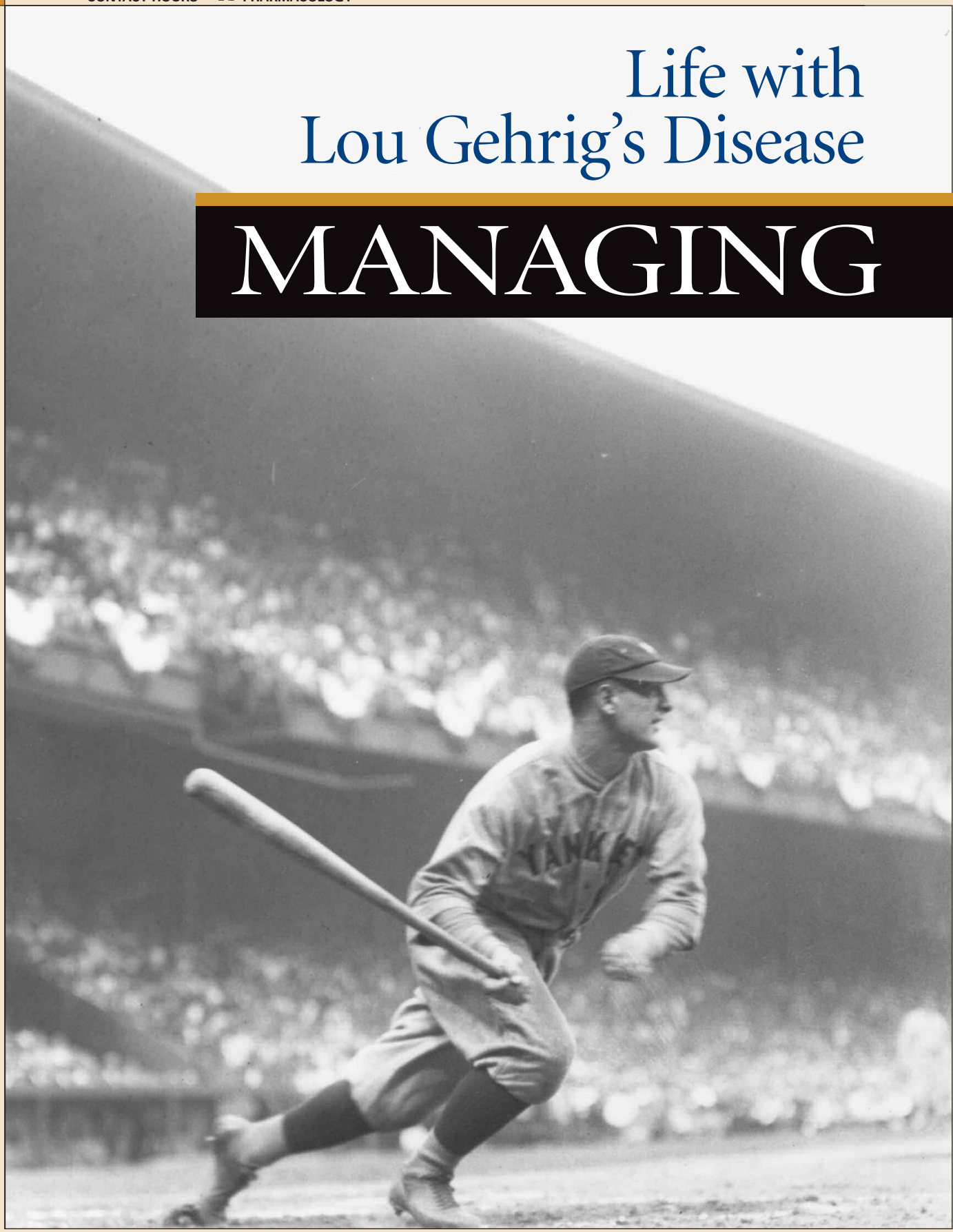
Life with Lou Gehrig's Disease

MANAGING



ALS SYMPTOMS

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Amyotrophic lateral sclerosis (ALS), or Lou Gehrig's disease, is a progressive neurologic disease that involves degeneration of the upper and lower motor neurons that control muscular activity and causes progressive muscle weakness, atrophy, and spasticity. ALS is a chronic, debilitating, and eventually fatal disorder with devastating symptoms.

ALS was first described in 1869 by Jean-Martin Charcot. Lou Gehrig first brought national and international attention to the disease when he abruptly retired from professional baseball in 1939.

Although the cause of ALS is unknown, genetics and toxic accumulations of glutamate have been suspected. In 5% to 10% of cases, the disease is familial with autosomal dominant inheritance. Because ALS is a rare disease, there are few controlled clinical trials for symptom management.

This article aims to clarify the patient's perspective, incidence, pathophysiology, diagnostic challenges, and management of the disease and its common symptoms. Because misdiagnosis is common, advanced practice clinicians need to understand the presenting symptoms and the patient's perspective. Nurse Practitioner's (NPs) can play an important role in communicating the diagnosis and management of ALS.

Incidence

Approximately 5,000 new cases are diagnosed yearly in the United States. ALS usually occurs between age 40 and 70, and 90% of cases represent a sporadic or idiopathic form of the disease. The incidence increases with each decade, and the average age at onset is 63 years. No racial differences are evident in the disorder. The male to female ratio is 1.3 to 1.5 but approaches 1 to 1 at ages older than 70 years.[1]

Early respiratory or bulbar symptoms and increasing age are poor prognostic indicators. More than one-half of ALS patients die within 3 years and 90% within 5 years of the first symptom(s).[1]

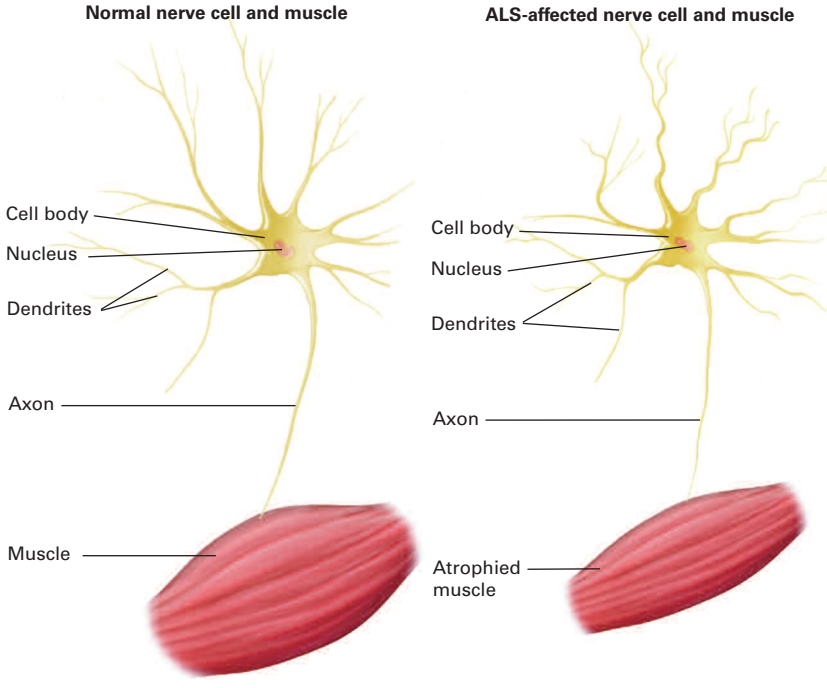
Pathophysiology

ALS is a degenerative motor-neuron disease that can affect lower and upper motor neurons (see Table: "Motor Neuron Changes in ALS"). These motor neurons degenerate and cause death in the brain and spinal cord. Lost neurons are replaced by astrocytic gliosis. Cortical motor cells disappear leading to retrograde axonal loss and gliosis in the corticospinal tract. The gliosis leads to bilateral changes in the white matter in the brain and is visible upon magnetic resonance imaging (MRI). The spinal cord atrophies and large myelinated fibers in motor nerves are lost. The affected muscles atrophy with a loss of frontal or temporal cortical neurons, particularly in frontotemporal dementia.[2]

Symptoms

Cramps in distal limbs are often the first sign of ALS. Weakness and wasting of the arms, legs, and hands develop over months. Symptoms typically include twitching, stiffness, and muscle cramps. Over time, walking or climbing becomes difficult and the person may stumble or drag a foot. Sometimes the oral muscles degenerate and cause slurred speech, hoarseness, or difficulty swallowing.[1] Mood swings, anxiety, and depression may occur. Most patients report a relentless and rapid deterioration of axial and limb muscle strength and endurance coupled with dysphagia (difficulty with swallowing) and dysarthria (difficulty with speech).[2] Early in the disease one limb is involved, and later in the course of the illness the other limbs become involved with hyperreflexia, spasticity, and atrophic weakness. Symptom management parallels the progressive functional decline (see Table: "Management of ALS Symptoms"). Death (usually from respiratory failure) typically occurs 3 to 4 years after diagnosis.[1]

Motor Neuron Changes in ALS



ALS causes progressive degeneration of the upper and lower motor neurons. Current research suggests that glutamate, the primary excitatory neurotransmitter, accumulates to toxic levels at the synapses. The affected motor units are no longer innervated, resulting in degeneration of axons and loss of myelin. In an attempt to maintain function, nearby motor nerves may sprout axons, but eventually normal neuronal tissue is replaced by nonfunctional scar tissue.

In clinical terms, the lower motor neurons have muscle weakness, atrophy, and fasciculations or twitches. The upper motor neurons demonstrate increased muscle stretch reflexes, spasticity, or hyperreflexia.[1] Other signs include a brisk jaw reflex, Babinski's sign, and pseudobulbar palsy with emotional lability. However, the senses of vision, touch, hearing, taste, and smell are not affected. Complications may include skin ulcers, respiratory dysfunction, aspiration pneumonia, chronic osteomyelitis, and pulmonary embolism due to deep vein thrombosis. A small group of patients have cognitive loss.[5]

Physical Examination

The patient's personal and family history is germane to an accurate diagnosis. Because some ALS in inherited, the family history should include questions regarding the disability in first-degree relatives.

Established risk factors include age and family history; suggested risk factors include:

- laborers engaged in agricultural work, factory work, heavy manual labor, and welding.
- repetitive muscle use, athleticism, trauma, and electrical shock.

Symptoms related to upper and lower motor neurons should be examined. Perform a thorough neurologic examination. It is speculated that some occupations may involve toxic exposure to minerals such as lead or aluminum.[5] A comprehensive evaluation includes laboratory and electrodiagnostic testing.

Diagnostic Challenges

Because symptom onset is insidious and mimics other diagnoses, such as arthritis, an accurate diagnosis is challenging. The patient may consult multiple clinicians before receiving an accurate diagnosis. Two sets of criteria, El Escorial and Airlie House revision[4] define the diagnosis. Diagnostic criteria include evidence of progressive lower and upper motor neuron dysfunction as measured by electrophysiologic, pathologic, or radiologic testing.

Blood and urine studies (thyroid and parathyroid hormone levels, 24-hour urine collection for heavy metals) may be done, as well as lumbar puncture; X-rays including of cervical spine; muscle and/or nerve biopsy.

No specific test is used for diagnosis, but electrical nerve tests (electromyography) examine the nerve currents while MRI may evaluate if a structural, spinal fluid, or infective cause exists. Electrophysiology studies confirm the diagnosis and show denervation and reactivation without conduction block.

Once diagnosis is made, it is correct more than 90% of the time.[1]

Communicating the Diagnosis

Healthcare professionals often have difficulty breaking bad news about a progressive and life-limiting disease. Practitioners should emphasize that the intellect is not affected and that many interventions and resources help to manage the physical symptoms and emotional distress. In their study of relatives of patients with ALS, Bolmsjo et al.[4] found that the interviewees reported that the information they received was inadequate and was not presented in a thoughtful, sensitive manner; the relatives considered the professionals to be incompetent.

The clinician who is delivering the diagnosis should preselect a quiet and private area, encourage the patient to bring

Management of ALS Symptoms

Symptom	Description	Treatment options
Muscles		
Muscle cramps	• Cramps are often the first symptom of the disease, occurring months before the patients notice weakness and wasting. • Vary from mild, without affecting daily activities and sleep, to disabling. • May involve any muscle or muscle group (jaw, abdominal, chest, back, neck, extremities).	• Quinine is effective and has a low cost. • Baclofen is less effective and more expensive. • Gabapentin is used but no better than placebo.
Spasticity and stiffness	• Individuals notice a sensation of varying degrees of stiffness or tightness in the affected muscles. • Loss of control of muscles or stiffness reduces daily activity. • Voluntary movement becomes increasingly difficult and the affected limb(s) might twitch or jump and are difficult to position or move.	• Passive stretching exercises may be beneficial. • Muscle relaxants (baclofen, dantrolene, or tizanidine) may have benefits but also increase weakness.
Musculoskeletal pain	• Pain in affected extremities.	• Muscle relaxants. • Nonsteroidal anti-inflammatory drugs. • Nonopioid analgesics.
Conditions of the Mouth		
Dry mouth	• Can be caused by medications with anticholinergic effects from mouth breathing during sleep and from treating sialorrhea.	• Hydration and a room humidifier can be effective during sleep.
Thick mucus	• Thick secretions often cannot be cleared with cough.	• Expectorant (guaifenesin). • Beta-adrenergic blocking agents (propranolol or metoprolol) effective in 75% of subjects.
Sialorrhea	• Reduced clearance of saliva from less frequent swallowing. • Occurs in 50% of ALS patients.	• The anticholinergic, glycopyrrolate, was 90% effective. Atropine may also be effective. • Botulinum toxin injected into parotid and submandibular glands has been successful but may have side effects of bulbar muscle and temporary masseter muscle weakness. • Amitriptyline may decrease secretions due to anticholinergic effects.
Emotions		
Labile emotional affect	• Laugh and cry easily and sudden loss of emotional control in response to nonspecific stimuli. • Occurs in up to 56% of patients.	• The tricyclic antidepressant, amitriptyline, is an effective and low-cost option.
Depression	• Affects about 33% of patients.	• Selective serotonin reuptake inhibitors (SSRIs), such as paroxetine or sertraline are the treatment of choice. • The tricyclic antidepressant, amitriptyline, may also be effective.
Anxiety	Occurs in 10% of patients and may increase to 25% to 55% near the end of life.	• Anxiolytics have similar efficacy, but lorazepam and diazepam are less expensive. • SSRIs such as paroxetine or sertraline may be effective. • The tricyclic antidepressant, amitriptyline, may also be effective.
Sleep problems	• Identify and treat the underlying cause: depression, dysphagia, cramps, inability to change posture.	• Chloral hydrate, diphenhydramine.

a supportive family member, and offer an opportunity for follow-up discussions.[1] Often a colleague with psychosocial expertise, such as a psychologist, social worker, or psychiatric nurse, can help break the news. The wise clinician asks what the patient suspects about the problem and what he/she wants to know. Giving the information that the patient wants is important and may take several discussions. Listening to what the patient says, suspects, or fears is as important as giving information. Using the patient's words to explain the disorder will improve communication and decrease medical jargon.

Management

Although management involves multiple disciplines, NPs are ideally suited to manage ALS symptoms because of their unique type of relationship with their patients, their psychosocial expertise, and their awareness of the patient's experience of symptoms. NPs are also well suited to educate, counsel, help patients cope with symptoms, and discuss end-of-life concerns. A multidisciplinary team provides close follow-up and management. The team includes a neurologist, dietitian, physical therapist, and others based in and out of the hospital community.

Presently, riluzole (Rilutek) is the only medication approved by the U.S. Food and Drug Administration to treat ALS.

Managing ALS involves addressing both psychosocial and biomedical issues. The diagnosis is stressful and elicits feelings of anger, fear, and lifestyle changes. The cornerstone of care is good symptomatic management, including nutritional support and ventilatory care with noninvasive ventilation. Consideration of the early use of technology has a major impact on the quality of life and survival by minimizing cachexia, starvation, and aspiration.

Nutrition Intervention

Prior to diagnosis, the patient took eating and swallowing for granted, now muscle weakness can cause fatigue, dysphagia, and serious problems. Eating and drinking become an exhausting chore and are accompanied by fear of choking and aspiration. The NP will collaborate with the dietitian to manage these issues. For instance, the NP may refer the patient with difficulty swallowing liquids to the dietitian for thickened liquids.

Patients with ALS have a high risk of malnutrition due to cachexia, dysphagia, the inability to grocery shop and/or prepare meals, inadequate nutrient intake, and unintentional weight loss. The dietitian evaluates and assesses these high-risk patients, identifies and addresses their concerns with nutrition interventions, and modifies the oral and/or enteral feeding regimen. The dietitian also teaches patients and caregivers about the appropriate oral or enteral diet prescription.

The dietitian and NP work closely with the speech pathologist to enhance the safety of a patient's meals. A speech pathology evaluation helps determine the patient's baseline swallowing function upon diagnosis. The speech pathologist can perform a swallow study to determine the most appropriate food and liquid texture for a patient with dysphagia. The dietitian should also assess the adequacy of a patient's nutrient intake. This can be determined using elementary tools such as 24-hour food recall, food frequency questionnaire, review of food journals, or direct observation of meal intake for inpatients. The gold standard to assess energy needs is indirect calorimetry to determine optimal caloric requirement as underfeeding an ALS patient can result in diaphragm impairment, and over-feeding can increase ventilatory load.[7]

As ALS progresses and a patient develops difficulty with chewing and swallowing, special focus on a high-calorie, high-protein diet becomes necessary. Nutrition supplements, such as protein powder or liquid beverages, may be needed. Certain oral nutrition supplements are used cautiously for patients with swallowing difficulty who need thickened liquids.

As ALS progresses a patient may not be able to meet their increased nutrient needs. Alternate routes for enteral nutrition may be needed. The most commonly used modes are the percutaneous endoscopic gastrostomy and the percutaneous radiographic gastrostomy (PRG). PRG is the preferred method of placing an enteral feeding tube as it requires less sedation, has a lower risk of aspiration associated with averting the endoscopic route of placement, and appears safer for ALS patients with moderate or severe respiratory impairment.[8]

Decisions regarding timing, safety, and utility of enteral feeding tube placement are crucial in the management of an ALS patient. Placement of an enteral feeding tube should be considered before forced vital capacity declines to 50% and/or body mass index is less than 18.5 kg/m.[3,9] Although at first a patient may be able to receive the combination of oral and enteral feeds, eventually exclusive use of enteral feedings becomes necessary as dysphagia worsens.[9] If a combination oral and enteral regimen is feasible, tube feedings

can be provided nocturnally on a compressed feeding schedule to maximize a patient's appetite and oral intake during the day. The dietitian can select an enteral formula and additional free water order to meet estimated nutrient needs. The dietitian should be mindful of unique fluid and fiber needs to prevent constipation that occurs in some ALS patients secondary to weakened pelvic muscles used for defecation.[9] The discussion about feeding tube placement may be initiated by the NP in collaboration with the dietitian to provide specific information about the nutrition care plan. The dietitian, along with nursing staff, can provide education on tube feeding administration for home use.[9] If a patient opts not to use a feeding tube, nutrition education can be provided to optimize oral intake.[9] Principles of a dysphagia diet, as indicated by speech pathology, along with aspiration-precaution guidelines should be reviewed to minimize risk of aspiration.

The role of nutrition supplements and complementary therapies require consideration. At present, riluzole (Rilutek) is the only medication approved by the U.S. Food and Drug Administration for use to treat ALS.[7] It reduces the presynaptic release of glutamate, protects neurons by reducing excitotoxicity, and is the only drug to impact survival. Riluzole acts on the 1A2 substrate. The dose is 50 mg orally every 12 hours on an empty stomach. Complete blood cell count and liver function studies should be monitored regularly. Discontinue riluzole if liver function tests exceed five times the upper limit of normal. Adverse drug reactions include nausea, weakness and dizziness, and decreased lung function.

A high-fat meal can decrease absorption of the drug by approximately 20%, therefore, the nutritionist considers the macronutrient composition of meals and the timing of medication administration. ALS patients often self-medicate with vitamins, minerals, herbs, and other dietary supplements.[8] This creates the potential for drug to drug or food to drug interactions.

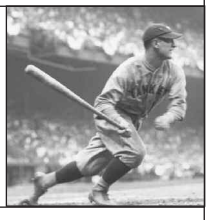
Scientific research on outcomes of dietary supplement use is in the preliminary stage; however, several interesting studies have suggested the use of supplements.[10] Special attention has recently been given to the use of antioxidants to inhibit production of free radicals which are toxic to cells and especially harmful to motor neurons.[10] Commonly used antioxidants include vitamins C and E, and coenzyme Q10. Since vitamin E is fat-soluble, it should be given with meals to maximize absorption. Since ALS patients are at such great risk of malnutrition, a multivitamin and mineral tablet should be prescribed.

The role of nutrition intervention is paramount in the treatment of an ALS patient. Proactive nutrition management independently impacts prognosis and survival. The dietitian plays an important role in the multidisciplinary team. Successful nutrition intervention can result in the stabilization of unintentional weight loss and improvement in quality of life.

Respiratory Symptoms

Weakness of the muscles involved in respiration can cause decreased ventilation and increased risk of dyspnea, aspiration, or sleep apnea. Hypoventilation at night can lead to fatigue, lethargy, and daytime somnolence. Respiratory support can provide symptomatic relief and increase life expectancy but the benefits need to be weighed against the burdens of fatigue. Elective and noninvasive ventilatory support can be provided with oxygen via a mask during sleep; this reduces dsypnea at night but it may not help patients with facial or bulbar weakness. An annual influenza vaccination is recommended to prevent respiratory complications.

Respiratory muscle weakness can cause decreased ventilation and increased risk of dyspnea, aspiration, or sleep apnea.



Bulbar Weakness

Bulbar palsy is one of the most distressing features of motor neuron disease; causes weakness of the tongue, pharynx, and facial muscles; and leads to loss of salivary control. The patient has difficulty with eating and may choke and drool. Providing adequate hydration and control of excess saliva are important. Anticholinergic agents, such as oral atropine or amitriptyline, hyoscine (scopolamine), absorbed in a patch; or glycopyrrolate may control these symptoms but also produce side effects such as confusion and exacerbation of glaucoma). Guaifenesin, the active ingredient in Humabid, has reduced thick phlegm. It can also be reduced by hydration, glycerin swabs, candy, and humidifiers. The parotid gland, which influences saliva, can be irradiated or paralyzed by injection of botulinum toxin. Other interventions include antimuscarinic agents, which thicken secretions, beta-adrenergic blocking agents, such as propranolol and metoprolol, have been reported to reduce secretions without increasing tenacity, and mucolytics (acetylcysteine) that break up secretions. In addition, speech and language therapy can evaluate swallowing and teach strategies to ease mastication and prevent aspiration.

Psychological Symptoms

As the patient watches muscle strength decline, feelings of depression commonly increase and suicidal thoughts may emerge. Symptoms include sad mood or loss of interest or pleasure and at least five other symptoms (fatigue, constipation, significant weight loss, insomnia, diminished concentration, thoughts of death or suicide) over a 2-week period.[11] The clinician can detect depression by asking the patient about these symptoms or using a screening tool, such as the Beck Depression Inventory.[12] The NP who is not skilled in mental health may manage psychological disorders in collaboration with the mental health specialist. Immediate referral is recommended for any patient considered a high suicide risk.

The United States Preventive Task Force recommends screening for all adults in primary care settings for depression.[13] Many well-tested self-report screening tools detect depressive symptoms, establish a baseline, and gather objective evidence. The Beck Depression Inventory[12] and the Yesavage Depression Inventory[14] are commonly used tools. Another recommended approach for screening adults is to ask the following two questions, "Over the past 2 weeks have you felt down, depressed, or hopeless?" and "Over the past 2 weeks have you felt little interest or pleasure in doing things?" These two questions may be as effective as longer tools.[10]

A professionally led caregiver support group can produce significant reductions in anxiety, depression, and sense of burden.

Depression is a complication that is frequently unrecognized and untreated. Although three-quarters of physicians believe that they treat depression well, only 14% to 20% of depressed patients are adequately identified and treated. Patients often do not report depression unless asked because they fear it is due to weakness, being "crazy", or an indication for hospitalization. Counseling, psychopharmacology, and social support can reduce major depression in 80% to 90% of patients.[15] Typically, selective serotonin reuptake inhibitors are the drug of choice for depression in ALS patients.

Patient Education

The NP plays a pivotal role in helping the patient understand the disease and symptom management. Education about the disorder, symptoms, resources, palliative care, and social support resources are needed. The patient needs to know the range of symptomatic and specific treatments, the option for a second opinion, resources such as the ALS Association or the Motor Neuron Disease Association, and the names of specialists, such as a neurologist, who will provide follow-up care. The patient may be worried about prognosis, pain and symptom management, causes of the disorder, and available treatment. Information should be understandable and provided verbally and in writing.

The patient needs to know that palliative care offers symptom management and resources. Palliative care aims to support both the patient and close relatives throughout the patient's illness and death. Researchers who examined the integration of end-of-life nationally developed guidelines for chronic, noncurable, life-limiting diseases found gaps in palliative care. End-of-life care should include content in 15 domains (such as epidemiology of death, symptom management, spiritual, family roles, settings of care), specific terms dealing with palliative care, integration of palliative care information, and descriptive variables. The current national guidelines offer little guidance in end-of-life care issues. Spirituality, ethics, advocacy, and family roles should be considered.[16]

The relatives of terminally ill people suffer emotional distress that NPs can address. Caregivers (family and friends) experience distress from the caring role, emotional toll, and limited support. Bolmsjo and Hermern[6] conducted a Hermeneutic analysis of the relatives' emotional stress and existential issues in caring for a loved one with ALS. Relatives noted that they received inadequate information about disability services, equipment, and available social services. Patients and relatives can benefit from clear discussions about end-of-life choices so the patient can use an advance directive to identify surrogate decision makers and identify desired treatments.

Social Support

Coping with the psychosocial aspects of the diagnosis is challenging and involves the fear of death. Support groups are widely used in ALS for treatment, counseling, and education. Groups help promote social support and health-related psychosocial adjustment and coping. A meta-analysis comparing group and individual therapy found that groups were equally or more effective than individual treatment.[15] Benefits include universality, problem solving, social skill development, altruism, and information as well as security, belonging, and companionship. A professionally led caregiver support group can produce significant reductions in anxiety, depression, and sense of burden. The research, literature, and evaluation data on support groups are limited.

Conclusion

NPs need to understand the diverse symptoms, diagnostic criteria, and symptom management of ALS. They need to know that misdiagnosis of this disease is common. Examination by NPs and other primary care providers is often the first step on a difficult road to diagnosis and management. Improved recognition of symptoms and appropriate referral may shorten the timeline of diagnosis. Referrals should include consultation with a dietitian and a social worker. Patients need to be treated by a multidisciplinary team including a neurologist, a speech therapist, and a social worker or psychologist. Understanding the plan of treatment and monitoring its effectiveness may help decrease the burden of ALS. Caring for a patient with a life-limiting illness requires some knowledge about palliative care and psychosocial support. No strategies for the cure or prevention of ALS exist and knowledge of etiology is limited.

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