Intramedullary spinal cord tumors health and social care essay

Health & Medicine

In this essay I will discourse a patient who had been enduring from Amyotrophic Lateral induration a signifier of Motor Neurone Disease which is a progressive, chronic and debilitating disease. I will discourse the consequence this disease has on the organic structure, look at differential diagnosing, the cardinal status impacting the organic structure by this disease, epidemiology, aetiology, pathophysiology and the associated pharmacological medicine that are all prevalent with Amyotrophic Lateral Sclerosis. Motor Neurone Disease is a general term for a few differential discrepancies of the diseases which are combined under the one umbrella (see appendix A) the specific disease I will discourse is Amyotrophic Lateral Sclerosis (ALS) .

From this point Motor Neurone Disease will be known as MND and Amyotrophic Lateral Sclerosis known as ALS.

The patient I attended was a 53 twelvemonth old who had been enduring from ALS. The patient when we arrived had died but had been in the terminal phases of the disease. In speaking with the household and acquiring a good societal and household history it became evident that this patient had led an active life style until she was diagnosed with ALS. In the early phases this patient managed herself but rapidly became unable to pull off her daily attention and required aid, due to the ALS the patient was known to us but I had ne'er attended to them before. In treatment with paramedic who I was on with they gave me a brief history of the patient so I was prepared when I went in. ALS comes under the umbrella of MND which affects the nervous system (see Appendix B) and can be divided into two countries the lower taking to muscle weakness/wasting (wasting) and the upper taking to

stiffness, abnormally active physiological reactions by and large nonvoluntary and each impacting different countries of the organic structure in changing grades. The forecast for ALS is Death, as it affects the upper and lower nervous system (see Appendix B1) . MND It is a chronic progressive neurodegenerative disease, nevertheless depending on countries affected it may take a few old ages or it can take longer. There is no remedy for MND and the pharmacological medicine is restricted. The pharmacological medicine for the patient was Riluzule in the early phases can decelerate down the devastation of the motor nerve cells as it is a neuroprotector, Baclofen is prescribed as a musculus relaxer which is used to handle the cramps and stiffness caused by the musculuss, Dantrolene is prescribed to handle long term musculus cramps and stiffness though it can besides do liver harm, Diazepam (diazepam) is prescribed for its ataractic consequence and helps command the musculus cramp and stiffness these along with other general medicines and with the aid of psychological and emotional support signifier household, friends and other bureaus all assisted the patient with get bying with their ALS. This patient was in ventilatoryfailurewhich is the terminal phase of the disease.

On Arrival the patient was still on the ventilator and the Patients ' household were around her they stated that she had been experiencing ailing different signifier the disease itself and that she had non been herself, the patient was still able to pass on utilizing her eyes although this was going a strain on her and was now passing greater lengths of clip asleep. The patient had retained her sense of temper throughout the disease. The household managed all the daily patient attention themselves as they stated that they knew her best

and as the disease progressed and the patient was unable to vocalize any longer they were still able to understand what was wanted.

On Examination the patient had no bosom beat and remained in cardiac arrest, patient students were fixed, all marks of life were negative apart from the ventilator. The household knew that the patient had died but we had to repeat that and do certain as they had requested us to go forth ventilator switched on at that clip which meant that there were breath sounds although false reading as it was mechanical aided airing and there was no existent external respiration attempt from the patient it was explained to the patients sister and hubby that although the ventilator is still on it may be confounding for any of the other relations that were coming to understand and particularly for the patients kids as there were no marks of life but the noise of the ventilator might intend they got confused. Dr was called one time we had arrived and we explained what was go oning and as it was the GP that usually attended to the patients ongoing attention we requested that they attend the house. Dr pronounced the patients ' decease and was able to exchange off the ventilator as this was what the household wished, during this clip asperity had begun. The household and Dr stated that the patient was cognizant of the result of the disease and that the patient had a DNAR in topographic point which the Dr brought with them as it was a new issue and the one the household had was out of day of the month. I realised through speech production with the household that the symptoms of this disease were similar to other nervous system diseases and that I knew really small about this I decided I would read up on the disease and larn how these types of neurological diseases affect people.

Differential Diagnosis:

In order to derive the right diagnosing of any disease you must first regulation out several other different diseases that affect the organic structure in a similar manner. In making so you are able to name and handle the disease efficaciously utilizing the right pharmacological medicine, psychologicalscience, or external aid. This is sometimes hard as they frequently present in this instance with muscular blowing particularly of the upper limbs which is the initial presentation in MND and ALS.

Some of the differential diagnosing for ALS and MND are:

Intramedullary tumour - See Appendix B.

Cervical spondylosis - Cervical myelopathy - see Appendix B

Peroneal muscular atrophy- see Appendix B

Chronic polymyositis- see Appendix B

Cervical rib- see Appendix B

Peripheral nervus lesions- See Appendix B (General Practice Notebook, 2012)

Once all the differential diagnosing information has been correlated and ruled out it is so clip to look at the factors of MND and it variants that prevarication within the MND umbrella. There are assorted types of MND with ALS being the most outstanding and besides the 1 that appears to be speedy in oncoming therefore a rapid diagnosing eases the patients

anxiousness and will besides give the patient the right pharmacological medicine and psychological science with support groups and healers to understand the forecast and the disease and its effects on the organic structure although everyone can bring forth different symptoms and no one individual has the same patterned advance through the disease it is in a sense individualised. MND has changing types under its umbrella some affect specific site and others whole countries of the nervousnesss.

Amyotrophic Lateral Sclerosis (ALS) , both upper and lower motor neurone harm.

Primary Lateral Sclerosis (PLS) , upper motor neurone harm entirely,

Bulbar Palsy, the bulbar paralysiss

Progressive Muscular Atrophy (PMA) , lower motor neurone harm entirely

Epidemology of ALS

ALS normally occurs between age 40 and 70, and 90 % of instances represent some signifier of MND disease. The incidence of the disease increases with each decennary, the mean age at oncoming is 63 old ages. The surveies of the addition of the disease are predominately due to one factor that we are all acquiring better wellness attention and that we are in the chief life thirster. Recent information has suggested that there may be some cultural variableness to the disease nevertheless there is still a lower incidence in non-whites or persons of assorted ethnicity. The male to female

ratio is 1. 3 in the ages between 40-70 but approachesequalityat ages older than 70 old ages. (Sharon M. Valente RN, 2007) (Carmel Armon, n. d.)

The highest rate of ALS in the universe is Finland. There has been a recent survey in Finland to happen out why they have such high rates they found that a mutant in chromosome 9p21 as the major cause of familial ALS. Finland is non the lone state with a high rate Guam has a prevalence of 70 in every 100, 00 this they believed was due to the toxins in the Cycad nut and the rhythm it went through to free it of the toxins. The nut was besides consumed by the Flying Fox (a chiropteran) which used to be portion of the diet. The toxins in the nut may hold been much more concentrated and as it was consumed it released the toxins. This statistic has been reduced as alterations in diet have occurred the people of Guam now have a prevalence of 7 in every 100, 000. (Sharon M. Valente RN, 2007) (Carmel Armon, n. d.)

Aetiology of ALS:

Between 5-10 % of instances are familial. 90-95 % of instances are sporadic. The mean oncoming for people with familial ALS is 10-20 old ages younger. The balance are sporadic. Diagnosis for this disease are blood trials, Electromyogram (EMG) , Magnetic Resonance Imaging (MRI) and one time every other neurological disease has been rejected so the diagnosing will be of MND so depending on clinical presentation a unequivocal diagnosing of what type of MND will go on next.

Pathophysiology of Amyotrophic lateral sclerosiss:

ALS is a disease of the Motor Neurones it is a chronic progressive neurodegenerative disease and it is fatal. There is no remedy for this disease and pharmacological medicine is limited to handling the symptoms as they occur. Famillia ALS is caused by a familial defect on chromosome 21 which is an enzyme (an enzyme allows a cell to transport out chemical reactions really rapidly) and helps destruct free groups (Free groups take negatrons from the cells and do a batch of harm) It is nevertheless non necessary to hold the faulty chromosome to develop ALS as non all bearers of the faulty chromesome contract the disease and people can contract the disease without a faulty chromesome. Sporadic ALS is mostly unknown in its cause nevertheless at that place have been links in the alterations to the organic structures nucleic acid through smoke have been shown to trip the disease oncoming. In ALS progressive musculus wasting predominately is found on the Lower motor neurones which reside in the anterior Grey horn of the spinal cord and in the encephalon root (corticospinal) . The upper Motor neurones predominately cause marking on the sidelong column of the spinal cord which may bring forth stiffness or abnormally active physiological reactions. There are instances although rare where the loss of prefrontal neurones may hold caused signifiers of cognitive damage. Although this is non typical in ALS as by and large there is no devolution of the five senses and these nervus cells remain integral. Nerve cell organic structures of the lower and upper motor neurone control the musculuss. The motor axons die by devolution and the larger motor neurones are affected to a higher extent than the smaller motor neurones. Equally long as the regeneration and devolution remains changeless so the nervus cell has the ability to maintain

the motor neurones within normal bounds hence no symptoms will be evident, merely when the demand for regeneration of these cells can non fit the devolution it is so the first symptoms of ALS becomes apparent. The axon interruptions and the environing Schwann cells catabolise the axon 's medulla sheath and steep the axon interrupting it into fragments. The breakdown causes ovoids which are little compartments which contain the fragments of the axon the ovoids are so ingested and destroyed by the macrophages the hungry white blood cells who come en mass to clean up. This nevertheless leaves a grade on the country and if supply and demand for regeneration are non met this procedure so causes the wasting of the motor nervousnesss go forthing them denervated and unable to execute. There are as with all types of disease clinical manifestations in which we are able to name certain conditions non all of them present at one time and all varying in badness depending on the length of clip the patient has had the disease. Muscle failing is the primary mark of ALS with the musculus cramp and stiffness associated with musculus wasting active nonvoluntary jerking a weakend clasp, slurred address, there is by and large no redness of the musculuss but the changeless vellication and contraction can do a batch of hurting. ALS begins in one limb before impacting the other limb. This disease can distribute through multiple sites on the organic structure and can impact the critical variety meats as they are made up of musculus tissue this is the terminal stages where the patient is unable to take a breath without the aid of a ventilator and as life anticipation for ALS is between 2-5 old ages it can be swift in oncoming. The pharmacological medicine for the patient was Riluzule in the early phases can decelerate down the devastation of the

motor nerve cells as it is a neuroprotector, Baclofen is prescribed as a musculus relaxer which is used to handle the cramps and stiffness caused by the musculuss, Dantrolene is prescribed to handle long term musculus cramps and stiffness though it can besides do liver harm, Diazepam (diazepam) is prescribed for its ataractic consequence and helps command the musculus cramp and stiffness these along with other general medicines and with the aid of psychological, Physiotherapy, Occupational therapy and emotional support signifier household, friends and other bureaus all assist the patient with get bying with their ALS. As ambulance service there is small that can be done to assist the patient apart from doing them comfy. We can utilize Professional-Professional calls to the necessary adviser and inquire for advice as to the best attention for the patient and if we need to set up for the patient to be admitted into infirmary or whether organizing another carepathway would be suited. The carepathway could besides be used and was in this instance a call to the patients ' ain GP bespeaking them to see as this was the want of the patient and the household explicating what had happened. The usage of any analgetic drug for the hurting would hold to be discussed with the adviser as this may hold inauspicious effects on status or may hold contraindicators to drugs regime the patient was already on. Following the Care program that is in topographic point meant that although the patient had died we could still follow their wants.

Pg1. APPENDIX A- The Nervous System

The nervous system is the organic structure 's communicating web. It plays a critical function in keeping homeostasis and consists of a immense figure of

cells called neurones. The nervous system can be split into two different divisions the Central Nervous System (CNS) which controls the encephalon and the spinal cord and the Peripheral Nervous System (PNS) which controls all the nervousnesss outside of the spinal cord and encephalon.

The peripheral nervous system can be sub-divided into the motor or motorial tract and the sensory or afferent tract.

The motor map is divided once more into- voluntary- controls motion of the musculuss and involuntary- which is portion of the autonomic nervous system that controls the cardiac musculus and its secretory organs, this system can be divided into two once more the sympathetic and parasympathetic. The nervous system is predominately made up of nervous tissue which consists of two types of cells- nervus cells or nerve cells and neuroglia, it besides includes the blood vass and the connective tissue.

Nerve cells

Nerve cells specialise in responding to physical or chemical stimulations from the alterations within their milieus. Nerve cells send and receive nervus urges. The construction of a nerve cell is like an arm with thenar and fingers. Dendrites are like your fingers projecting from the thenar of your manus they are little projections coming from the axon

APPENDIX B

INTRAMEDULLARY SPINAL CORD TUMORS

Pain and failing are the most common presenting symptoms of intramedullary spinal cord tumours.

Pain is frequently the earliest symptom, classically happening at dark when the patient is supine. The hurting is typically local over the degree of the tumour but may radiate.

Progressive failing may happen in the weaponries (cervical tumours) or legs (cervical, thoracic, conus tumours) . Impaired intestine, vesica, or sexual map frequently occurs early. Patients may hold hapless balance. Rarely, symptoms of subarachnoid bleeding may be present. Examination may uncover a combination of upper and lower motor nerve cell marks. Lower motor marks may be at the degree of the lesion and may help in localisation. Other marks apparent upon physical scrutiny may include spine tenderness, stiffening of pace, trophic alterations of appendage, centripetal loss, hyperreflexia, clonus, and scoliosis

cervical spondylosis - Cervical myelopathy

Cervical myelopathy occurs when terrible cervical spondylosis causes narrowing of the spinal canal (besides known as stricture) and compaction of the spinal cord.

When the spinal cord is compressed, it interferes with the signals that travel between your encephalon and the remainder of your organic structure. Symptoms can include:

a deficiency of co-ordination, for illustration you may happen undertakings such as buttoning a shirt progressively hard, heaviness or failing in your

weaponries or legs, jobs walking, less normally, intestine and urinary incontenance, centripetal loss is normally present but the upper limb failing and lower limb spasticity may be unusually similar to MND. MND has a more rapid myelopathy and cervical phonograph record bulge will be absent on X-ray. Occasionally, MND may co-exist with cervical spondylosis.

cervical rib - fasciculation absent, hurting prominent, centripetal loss normally present, characteristic radiology

peripheral nervus lesions - localised cachexia, normally accompanied by centripetal loss.

peroneal muscular wasting - centripetal loss of the peripheral nervousnesss become damaged it can do the undermentioned symptoms:

numbness and prickling in the pess and custodies

a combustion, knifing or hiting hurting

loss of co-ordination in the affected organic structure parts

musculus failing chronic polymyositis - The history of patients with polymyositis or dermatomyositis typically includes the followers:

Symmetrical, proximal musculus failing with insidious oncoming

Muscles normally painless Myalgias occur in fewer than 30 % of patients.

Dysphagia (30 %) and aspiration, if pharyngeal and oesophageal musculuss are involved

Arthralgias may be associated

Trouble kneeling, mounting or falling stepss, stepping onto a kerb, raising weaponries, raising objects, combing hair, and originating from a seated place

Weak cervix extensors cause trouble keeping the caput up

Engagement of pelvic girdle normally greater than upper organic structure failing

Cardiac engagement may do symptoms of pericarditis or myocardiopathy

Characteristic roseola of face, bole, and custodies seen in dermatomyositis merely

Patients with polymyositis normally present with symmetrical, proximal musculus failing in the upper and lower appendages. Weakness of cervix flexors besides occurs. Patients with polymyositis may describe musculus hurting and tenderness, which may be confused with symptoms of polymyalgia rheumatica. The disease may be for several months before the patient seeks medical advice, and all of the musculuss of the thighs, bole, shoulders, hips, and upper weaponries are normally involved. Muscle failing may fluctuate from hebdomad to hebdomad or from month to month.

Fine motor motions that depend on the strength of distal musculuss, such as buttoning a shirt, run uping, knitting, or authorship, are affected merely tardily in the disease.

Dysphagia secondary to oropharyngeal and oesophageal engagement occurs in approximately one tierce of patients with polymyositis and is a hapless predictive mark. Dysphonia is besides a hapless predictive mark but is much less common.

Ocular musculuss are ne'er involved in generalised polymyositis. However, stray orbital myositis, an inflammatory upset affecting the extraocular musculuss, is good described. Facial and bulbar musculus failing is highly rare in persons with polymyositis.

A household history of neuromuscular disease, endocrinopathy, or exposure to myotoxic drugs or toxins is absent.

differentiate by electromyography and musculus biopsy

myasthenia gravis - bulbar marks but seldom muscular cachexia ; responds quickly to anticholinesterase