American Jurisprudence

PROOF *of* FACTS

3d Series

PROOF OF NEGLIGENCE IN DIAGNOSIS AND TREATMENT OF ARTHRITIS

Carla R. McBeath, J.D.

PROOF OF NEGLIGENCE IN DIAGNOSIS AND TREATMENT OF ARTHRITIS*

*CARLA R. MCBEATH, J.D.***

Topic of Article: This article is focused on negligence in diagnosis and treatment of "arthritis," which is a breach of the standard of medical care in diagnosing and/or treating a number of degenerative rheumatic diseases which affect the joints, resulting in progression of the disease to the point where plaintiff is seriously disabled, or suffers premature mortality.

This issue may arise in personal injury or wrongful death actions.

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* This article supersedes Negligent and Deceptive Practices in Diagnosis and Treatment of Arthritis, 26 Am. Jur. Proof of Facts 589.

** Carla R. McBeath, J.D., is an attorney admitted to practice in California, a former member of the Los Angeles Trial Lawyer's Association, and former associate with the law firm of Grayson, Maxwell & Sugarman, in Glendale, California. A graduate of Pepperdine University School of Law, and now a resident of New York City, New York, Ms. McBeath has written in the medico-legal and litigation areas of law.

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I. INTRODUCTION

A. Preliminary Matters

§ 1. Introduction; scope of article

Current medical research regarding the mechanism, manifestations, progression and treatment of the arthritic diseases, including information regarding accurate diagnosis and differential diagnosis, is summarized, in order to enable the medical malpractice practitioner to apply medical evidence to facts of the case for the purpose of defining those types of arthritis that require medical intervention as early as possible after diagnosis and to determine the most current state of medical knowledge regarding treatment.[1] Standard criteria for classification of arthritic diseases is outlined,[2] to present source information for the selection of a medical expert and to serve as a checklist, along with expert testimony, as to a general standard of care for rheumatoid arthritis, as well as other related diseases. A summary of legal issues involving a prima facie case is discussed in an

1. See §§ 3-5.

2. See § 3.

overview,[3] as well as in detail,[4] with particular emphasis on the "loss of chance" doctrine, as it relates to medical malpractice recovery for loss of a chance of a better recovery, as a result of misdiagnosis or delay in diagnosis.[5] As rheumatoid arthritis has been the subject of a dramatic change in treatment approach in the last five years, requiring a faster, more aggressive course of treatment than before, this doctrine is directly applicable to potential arthritis causes of action that may arise in the next few years, as physicians apply the new treatment methods or fail to apply them.[6] Defenses to a medical malpractice claim are addressed,[7] and checklists of recoverable damages[8] and the elements necessary to prove negligence in the diagnosis or treatment of arthritis are provided,[9] along with sample interrogatories[10] and sample testimony.[11] Finally, a bibliography is provided to assist the practitioner in further research on this subject.[12]

B. Medical Background

§ 2. Classification and clinical definition of arthritis-related diseases

Arthritis is a rheumatologic disorder of the joints, characterized as an autoimmune disorder, with multisystem involvement.[13] Its two major classifications are osteoarthritis and rheumatoid arthritis, although it has infectious forms (septic and reactive arthritis), a manifestation with onset under age 15 (juvenile rheumatoid arthritis), a manifestation developing from joint trauma (traumatic arthritis), a manifestation accompanied by a skin disorder (psoriatic arthritis), associated with malignancy (metastatic carcinoma) and associated with human immunodeficiency virus (arthritis

3. See § 7.

4. See §§ 8-11.

5. See § 12.

6. See § 6.

7. See § 13.

8. See § 14.

9. See § 15.

10. See § 16.

11. See §§ 17-22.

12. See § 23.

13. Scutellari, Rheumatoid Arthritis: Sequences, 27 Supp. 1 Eur. J. Radiol. S31 (May 1998).

and HIV). These forms of arthritis, which are not an exclusive list of all reported manifestations of the disease, are discussed in detail in this article.[14]

Osteoarthritis

The most common human joint disorder, osteoarthritis can be clinically detected in a majority of individuals over the age of 65. As it affects many joints, and takes a number of forms, the disease has been defined by the American College of Rheumatology as "a heterogeneous group of conditions that lead to joint symptoms and signs which are associated with defective integrity of articular cartilage, in addition to related changes in the underlying bone at the joint margins."[15]

The process of development of this disease takes two paths. There is an interruption of the ongoing process of deterioration and repair of the articular cartilage and subchondral bone, which results in loss of some cartilage and formation of new bone around the joint. The first path is when mechanical stress to the joint is not increased or unusual, but the supporting structures of the joint are unable to bear them. Joints may have inadequate musculature, bones, ligaments or cartilage to bear normal stresses. The second path is when the structure of the joint is adequate for normal stress, but the joint is affected by the cumulative effect of abnormal stress over a period of time, or continuous abnormal stress. Because the effect is a result of continuous stress, it is more likely that the source of abnormal stress is occupational, rather than recreational. In any event, the chondrocyte (the central cellular structure of cartilage, which supplies nutrients to maintain the elasticity of cartilage) is eventually injured, cartilage is lost or broken down and osteoarthritis develops.[16]

As a disease, osteoarthritis is classified as either idiopathic (primary), when the disease is unrelated to another disorder, and secondary, in which the disease is related to another condition. Focused on destruction of joint cartilage, idiopathic osteoarthritis is associated with a number of risk factors other than age which affect cartilage health and maintenance, including recreational and occupational injuries and trauma. One major risk factor in young patients for later developing osteoarthritis is obesity. Overweight young adults have been found to have an increased risk for

14. See §§ 3, 5.

15. Hahn & Edwards, Osteoarthritis: Presentation, Pathogenesis and Pharmacologic Therapy, Clin. Rev. 9 (Summer 1998).

16. Hahn & Edwards, Osteoarthritis: Presentation, Pathogenesis and Pharmacologic Therapy, Clin. Rev. 9 (Summer 1998).

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16. Hahn & Edwards, Osteoarthritis: Presentation, Pathogenesis and Pharmacologic Therapy, Clin. Rev. 9 (Summer 1998).

osteoarthritis during the next 36 years.[17] These risk factors may be joint-specific. There is a correlation between obesity in women and osteoarthritis of the knee, but not of the hip. Furthermore, traumatic injuries to the joints, such as ligament tears and fractures, lead to osteoarthritis. [18] There is a debate in the medical community as to the role certain occupations, such as coal mining, play in the pattern of osteoarthritis, and that issue is under study.

The significance of an understanding of secondary osteoarthritis is that the underlying condition may require therapeutic intervention, and a discovery of the osteoarthritis may lead to a discovery of the underlying condition in time to initiate successful therapy. Underlying causes have been divided into five main groups of disorders which accompany secondary osteoarthritis:

1. Inflammatory disorders: rheumatoid arthritis, septic (infectious) arthritis, hemophilic arthropathy;
2. Chemical disorders: trauma (either acute or chronic), neuropathic arthopathy, and avascular necrosis;
3. Endocrine-related disorders: diabetes mellitus, acromegaly, Iatrogenic Cushing's syndrome, sex hormone abnormalities, hyperparathyroidism, hypothyroidism;
4. Metabolic disorders: chondrocalcinosis, gout, hemochromatosis, ochronosis, Wilson's disease, Paget's disease, Gaucher's disease, osteopetrosis; and
5. Developmental abnormalities: congenital hip hysplasia, epiphyseal dysplasia, slipped capital femoral epiphysis, Legg-Calve-Perthes disease, femoral head and neck abnormalities, Morquio's syndrome.[19]

Rheumatoid Arthritis

A chronic inflammatory process involving multiple joints, rheumatoid arthritis (RA) is a very common disease, with a variation of symptoms among patients. The usual age of onset is from 40 to 50 years, and prevalence in women is two to three times that seen in men. The trigger, or origin of the inflammation, has, so far, not been discovered, although

17. Hahn & Edwards, Osteoarthritis: Presentation, Pathogenesis and Pharmacologic Therapy, Clin. Rev. 9 (Summer 1998).

18. Hahn & Edwards, Osteoarthritis: Presentation, Pathogenesis and Pharmacologic Therapy, Clin. Rev. 9 (Summer 1998).

19. Hahn & Edwards, Osteoarthritis: Presentation, Pathogenesis and Pharmacologic Therapy, Clin. Rev. 9 (Summer 1998).

there are theories that a bacterium or virus has a role in initiating the disease. [20]

Typical symptoms are pain and swelling in joints commonly affected by the disease, and infrequently involved in other diseases, such as wrists, ankles, metacarpophalangeal, metatarsophalangeal, knees, elbows and shoulders. However, as rheumatoid arthritis is a systemic disease, most patients experience inflammatory processes in other systems, leading to a number of extraarticular symptoms. Commonly seen are rheumatoid nodules, firm subcutaneous nodules appearing around joints or tendons, pulmonary lesions (serious pulmonary and cardiopulmonary complications may be a result of progression of the disease), vasculitis, and vasculitic skin lesions.[21] There may also be renal complications, such as amyloidosis, or ocular manifestations, such as keratitis or scleritis.[22]

Classification of rheumatoid arthritis has been standardized by two diagnostic guides: the classification criteria of the American Rheumatism Association (ARA) and the American College of Rheumatology (ACR), which are virtually identical lists of symptoms, compiled in 1987. The ARA lists the following symptoms, and directions for their application:

1. Morning stiffness or inactivity in and around joints for at least 1 hour before maximal improvement;
2. Soft-tissue swelling or fluid (not bony overgrowth alone) of three or more joint areas (observed by diagnostician). Areas are right or left proximal interphalangeal (PIP), metacarpophalangeal (MCP), wrist, elbow, knee, ankle, and metatarsophalangeal (MTP) joints;
3. Swelling of at least one PIP, MCP, or wrist joint;
4. Symmetric arthritis—simultaneous bilateral involvement of same joint areas as in (2). Absolute symmetry of PIP, MCP or MTP points is not necessary;
5. Rheumatoid nodules;
6. Presence of abnormal amounts of serum rheumatoid factor;
7. Characteristic radiographic erosions and/or unequivocal bony decalcification in or adjacent to hand and/or wrist joints.

Note:

- Four or more of the above criteria are needed for an RA diagnosis
- Criteria 1 through 4 must be present for at least 6 weeks

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20. Wise, Rheumatoid Arthritis: Clinical Manifestations and Diagnosis, Clin. Rev. 23 (Summer 1998).

21. Wise, Rheumatoid Arthritis: Clinical Manifestations and Diagnosis, Clin. Rev. 23 (Summer 1998).

22. Pagano, Rheumatoid Arthritis—an Update, 6(8) Clin. Rev. 65 (1996).

- The presence of criteria is not conclusive evidence for RA diagnosis
- Absence of criteria is not conclusively negative[23]

Prognosis for chronic rheumatoid arthritis is poor, as it directly causes serious disability and shortens life expectancy. It frequently leads to irreversible deterioration of the joint, including destruction of cartilage, erosion of the bone itself, and damage to certain ligaments and tendons. As the joints become affected, muscle wasting occurs and loss of motion is common.

As this is a disease which progresses over many years, its later stages include marked deformities of the joints, which vary according to the areas affected. Most common deformities are in the hands, fingers and wrists, cervical spine, damage and deformities in the elbows, shoulders and hips, as well as loss of motion and stability in the knees. In addition, the ankles and toes may show marked deformities. With deformity, there is a correlating limitation of movement of the joint, loss of use of the muscle and increased pain.[24]

The effect of this progression has led to disability in 50% of patients after 10 years, according to one source. Furthermore, mortality is higher in patients with the disease, in that life expectancy is 10 years less than that of a control group, and there is a higher incidence of mortality from infection, pulmonary disease or complications, renal complications and gastrointestinal causes.[25] One researcher reports that mortality in patients with severe rheumatoid arthritis or extra-articular symptoms is equal to that of patients with triple artery coronary disease or stage IV Hodgkin's lymphoma.[26]

Juvenile Rheumatoid Arthritis

Considered to be a separate disease from adult-onset rheumatoid arthritis, juvenile rheumatoid arthritis affects children and adolescents under the age of 15. It is a chronic, painful, inflammatory, multisystem disease that manifests some identical and some very similar clinical symptoms as adult rheumatoid arthritis. As a result, it is not described in detail in this article. In addition, however, as it affects children during

23. Pagano, Rheumatoid Arthritis—an Update, 6(8) Clinician Review 65 (1996).

24. Wise, Rheumatoid Arthritis: Clinical Manifestations and Diagnosis, Clin. Rev. 23 (Summer 1998).

25. Wise, Rheumatoid Arthritis: Clinical Manifestations and Diagnosis, Clin. Rev. 23 (Summer 1998).

26. Buckley, Science, Medicine and the Future: Treatment of Rheumatoid Arthritis, 315(7102) Brit. Med. J. 236 (July 1997).