APPROACH TO VERTEBRAL FRACTURES DUE TO OSTEOPOROSIS

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ABSTRACT:

Osteoporosis which is characterized by a decrease in bone mass, is a common disease of the elderly. Primary osteoporosis type I which effects trabecular bone, increases the risk of vertebral fractures. Although standart therapy for such fractures is conservative, in case of high risk of neurological complication, surgical intervention via anterior or posterior approach is necessary. Along with fracture treatment measures to prevent bone loss and increase bone mass should be taken. The best way for individuals to prevent osteoporosis is to take calcium and vitamin D and to engage in weight bearing exercise. Women should consider taking estrogen after menopause. Biphosphonate or calsitonin are recommended for patients with significant bone loss. A late complication of osteoporotic vertebral fractures is kyphosis.

Key Words: *Osteoporosis, vertebral fractures.*

INTRODUCTION

Osteoporosis is a common disorder that represents a decrease in bone mass and leads to fracture after minimal trauma. The disease is becoming more prevalent as the average life span increasing (3).

Bone mass in men and women reaches its peak at approximately 30 years of age. Bone loss then proceeded at a rate of 0.5-1% per year in both sexes. Women have a precipitious drop in bone mass over 2% per year that starts with menopause and contious drop in bone mass over 2% per year that starts with menopause and continues for approximately ten years. Apart from these ten years, women lose bone at the same rate as man; approximately 0.5% per year (3, 9, 16).

The causes of osteoporosis are classified as primary or secondary. Primary osteoporosis has been divided into two categories. Type I (postmenapausal osteoporosis) and type II (senile osteoporosis) (13). Secondary osteoporosis has multiple etiologies, including endocrine diseases (thyrotoxicosis, primary hiperparathyrodism, Cushing's syndrome and hipogonadism), malignancy (malignant myeloma), osteomalacia (malabsorption syndromes and malnutrition) and long term use of such drugs as corticosteroids and heparin.

Type I osteoporosis is the most common type of osteoporosis and it's six to eight times as common in women as in men. It occurs within 15 to 20 years after menopause. Estrogen deficiency in women and testosterone defiency in man are the primary causes of this type of osteoporosis. Type I osteoporosis affects trabecular bone more than cortical bone, and manifests itself mainly by vertebral fractures of the painful crush type. Calcium deficiency plays no role (3, 13). Type II osteoporosis often leads to hip fractures and to vertebral fractures of the painless multiple wedge type. Type II osteoporosis is twice as common in women as in men. A long history of calcium deficiency is largely responsible for this condition, which is called senile osteoporosis because it occurs in men and women over the age of 70 (3, 16).

VERTEBRAL FRACTURES DUE TO OSTEOPOROSIS

Osteoporosis is a common disease of the middle-aged and elderly. In women, vertebral fracture incidence increases after menopause and this increase continues with age (3, 8, 13, 16). Trauma such as weight lifting while arms extended, falls while in a sitting position, horse riding may cause vertebral fracture in osteoporotic people. In severe cases of osteoporosis even sneezing or a simple cough can cause a vertebral fracture. Osteoporosis is the cause of 20% of all vertebral compression fractures (7).

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Common type of osteoporotic vertebral fractures include wedge fractures of the throracic spine and crush fractures of the lumbar spine (7, 14). Osteoporosis does not commonly cause wedge fractures above T7. Such a fracture should suggest an infection or metastasis (4).

CLINIC FEATURES

Usually the initial symptom of osteoporotic vertebral fracture is sudden and serious back pain. However, asymptomatic kyphosis or short stature can also be the first sign. A newly developed vertebral fracture may be accompained by local tenderness and muscle spazm. Transient ileus or urinary retantion may be observed. Although radicular pain neurological defects do not commonly occur, the possibility of spinal chord compression should always be kept in mind.

In some cases due to the wedging of multiple vertebrae progressive kyphosis and short stature develop. Thoracic and abdominal cavity volumes are decreased because of the shorthening of vertebral column. Lung capacity decreases and a increase of pressure into the abdominal cavity causes a potbelly appearance. As a result of malfunction of the gastrointestinal organs, weight loss is observed. With the progression of the kyphosis a restrictive lung disease is added to the clinical picture.

DIFFERANTIAL DIAGNOSIS

The first step in working up the patient presenting with a vertebral crush fracture is to rule out trauma, tumor or the four major forms of osteopenia (bone marrow abnormality, endocrinopathy, osteomalasia and osteoporosis).

If the high-energy trauma is eliminated and there is no evidence of local pathology, such as tumor, the physician should rule out osteopenia using one of the photon absorptiometry method. The physician then investigates the following signs of a bone marrow disorder; anemia, elevated ESR, abnormal serum protein and electrophoresis results. If any of these are positive bone marrow analysis may be needed. Approximately 2% of individuals with osteopenia will have a bone marrow etiology. In the face of a negative bone marrow screen, the common endocrinophaties (hyperthroidism, hyperpara-throidism, type I diabetes and iatrogenic Cushing's syndrome) should be rule out by checking plasma levels of these hormones and complete history. Approximately 5-15% of individuals with osteopenia will have a endocrinopathy. If endocrinophaties are rule out, more attention should be directed toward osteomalasia. Eight percent of individuals with osteopenia will have some evidence of this syndrome, which produces decreased levels of calcium and phosphorous, a highly increased level of alkaline phosphatase, decreased 25 hydroxy vitamin D and increased prathyroid hormone. A negative analysis for osteomalasia leaves primary osteoporosis as the cause of vertebral crush fracture.

EARLY PHASE CONSERVATIVE TREATMENT

Main purpose is to stop the pain. If there is no neurological complication, bed rest and analgesics are the standart therapy. Early phase cold application for a few hours could be beneficial. After then, wet and hot compresses every 20-30 minutes should be used to combat the muscle spasm. For cases with very severe pain narcotic analgesics can be employed but such drugs should only be used for a short period of time and closely monitorised for various side effects.

In order to prevent neurological complications and to ease the pain orthoses can be used. A thoracolumbosacral or lumbosacral orthose should be used depending on fracture type or location. If there is a serious risk of neurological complications a Jewet type hyperextantion brace can also be used (3, 4).

EARLY PHASE SURGICAL TREATMENT

If the osteoporotic crush fracture is burst type and associated with a high risk of neurological compromise, surgical treatment is indicated. This treatment includes anterior decompression and reconstruction. Anterior reconstruction is accomplished by use of allograft or methylmethacrylate. Allograft, autograft or methylmethacrylate is conjunction with internal fixation ensure immediate stability. Posterior spinal instrumentation is an alternative techniques. This technique does not allow a direct decompression but indirect decompression obtained by this method is sufficient. This technique is particularly recommended for the patients with multiple fractures requiring lengthy posterior instrumentation or multiple level chord compression or those who can not tolerate anterior surgery (3, 4, 5, 15).

PREVENTION

Along with fracture treatment, prophylactic drug therapy for osteoporosis should be started and necessary measures should be taken in order to prevent furtheir bone loss.

In order to treat osteoporosis, the histology of the disorder must be understood. High-turnover osteoporosis is associated with increased osteoclastic resorption, while low-turnover osteoporosis is associated with decreased osteoblastic formation. There are two main groups of drugs used for osteoporosis prophylaxis.

1. Antiresorptive Agents: Calcium salts, vitamin D, estrogen, calcitonin and biphosphonate.

2. Bone-stimulating agents: Sodium fluoride and recombinand parathyroid hormone.

Calcium Salts: These cause a decrease in parathyroid hormone secretion by a negative feed back mechanism and are a safe and effective drug for mild cases of osteoporosis (8, 11).

Vitamin D: Supplementation with vitamin D is beneficial in case of a deficiency. However, a daily dose of 400-800 IU is usually suggested (8).

Estrogen: Women may benefit from low dose of estrogen supplementation therapy. Such a long term therapy increases the risk of endometrial carcinoma. In order to minimalize this risk, estrogen is given in combination with progestin. Osteoporosis prophylaxis with estrogen is effective for only 1-2 years. After then there is no benefit. However, in cases with severe bone loss estrogen monotherapy is not sufficient, combination with calcium salts and vitamin D is necessary (1, 8, 10).

Calcitonin: Calcitonin is the physiologic parathyroid hormone antagonist. It inhibits osteoclast activity, calcium mobilisation from bone and destruction of the protein matrix. In severe cases of osteoporosis a dosage of 50-100 U im-sc every other day or three times a week improves bone density, lowers the risk of vertebral fractures and diminishes pain due to compression (2, 8). Similar results can be achieved by daily 50-100 U intranasal application (11). Calcitonin treatment should be in combination with oral calcium supplementation. After long term treatment with calcitonin, the efficiency of this drug decreases (8).

Biphosphonates: These antiphagocytic agents settle in bone crystals and slowly hydrolized. Of first generation biphosphonates etidronate is given for two weeks with a 400 mg per oral daily dose and this treatment is repeated every three months for two years. In order to prevent the minerilazation inhibiting effects of this drug newer forms such as pamidronate and alendronate have been developed (3, 6, 8, 10).

Sodium Fluoride: Sodium fluoride is given 60 mg per oral daily but has a rather small therapeutic index. Although it increases bone density, this drug does not reduce the incidence of vertebral fractures for it causes thinning of cortical bone. Even, it causes an increases of lower extremity fractures (8, 12).

Consequently, the best way for the individuals to prevent osteoporosis is to take calcium and vitamin D and engage in weight-bearing exercise. Women should consider taking estrogen after menopause. Biphosphonate or calcitonin are recommended for patients with significant bone loss.

LATE PHASE CONSERVATIVE and SURGICAL TREATMENT

In this stage, the aim of treatment is to relieve the symptoms caused by developing kyphosis and to prevent kyphosis progressing. These aim can be achieved by conservative measures in most cases. These measures include non-steriodal anti-inflammatory medication, analgesics and a variety of orthotics ranging from reinforced support garments to molded-plastic braces (3.4).

The indication for surgical intervention are pain that not respond to medication, progressive deformity and neurological impairment. The goals of surgical treatment must be appropriately limited to alleviate pain and restore or preserve neurologic function. Correction of the deformity should not be a priority in the surgical treatment of osteoporotic kyphosis. Posterior instrumentation and cansellous allograft fusion is recommended. The instrumentation should be extended both proximally and distally of the kyphosis to limit adding on by further compression fracture (3, 4).

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