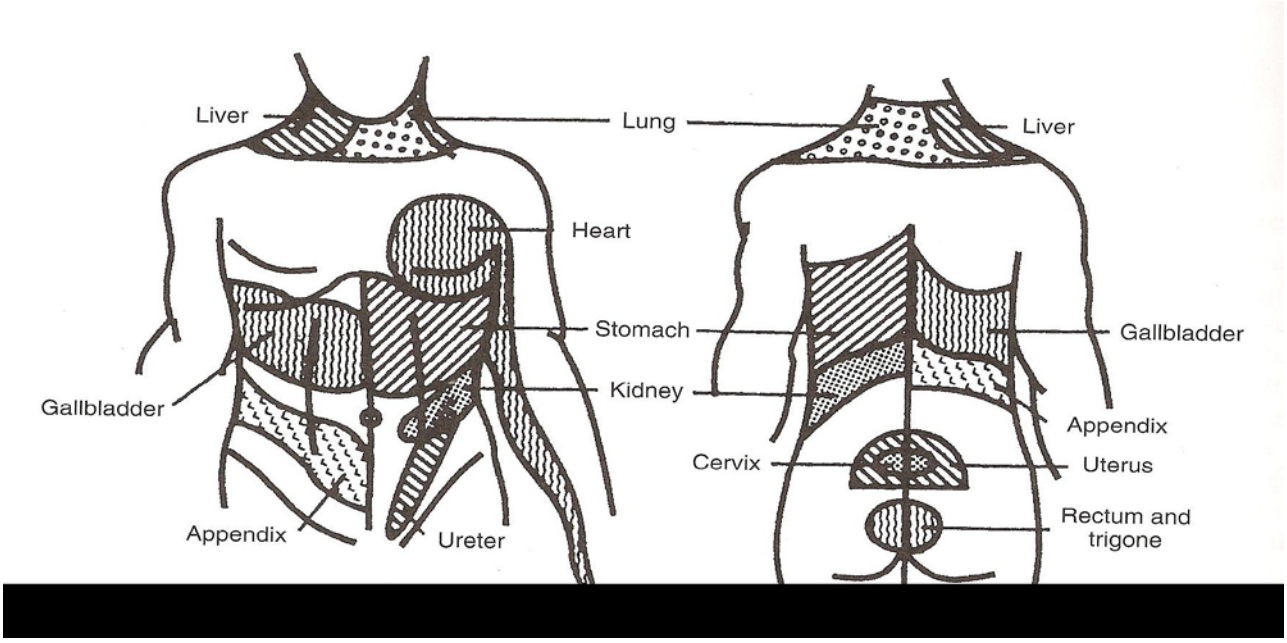

Figure 2. Common sites for referred pain from the liver/gallbladder [6]

Figure 3. Conditions associated with pain in various abdominal quadrants (Google Images)