Review

doi: 10.35366/102927

Anales Médicos
Vol. 66, No. 4
Oct. - Dec. 2021
p. 273 - 282

Malignant spinal cord compression and it's induced microvascular changes

Compresión maligna de la médula espinal y los cambios microvasculares inducidos

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How to cite: Shveid GD, Noguez RA, Moreno JS. Malignant spinal cord compression and it's induced microvascular changes. An Med ABC. 2021; 66 (4): 273-282. https://dx.doi.org/10.35366/102927

ABSTRACT

Spinal cord compression is considered a neuro-oncologic emergency. Any patient with back pain should receive a thorough evaluation and opportune treatment. Although the true incidence is unknown, *post mortem* analysis has shown that metastatic spinal cord compression may be present in 5-10% of patients with advanced cancer. Spinal cord compression may be the initial manifestation of cancer in up to 20% of patients with malignant neoplasms especially as a great number of them are metastatic in origin. The neoplasm which is most commonly associated with spinal cord compression is lung cancer (15% of cases), breast and prostate cancer in women and men, respectively. Vascular compression, specifically of the epidural venous plexus, may result as a secondary lesion, causing venous infarction and irreversible neurologic affection, as well as increased vessel permeability and subsequent edema, of which several essential pathological mechanisms and interactions take place. The goal of treatment is to preserve nerve function, reduce pain and symptoms associated to the compression and prevent disability. It is essential treatment must start within 24 hours of diagnosis and may range from steroids, to radiotherapy, surgery and palliative care. Overall, the prognosis of spinal cord compression from a malignant neoplasm is very poor,

RESUMEN

La compresión de la médula espinal es considerada una emergencia neurooncológica. Cualquier paciente oncológico con dolor en la columna debe recibir una evaluación completa y tratamiento oportuno. Aunque la incidencia específica es desconocida, análisis post mortem *han mostrado que puede existir una compresión medular metastásica en 5-10% de pacientes con cáncer avanzado. La compresión de la médula espinal puede ser la manifestación inicial de una neoplasia en hasta 20% de pacientes con neoplasias malignas, en especial aquellas que debutan de manera metastásica. La neoplasia más asociada a dicha compresión es el cáncer de pulmón en 15% de los casos y mama y próstata en mujeres y hombres, respectivamente. La compresión vascular, específicamente del plexo venoso epidural, puede resultar de una lesión secundaria, causando infartos venosos y afección neurológica irreversible, al igual que aumento en la permeación vascular y generación subsecuente de edema, los cuales cuentan con diferentes mecanismos e interacciones patológicas. El objetivo del tratamiento es preservar la función nerviosa, reducir el dolor y los síntomas asociados, al igual que prevenir la discapacidad. Es de suma importancia que el tratamiento inicie en las primeras 24 horas de que se haga el diagnóstico y puede variar desde esteroides a radioterapia, cirugía y tratamiento paliativo.*

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Received: 26/05/2021. Accepted: 11/11/2021.

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oscillating between three and six months survival post-diagnosis.

Keywords: Spinal cord compression, oncologic emergencies, microvascular changes.

Level of evidence: III

Por lo general, el pronóstico de una persona con compresión medular por una neoplasia maligna es pobre, oscilando entre tres y seis meses de supervivencia posterior al diagnóstico.

Palabras clave: *Compresión de la médula espinal, emergencia oncológica, cambios microvasculares.*

Nivel de evidencia: *III*

INTRODUCTION

Epidural spinal cord compression is considered a neuro-oncologic emergency. Any patient with back pain should receive a thorough evaluation and opportune treatment, should it be warranted. It is considered that approximately 5-14% of all patients with cancer will develop clinical evidence of spinal cord compression, typically neurologic dysfunction and paraplegia. Although the true incidence is unknown, post mortem analysis has shown that metastatic spinal cord compression may be present in 5-10% of patients with advanced cancer.[1] Of this 5%, epidural metastasis are distributed as the following: 70% occur in the thoracic spine, 20% in the lumbosacral spine, 10% in the cervical spine and between 10 to 38-40% manifest as a multifocal epidural tumor. Spinal cord compression may be the initial manifestation of cancer in up to 20% of patients with malignant neoplasms especially as a great number of them are metastatic in origin.

The neoplasm which is most commonly associated with spinal cord compression is lung cancer (15% of cases), although myeloma, lymphoma, prostate, breast and carcinoma of unknown primary site are considered to account for 10% of cases, respectively, the most common of the latter being breast and prostate cancer in women and men, respectively. Multiple myeloma may cause spinal cord compression by a variety of factors, including generation of a vertebral fracture, amyloidoma or plasmacytoma. Breast and lung cancer tend to involve the thoracic spine while tumors of the colon, pelvis and prostate are more akin to the lumbosacral segments.

There are several ways in which a neoplasm may induce spinal cord compression. The first and most common is when metastasis to a vertebral body extends onto the epidural space, which results in spinal cord compression. Collapse of a vertebral body may occur, causing bone and/or tumoral fragments to be displaced into the spinal cord. Another manner in which this may occur is in the case of neuroblastoma or lymphoma, where the tumor may grow into the spinal canal, through the neural foramina, destroying osseous tissue, resulting in compression. Vascular compression, specifically of the epidural venous plexus, may result as a secondary lesion, causing venous infarction and irreversible neurologic affection, as well as increased vessel permeability and subsequent edema. Finally, a rare cause of spinal cord compression is direct metastasis to the spinal cord.

ANATOMY AND PHYSIOLOGY

There are several types of vertebral column tumors, as well as those pertaining to the medullary canal, creating a division of intramedullary and extramedullary tumors, as well as tumors which are associated to a ductal space. Tumors which arise in the bones of the vertebral column are usually metastatic in origin, usually via a hematogenous dissemination. Examples of tumors which arise in the vertebral column are osteoid osteoma, osteoblastoma and giant cell tumor, osteogenic sarcoma, chordoma, chondrosarcoma and Ewing's sarcoma. Intradural, extramedullary tumors are located inside the dure but outside the spinal cord itself. Meningiomas, schwannomas and neurofibromas comprise the majority of the tumors which originate in this location. Intramedullary tumors which are located inside the spinal cord arise from glia. Astrocytomas and ependymomas account for the majority of these types of tumors, although hemangioblastomas may also occur.

On a tissular level, there are three main components to the blood-brain barrier: endothelial cells, astrocytes, and pericytes.[2] The astrocyte processes involve endothelial cells, which are surrounded by an extracellular matrix that may promote the expression of diverse cells.[3] Astrocytes induce specific characteristics in endothelial cells in relation to the blood-brain barrier.[4]

Glycoproteins such as neurotheline promote the induction of such characteristics, in addition to being involved in neuron and vascular differentiation.[5]

Pericytes regulate cerebral angiogenesis, the formation of tight junctions in endothelial cells, and the differentiation of the blood-brain barrier, as well as contribute to structural stability and microvasculature's dynamic capacity.[6] Tight junctions between cerebral endothelial cells make up a diffusion barrier, which prevents the majority of substances from the bloodstream to reach the brain. The majority of membrane's conduction is regulated by potassium, which in the brain is liberated into extracellular fluid to depolarize adjacent astrocyte processes.[7]

The macrovasculature of the spinal cord is an essential component of spinal cord structures, which, in case of lesion, could develop into medullar injury.[3,8] The main vessels involved are the anterior spinal artery and two posterior spinal arteries, as well as the radicular arteries and the pial plexus.[9-11] There is a close relationship between the metabolic requirements of nervous tissue and the distribution of blood vessels, in which specifically, the nervous tissue receives a sufficient amount of nutrients to fulfill its basic needs.[9] Microcirculation of the blood-brain barrier is controlled by an exchange of signals between neurons and astrocytes.[2] It is distributed in a uniform manner by the white matter of the spinal cord, although it varies in density with respect to grey matter.[12]

PATHOPHYSIOLOGY

There are two main mechanisms by which the spinal cord is injured. The first one relates to the traumatic event which the cord suffered, be it acute compression, impact, or laceration.[13] This mechanical disruption of capillaries in the spinal cord composes an immediate loss of vascular medullary support, which in turn initiates multiple molecular cascades that result in microvascular dysfunction, giving way to the second mechanism of lesion.[14,15] The vascular changes that compose the second mechanism of lesion contribute to the loss of neural elements, as there exists a high metabolic demand of grey matter, which promotes vasospasm, vasoconstriction, and ischemia. Ischemia results from vasogenic edema consequent to a rupture in the blood-brain barrier or blood spinal cord barrier, direct compression by adjacent tissue, vasospasm following mechanical trauma, or exposure to erythrocyte components, including endothelin and oxyhemoglobin.[16]

This condition causes failure of essential mechanisms such as glycolysis and oxidative phosphorylation, promoting energy loss and activating the process of necrosis. A cycle of ischemia, anoxia, acidosis, edema and compression is created which further perpetuates injury causing apoptosis and tissue necrosis.[17,18] Vascular events not only influence the evolution of secondary spinal cord injury but additionally define an environment which propagates neuroplasticity in the chronically injured spine.[16]

In a more specific manner, vascular, electrolytic, biochemical, and loss of energy metabolism may be seen as part of the secondary mechanism of injury.[13,19] With respect to vascular changes, a reduction in blood flow by thrombosis, loss of microcirculation, autoregulation, hemorrhage and systemic hypotension is observed, which may lead way to neurogenic shock. The generation of angiogenesis in response to this is mediated by sensors, transducers and mediators, which promote the synthesis and liberation of substances which influence cellular growth, death, or the migration of the components of the extracellular matrix.[20] In addition to PDGF, other mediators of vascular remodeling are neurotransmitters such as serotonin and norepinephrine, vasopressin, IGF 1, IL 1, IL8, IL6, and matrix metalloproteinases ADAM-8 and MMP-9.[10,15,21,22]

Proteolysis mediated by matrix metalloproteinases permit the invasion of endothelial cells to the surrounding matrix, activating and liberating growth factors which results in the increase of permeability of the blood-brain barrier and the blood spinal cord barrier by breaking down the basal lamina which surrounds blood vessels.[16] Concretely, ADAM-8 is a metalloproteinase that promotes the proliferation and migration of endothelial cells during angiogenesis, affecting in turn leukocyte migration, activating TNF, and VCAM-1.[21,23]

MMP-9 can limit functional recovery by inactivating a1-antitrypsin, thereby inhibiting leukocyte migration.[22] These mediators, specifically VEGF, contribute to the development of hyperpermeability and, ergo, edema after the traumatic event.[24] New vessels never acquire mature phenotypes, showing immature type junctions and the aforementioned hyperpermeability. The neovascularization of affected tissue additionally promotes a decrease in the potential transportation of glucose.[15]

Electrolyte changes include an increase of intracellular calcium, extracellular potassium, and permeability to sodium, which is propagated by mitochondrial lysis.[13,25,26] Upon alteration of

arterial PO_2 as a consequence of modification in cerebral blood flow, the product of the surface area is modified, giving way to redistribution of blood flow and an alteration in the magnitude of said flow.[6] Among the biochemical changes which take place, an accumulation of neurotransmitters, lipid peroxidation, free radical production, liberation of arachidonic acid, and production of prostaglandins may be observed.[13] The excitotoxicity which results from an excessive liberation of neurotransmitters and the increase in glutamate concentrations produces an overstimulation of ionotropic receptors such as NMDA and AMPA, which activate sodium dependent channels, altering the intracellular concentration of sodium, inducing intracellular lysis.[26] Lipid peroxidation mediated by oxygen free radicals is one of the most important factors in relation to the secondary mechanisms of injury which contribute to vascular damage by liberating vasoactive amines, glutamate and prostaglandins.[25] A polish study conducted in 2003 demonstrated a strong correlation between severity of SCI and the intensification of the lipid peroxidation process and plasma antioxidant potential decrease.[27]

Prostaglandins modulate alterations such as microvascular permeability, blood flow, edema and serotonin levels after SCI.[28] Other important factors in terms of blood-brain barrier and blood spinal cord barrier dysfunction are specific to neuroinflammation, particularly nitric oxide and endothelin 1, among others.[14] The inflammatory lesion, excitotoxicity, cytokines, and the damage provoked by free radicals induce an apoptotic cascade contributing to cellular loss, which significantly affects post-lesion neurological state.[26,29-32] These changes may be further divided into two phases: acute and chronic. The acute phase lasts three to five days after the initial spinal cord injury. In it, grey matter hemorrhage and white matter axonal and periaxonal inflammation as well as myelin disruption may be observed.[33]

At the beginning of this phase a fast inflammatory response with an elevated quantity of neutrophils and peripheral macrophages migrate to the injury site, endothelial inflammation and vacuolization can be observed.[26,34,35] Vascular hyperpermeability occurs in the first or second week after injury and relates to present tissue edema and reactive astrocytes or astrogliosis, which mediates the blood-brain barrier.[24] In relation to the structural alterations of microcirculation, an opening in the tight junctions of vascular endothelium, separation of the endothelium with recanalization, numerous plaquetary thrombi, and a disruption in vessel walls with cellular and plasmatic extravasation to the extracellular compartment can be noted.[17] The opening of tight junctions does not impede the migration of edematous fluid.[36]

Duration of the chronic stage oscillates between three and nine months. During this time, necrotic areas indicating ischemia, large cavitations and obstruction of small arteries and inter medullary veins are observed. Evidence of chronic injury may be determined by layers of collagen fibers organized in a perpendicular fashion in the perivascular compartments which surround endothelial cells.[37] Complications, such as the formation of cysts, occur in three phases: necrosis, repair, and stability.[25] In addition, a decrease in the density of vascular connectivity throughout the healing process after SCI may be observed.[38]

The capacity to regenerate neurological tissue in the mammalian central nervous system (CNS) is poor due to the presence of inhibitory factors and the lack of a supportive substrate, not because of the neurons lack of regenerative potential.[39] The capacity of the microglia to produce proteolytic enzymes and neurotoxins as well as pericytes' capacity, in pathological conditions characterized by edema and protein extravasation, to express enzymes such as g-glutamiltranspeptidase and glutamic acid, exemplify this.[37]

CLINICAL MANIFESTATIONS AND COMPLICATIONS

Spinal cord compression as a manifestation of disease progression commences generally as localized back pain, which is followed in turn by radicular symptoms and finally, myelopathy. The localized back pain may be acute or subacute, whereas radicular symptoms are typically an early symptom. Back pain is present in the vast majority of patients with spinal cord compression of oncologic origin (> 95%), most often localized in the upper back. Pain pertains to a biologic compromise of the spinal cord, whereas mobility becomes affected by compensatory mechanisms. Other elements of clinical importance include pain, which is exacerbated by movement, neck flexion, the Valsalva maneuver and is worse at night.

Radiculopathy may involve sensory or motor loss in involved dermatomes, or may be limited to pain. Thoracic malignancy is associated with bilateral radiculopathy, meanwhile cervical and lumbar disease are linked to unilateral radiculopathy.

Myelopathy has a rapid progression, as once paresthesia or numbness of the pelvic extremities occurs, complete myelopathy may develop within hours of onset including loss of bladder and bowel function. Weakness, as the initial clinical manifestation of myelopathy, is the second most common clinical symptom y spinal cord compression due to neoplasm, typically beginning in the lower extremities, culminating in paraplegia. Sensory symptoms such as Lhermitte's sign may also be present, as well as paraparesis, hyperreflexia, clonus, or even Horner's syndrome when there the sympathetic plexus is involved causing miosis, ptosis and anhidrosis. Myelopathy without back pain may be present in metastatic disease.

Important complications include massive hemorrhages, ischemic stroke, and deterioration of vision, which may lead to blindness.[7,17] The secondary mechanism of the lesion may provoke systemic effects such as hypotension and a reduced cardiac output.[13] To maintain tissue perfusion the most important variable is microvascular blood flow (MVBF).[40]

SCI implies serious disturbances in autonomic nervous system function, including imbalances on the cardiovascular and respiratory system as well as temperature regulation. In particular, pulmonary edema, hyponatremia, dysfunctions in motor behaviors such as locomotion, sexual response, defecation and micturition may be observed.[41-46] Paralytic ileus and consequentially, vomiting and aspiration may further impair respiratory function.[47] With respect to the cardiovascular system, spinal reflexes dominate cardiovascular control after SCI.[48]

DIAGNOSIS

In addition to the patients' history, which allows to establish an initial diagnosis of disease, it is necessary to corroborate the extension and severity of the lesion, structures involved, and the time of disease progression to be able to arrive at an accurate diagnosis and identify possible treatment.

The diagnosis of epidural metastasis must precede the onset of myelopathy, as the degree and rate of neurologic progression is directly proportional to the patient's prognosis.

Magnetic resonance imaging is considered the procedure of choice in terms of evaluation of spinal cord compression, tumor involvement of the vertebral body, para-vertebral area and leptomeninges. Not only does it evaluate the extent of osseous and nervous tissue involvement but it permits, as well, an accurate detection of other pathologies, which must be considered in the differential diagnosis of myelopathy. In addition, the entire vertebral column may be imaged, and contrast it not necessary to detect metastasis or spinal cord involvement. It holds a 93% sensitivity and a 97% specificity with respect to the pathology in question.

Computed tomography and myelography may be used if there exists contraindication to conduct magnetic resonance imaging, such as patients with pacemakers. Contrast agents must be administered to establish the full extent of the disease. Cerebrospinal fluid should be sent for cytologic examination. Conventional myelography is rarely utilized as may produce thrombocytopenia and coagulopathy. Bone scans identify metastatic disease in the spinal column and may evaluate the degree of tumor involvement. This study is contraindicated in patients where spinal cord compression is suspected. A thoracic radiograph is merely supportive of diagnosis, as in the case of blastic or lytic vertebral body lesions.

To determine the severity of the injury and evaluate neuroprotective studies, recent studies have shown that the use of Evans blue dye is quick, safe, simple, and of low cost, although this has only been shown in animal models.[25] In order to regulate microvascular endothelial cells at a molecular level trascriptonic screening may take place, in which spinal cord endothelial cells are separated and labeled with intravitallectin, thereby regulating vascular dysfunction.[49] To determine the presence of angiogenic response, immunopositive SM171/RECA-1 endothelial cells which colonize the basal lamina are identified.[50] Another way to observe the functional state of the neoformation of vessels and also identify a subpopulation of reactive microvascular elements is the administration of *Griffoniasimplicifolia isolectin* B4 rats.[51] Once the presence of angiogenesis has been identified, therapy that may counter its effects may be programmed.

Among the available tools in relation to diagnosis of SCI, intraoperative spinal angiography is also used as a surgical treatment in certain cerebro-vascular lesions. The microangiography and histopathology of SCI denote four injury zones. The first zone is characterized by necrosis of all elements involved, including neurons and capillaries. The second zone comprehends the intermediate stage of the injury, showing damage in axons and neurons. The third zone is a filling and scarring specific to the last phase of the traumatic lesion and myelopathy, where the arteries of gliomesenchymal scars are bent, thin, and

short. The fourth and last zone is a hypervascular area surrounded by areas of compression which is considered a reaction against gradual compression.

TREATMENT

The goal of treatment is to preserve nerve function, reduce pain and symptoms associated to the compression and prevent disability. It is essential treatment must start within 24 hours of diagnosis. Depending on symptoms, ranging from mild paresthesia to paralysis, MRI is indispensable to denote the area of compromise.

Corticosteroids have long been considered the medication of choice when dealing with spinal cord compression, alleviating pain and controlling neurologic symptoms. It must be started in a timely fashion in order to prevent permanent neurologic injury. Vasogenic spinal cord edema is considered a key target. Dexamethasone, should be administered as soon as possible, excluding cases of lymphoma where the administration of steroids may mask presence of the disease on magnetic resonance imaging as well as induce tumor regression. According to Dr. Lisa M. DeAngelis, professor of neurology at Weill Medical College of Cornell University, for radiculopathy, the suggested dose is 16 mg IV followed by 8-12 mg IV every 12 hours. In case of myelopathy, Dr. DeAngelis suggests 100 mg iv followed by 24 mg iv every six hours.

Radiotherapy has also been considered amongst the most important initial treatments for spinal cord compression, alleviating pain and retarding tumor growth. External beam radiation therapy is the therapy most widely utilized. A treatment dose of 30Gys is delivered through a 10 day span, where the patient receives 3 Gy daily. Fractionated radiotherapy may be considered the treatment of choice in patients with epidural tumors without neurological impairment, spinal instability or pain. Although preoperative radiotherapy is not suggested, postoperative fractionated radiotherapy should be offered routinely. If patients are not suitable for spinal surgery urgent radiotherapy may be indicated unless there is paraplegia for more than 24 hours, pain had been well controlled or overall prognosis is considered to be too poor.[1] In contemporary guidelines, a combination of radiosurgery and chemotherapy may be conducted. Therapy choice depends upon the grade of edema the patient had and the effect and disability it produced in the patient, the time elapsed between the beginning of the symptoms and the possibility surgically producing anatomical changes which will relieve spine disfunction.

Spinal decompression surgery is important to relieve symptoms such as pain, numbness, tingling, weakness and unsteadiness. Severe cases where bladder of bowel disfunction has ensued are considered to be oncologic emergencies in which treatment is needed to halt progression. Surgery may be used in certain cases in addition to radiotherapy, such as melanoma, sarcoma and renal cell carcinoma. Examples of surgical approaches may include resection of the vertebral body, laminectomy. A surgical approach is especially useful when there is need for a pathologic diagnosis, as in the case of an unknown primary tumor, progression of neurological symptoms during radiotherapy, recurrence of spinal cord compression after receiving radiotherapy in that area, and spinal instability. Patients with hyper vascular metastases from the aforementioned renal cell or thyroid carcinoma mat require preoperative arterial embolization in order to prevent intraoperative hemmorage.[7] Aside from achieving spinal cord decompression, it is also imperative to maintain durable spinal column stability.[1]

Chemotherapy is used to treat primary tumors, as in case of breast, lung or prostate cancer. In rare cases such as germ cell tumors or lymphoma, considered highly responsive to chemotherapy is it given.

In the long term, the principal mechanisms in terms of recovery of SCI are the regeneration and repair of neurons and the reorganization of neurological circuits.[52] The incapacity to develop neurological tissue in the CNS has been attributed to a reduced growth capacity and an environment that does not permit axonal elongation, specifically because of inhibitory molecules associated to myelin, such as Nogo and MAG. Chemorepulsive effectors, such as semaphorins, colapsin, and Netrin, and a glial scar at the injury.[53] Several pharmacologic therapies which attempt to diminish or neutralize these effects exist, although unfortunately there is no therapy which improves neurological results in a satisfactory fashion.[26] Microvascular changes in SCI play an essential role in the disease pathophysiology, such that treatments whose objective to diminish them could improve the final neurological result considerably.

Within the microvasculature changes previously discussed, the ischemic process and the associated vasoconstriction, alterations in blood flow and angiogenesis are the therapeutic targets that currently have the greatest emphasis. Most

experimental studies of this nature are done on animals, thus we have yet to be able to reap the benefit of these therapies on humans. Pharmacologic blockade of endothelin 1 mediated vasoconstriction has been shown to attenuate the decomposition of the blood spinal cord barrier after SCI.[14] In terms of angiogenic regulation, VEGF has been found to promote a better functional therapy. The importance of these therapies, argues J. Widenfalk in *Neuroscience*, is «changing the balance from angiogenesis to angioprotection».[54] To obtain optimal results, therapies directed to the angiogenic processes between the third and seventh day after the initial injury are suggested.[50]

In a study published in 2008 in the American Journal of Neuroradiology, 18 patients with vertebral arterial lesions received endovascular therapy, resulted in clinical improvement of all patients involved and immediate lesion total occlusion in 89% of thus population, promoting endovascular techniques as a safe and efficient treatment.[8] Among the pharmacologic therapies which may improve and stabilize spinal cord blood flow and function are nimodipine, volume expansion by dextran, and glutathione monoethyl ester.[13] Glutathione monoethyl ester also inactivates nitric oxide, diminishing vasodilatation.[55]

To counter effects within the inflammatory process and promote neuroprotection, cyclooxygenase inhibitors, such as indomethacin, have been greatly utilized. With respect to the vasculature of the spinal cord, indomethacin improves blood flow as well as inhibits the synthesis of prostaglandins, reduces edema, and causes changes in permeability.[28,56-62] Other compounds which have shown improvement in relation to the inflammatory process are antioxidants, calpain inhibitors, apoptosis inhibitors, steroid hormones, sodium cannel blockers, NMDA and AMPA-kinate receptor antagonists can be used.[46,63-66] Antioxidants such as glucocorticoids have been extensively used in this disease by notably diminishing inflammation and ischemia. Steroid hormones such as progesterone and estrogen have been used to improve vascular endothelial function, increase blood flow to the brain, as well as maintaining calcium homeostasis, blocking the activation of amino acids and modifying both cellular and humoral immune responses.[67-73]

There are studies being conducted throughout the globe, either pharmacological, physical or surgical to be able to attenuate the devastating effects of SCI. Systemic hypothermia, induced to 30-34 ºC has been shown to reduce apoptosis, myeloperoxidase activity, vasogenic edema, and tissue damage in addition to improve hind limb function. By retarding tissue damage and reducing neurological deficits, it has also been noted to improve functional recovery and reduce overall structural damage.[27,74-77] Among the surgical advances relating to the amelioration of the neurological effects post SCI, recent Turkish studies have demonstrated that splenectomy prevents neuronal loss after SCI injury.[78]

PROGNOSIS

If treatment is initiated before the start of spinal cord compression symptoms, the prognosis tends to be more favorable. Patients who receive treatment after the onset of neuromuscular oncologic complications i.e. patients who are not able to walk, or patients with primary tumors which are refractory to treatment, receive a poorer prognosis, associated with a non-favorable progression free survival.

Overall, the prognosis of spinal cord compression from a malignant neoplasm is very poor, oscillating between three and six months survival post-diagnosis. Patients who receive both chemo and radiotherapy have been documented to survive longer. Patients who receive radiotherapy as well as surgery have demonstrated to have a better overall survival in comparison to those who receive radiotherapy alone, as well as fewer complications in relation to paraplegia such as venous thromboembolisms or sepsis.

CONCLUSION

SCI is a disease that causes severe neurologic incapacity.[1] Current concepts have permitted the development of specific therapies which reduce the damage caused and promote functional recovery, although these are mostly experimental and mainly done on animals. In relation to microvascular changes, ischemic process and the associated vasoconstriction, alterations in blood flow and angiogenesis are the therapeutic targets that currently have the greatest emphasis.

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