Cancer-related medical emergencies

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Pulmonary tumor embolism

The lungs are a prominent target for the embolization of any material larger than ~10 μm that gains access to the venous circulation. This includes thrombi, air, amniotic fluid, fat, injected foreign material, and tumor. It is important that the correct type of pulmonary emboli be identified, as treatment and prognosis vary considerably.

Pathophysiology

Tumor cells gain access to the circulatory system through the invasion of small veins, or the release of tumor fragments into the tumor's neovasculature. Most of the circulating tumor cells are then destroyed by circulatory mechanical forces, shear forces, or the host's immune system. However, some tumor cells reach the lungs and become trapped within pulmonary capillaries.

Tumor cells trapped within pulmonary capillaries may trigger the coagulation cascade and obstruct the pulmonary vessels. The obstruction is primarily due to both the tumor cells and the associated clot, but reactive concentric medial hypertrophy and intimal fibrosis of the pulmonary vessels may also contribute. Such secondary changes may progress to complete and irreversible obstruction of pulmonary vessels, whereas occlusion by pure thrombi or thromboemboli is generally followed by recanalization. When the pulmonary vasculature becomes obstructed, ventilation–perfusion matching becomes impaired and pulmonary vascular resistance increases, which may impair gas exchange and cause Cor pulmonale.

Intraluminal tumor emboli do not proliferate or spread locally; thus, they should not be considered as metastases. Lung metastasis requires malignant cells to adhere to lung-specific adhesion molecules on the endothelium, produce enzymes that degrade the basement membrane and lung extracellular matrix, and grow in association with lung-specific growth factors. It has been proposed that tumor emboli may contribute to lymphangitic carcinomatosis; however, this is uncertain, as the two entities are not universally found together and their clinical manifestations are different.

Acute tumor lysis syndrome

What it is

Acute tumor lysis syndrome is a severe metabolic derangement caused by the release of intracellular contents from malignant cells rapidly dying after recent cancer treatment.

When to consider it

Acute tumor lysis syndrome should be considered in cases of high-grade non-Hodgkin lymphoma and acute lymphocytic leukemia after any form of cancer treatment. Spontaneous tumor lysis syndrome (in the absence of recent cancer treatment) can occur in aggressive lymphomas and leukemias (Burkitt lymphoma, acute lymphocytic leukemia, large T-cell lymphoma). TLS is uncommon in solid tumors but can occur in testicular cancer, small-cell lung cancer, neuroblastoma, and breast cancer. Renal failure is a predisposing factor. Any type of cancer treatment can precipitate tumor lysis syndrome, including glucocorticoids, radiation therapy, tamoxifen, and systemic chemotherapy.

What to watch out for

Hyperkalemia, hyperphosphatemia, hypocalcemia (caused by binding with excess phosphate), and hyperuricemia. Hyperkalemia may cause cardiac arrhythmias; excess phosphate and uric acid may cause or worsen renal failure. Acute kidney injury during acute tumor lysis syndrome is an independent and an important marker for mortality risk.

Acute tumor lysis syndrome: what to do

Give intravenous volume

Upon the first suspicion for acute tumor lysis syndrome, authors recommend aggressive intravenous fluids at twice the expected maintenance rate.

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Do not alkalinize urine

Authors do not recommend alkalinization of the urine (to increase uric acid solubility), because it may precipitate calcium phosphate and xanthine crystals, which can worsen nephropathy.

Hyperkalemia

Hyperkalemia can be treated with kayexalate, insulin/dextrose, sodium bicarbonate (cautiously to avoid inadvertent hyperkalemia, metabolic alkalosis, or the creation of calcium crystals), or calcium gluconate.

Hyperphosphatemia

Hyperphosphatemia can be treated by flushing with intravenous fluids and giving phosphate binders as needed. Renal replacement therapy may be considered in severe cases or when medical management fails.

Hypocalcemia

Authors suggest that hypocalcemia should not be treated unless symptoms are present, so as to avoid the risk of nephropathy.

Hyperuricemia

Rasburicase (recombinant urate oxidase) catabolizes uric acid and rapidly normalizes uric acid levels; authors recommend rasburicase as a first-line therapy for patients with tumors prone to rapid lysis, or those who have acute tumor lysis syndrome with pre-existing renal failure and high uric acid levels. Rasburicase is contraindicated in patients with G6PD deficiency, and it can cause hemolytic anemia or methemoglobinemia. Rasburicase is currently recommended on faith and its plausible mechanism; no randomized trial has shown that it prevents renal failure or death. Allopurinol is used prophylactically before chemotherapy, but is not recommended for acute tumor lysis syndrome.

Acute kidney injury

Renal replacement therapy (dialysis) is recommended for patients with acute tumor lysis syndrome and significant acute kidney injury, who are not responding well to medical management, or who have potentially life-threatening electrolyte disturbances. No one knows whether hemodialysis or continuous renal replacement is a superior method.

Superior vena cava syndrome

What it is

Superior vena cava (SVC) syndrome is obstruction of flow through the SVC into the right atrium; hence its other name, SVC obstruction syndrome.

What causes superior vena cava syndrome (Who gets it?)

SVC syndrome is caused by compression or invasion by mediastinal masses (tumors and/or lymphadenopathy), stenosis of the SVC, or thrombosis.

In the preantibiotic era, infections (tuberculosis) were the most common cause of SVC syndrome. By the 1980s, malignancy accounted for 90% of the cases of SVC syndrome. With the increase in the placement of catheters in the large vessels over recent decades, line-related thrombosis, vessel stenosis, and other benign causes such as fibrosing mediastinitis now account for an estimated 20–40% of cases of SVC syndrome.

Infection-related superior vena cava syndrome

Tuberculosis, histoplasmosis, and the infectious sequela fibrosing mediastinitis are uncommon causes of SVC syndrome in the USA, but still occur.

Malignancy-related superior vena cava syndrome

Lung cancer and non-Hodgkin lymphoma together cause about 95% of cancer-related SVC syndrome. About 2–4% each of patients with lung cancer or non-Hodgkin lymphoma develop SVC syndrome during their illness. Puzzlingly, Hodgkin lymphoma rarely causes SVC syndrome despite it often affecting the mediastinal lymph nodes.

Thrombosis-related superior vena cava syndrome

Placement of intravenous catheters in the large vessels is believed to be causing an increasing proportion of cases of SVC syndrome, although precise numbers are hard to come by because the absolute risk for any given catheter placement is extremely low (e.g. hundreds of thousands of central venous lines are placed in the USA each year). Many cases of line-related thrombosis causing SVC occur in hypercoagulable cancer patients.

Collateral vessels can dilate and proliferate in response to a slow obstruction of flow through the SVC, resulting in a compensated state for weeks before symptoms develop.

What to look out for in suspected superior vena cava syndrome

Facial edema is the most common symptom of SVC syndrome. This can be subtle; a patient may describe feeling bloated or 'puffy'. Venous distension in the neck may be present on examination.

In patients with malignancy, dyspnea, cough, chest and shoulder pain, and hoarseness are more commonly noted compared with 'benign' compression. Dyspnea may be worse when leaning forward or lying down. Arm swelling is another common symptom of SVC syndrome.

What to do for superior vena cava syndrome

Although SVC syndrome has been traditionally regarded as a medical emergency, authors imply it should perhaps be reclassified most often as an urgency, with time for deliberation and testing to guide thoughtful decision-making. Before rushing the patient with 'typical' SVC syndrome to radiation therapy, consider that the resulting tissue necrosis could make a biopsy uninterpretable and seriously compromise the diagnosis and management of cancer, if present. (This assumes that frank airway obstruction or cerebral edema are not present — two situations in which SVC syndrome is a true emergency and immediate radiation therapy and/or stent placement are essential.)

Diagnosing the underlying cause of SVC syndrome is essential, whenever possible, because many causes of SVC syndrome are benign. A contrast-enhanced computed tomography (CT) scan of the chest is the most useful imaging study. Authors advise the careful use of selected invasive diagnostic procedures such as cytology of sputum and/or bronchial washings, thoracentesis for pleural effusions, and needle biopsy of any palpable lymph nodes. They do not mention bone marrow biopsy, but some would say suspicion for lymphoma should make this a consideration. Standard physician thinking has been that intrathoracic or transthoracic needle biopsies in patients with SVC syndrome generally carry an unacceptable risk for life-threatening bleeding, but each patient's situation should be considered individually.

Specific interventions employed for superior vena cava syndrome include

Endovascular stents

Self-expandable stents are highly effective at relieving the symptoms of SVC syndrome. Over 90% of patients report symptom relief after stent placement, and the technical success rate of placement is greater than 95%. Multiple stents may need to be placed in series (so-called 'kissing stents'). Stents may even be placed in a SVC that is totally occluded or contains thrombus; catheter-directed thrombolysis, balloon angioplasty, or mechanical thrombectomy may be required before stent placement.

SVC stents have the advantage of not requiring a diagnosis before implementation. Complication rates of placement are 3–7%, but potentially catastrophic (infection, pulmonary embolus, stent migration, bleeding, or perforation of the SVC). SVC stents should therefore be reserved for patients who require urgent or emergent intervention due to symptoms. Most other patients can be managed more conservatively – for example, with chemotherapy and/or radiation, if malignancy is the cause of SVC syndrome.

Short-term anticoagulation is provided at many centers after SVC stent placement. Whether long-term anticoagulation is needed or helpful after stent placement not due to SVC thrombus is unknown. Some authors have recommended warfarin for months, whereas others suggest antiplatelet therapy alone.

Chemotherapy

Initial chemotherapy is the treatment of choice for SVC syndrome because of small-cell lung cancer, non-Hodgkin lymphoma, or germ cell cancers. Radiation therapy is not used alone in these patients and could possibly be counterproductive.

Patients with non-small-cell lung cancer with SVC syndrome do not respond well to chemotherapy and are usually managed with radiation therapy and/or endovascular stent.

Radiation therapy

Most patients with SVC syndrome due to cancer have radiation sensitive tumors. Radiation therapy can result in rapid improvement in patients with SVC syndrome due to lung cancer, in less than 72 h usually. Some patients may require weeks to respond to radiation, and about 20% of patients with SVC syndrome do not achieve symptomatic benefit from radiation therapy.

Catheter-directed thrombolytic therapy

Catheter-directed thrombolytic therapy has been used on a case-by-case basis to treat obstruction of the SVC due to thrombus. Anecdotal reports suggest effectiveness, but the true benefits and risks in SVC syndrome due to thrombus are unknown.

Glucocorticoids

Glucocorticoids may be helpful in patients with lymphoma or thymoma or other steroid responsive malignancies. Steroids are not recommended for use in patients with SVC syndrome due to lung cancer or other causes. Corticosteroids are commonly used prophylactically to reduce swelling during radiation therapy, but this is a separate indication (and without evidence of benefit).

Diuretics

There is no evidence to suggest that diuretics are beneficial in treating SVC syndrome, although they are commonly used.

Cancer-associated hypercalcemia

Hypercalcemia has been reported to occur in 10–20% of patients with malignancies. Malignancy is one of the most common causes of hypercalcemia. Certain cancers are more likely to cause hypercalcemia compared with

others. The most common cancers that are associated with the development of hypercalcemia are squamous cell lung cancer, squamous cell head and neck cancers, breasts cancer, multiple myeloma, T-cell lymphomas, renal cell cancer, and ovarian cancer.

Hypercalcemia may be associated with bone metastases in patients with solid tumors (e.g. metastatic breast cancer). Increased bone resorption by osteoclasts leads to hypercalcemia. The increased bone resorption in this setting may be mediated by prostaglandins or other factors. In addition, tumor cells may be able to resorb bone directly.

Hypercalcemia is also associated with hematologic malignancies such as multiple myeloma or T-cell lymphomas. Again, hypercalcemia in these cases results from increased bone resorption by osteoclasts, mediated by lymphokines.

In recent years, hypercalcemia associated with solid tumors without bone metastases (e.g. squamous cell lung or head and neck cancers) has been studied most widely. Hypercalcemia in these cases is due to a systemic humoral factor(s) that is produced by the tumor, the so-called humoral hypercalcemia of malignancy. Recent investigations have identified parathyroid hormone-related protein as a probable mediator of humoral hypercalcemia of malignancy. Parathyroid hormone-related protein may act in conjunction with other factors (e.g. transforming growth factor α, tumor necrosis factor, interleukin-1) to cause the effects seen in humoral hypercalcemia.

The proximal cause of hypercalcemia in all of these situations is increased bone resorption. However, the kidneys help to maintain calcium homeostasis by increasing urinary calcium excretion when bone resorption increases. Changes in renal handling of calcium, then, are important in precipitating hypercalcemia in patients with increased bone resorption. Normal calcium reabsorption in the proximal renal tubule is linked with sodium reabsorption and with volume status. Hypercalcemia is associated with a decreased effect of ADH on the renal tubules, leading to dehydration. Dehydration leads to a decrease in GFR, increasing sodium and thus calcium reabsorption and worsening the hypercalcemia. Other factors such as vomiting may also contribute to precipitating or maintaining hypercalcemia.

Normal total serum calcium is about 8.5–10.5 mg/dl. About 40% is bound to proteins, mainly albumin. Fifteen percent is complexed to anions, and 45% is the free, ionized, active form. Formulae are available for correcting calcium concentrations for changes in albumin. These are supposed to estimate ionized, active calcium, although the correlation with measured ionized calcium is questionable. However, ionized calcium is usually not measured, and the formulae, based on albumin, are frequently used [e.g. corrected serum calcium = {(4–albumin) ×0.8}+measured serum calcium].

Clinical manifestations of hypercalcemia include the following: GI-related manifestations: nausea, vomiting, constipation; neurologic manifestations: weakness, lethargy, confusion, coma; and renal manifestations: polyuria, thirst. Symptoms may depend on the rate of rise of calcium — for example, slow, gradual increases may be less symptomatic compared with abrupt increases. Progressive hypercalcemia can lead to death. Prompt treatment is initiated in patients who are symptomatic and/or whose calcium is very high (e.g. ≥13 mg/dl). Other patients are treated, but perhaps less urgently. Remember, also, that certain drugs can contribute to hypercalcemia (e.g. thiazides, lithium).

Treatment

The first line of treatment for cancer-associated hypercalcemia is hydration with saline. Hydration repletes volume and increases calcium excretion. Promotion of sodium diuresis leads to calciuresis as noted above. Hydration over 2 days (2–8 l/day, depending on hydration status, of 0.9% NaCl) can decrease serum calcium by approximately 2 mg/dl. Note that unless other treatment is initiated, or the underlying malignancy treated, calcium will rise again.

Furosemide may be used to prevent fluid overload from hydration (e.g. in patients with CHF). Furosemide also has a calciuric effect and has been suggested as a treatment in addition to, or following, hydration. Reports of successful lowering of calcium with furosemide involved very high doses along with large volumes of fluid, strict electrolyte monitoring and replacement, and intensive care monitoring. Outside of this setting, dehydration from furosemide can offset any calciuric benefit, and its use should probably be reserved for patients who cannot tolerate hydration, as noted above.

Bisphosphonates: today, bisphosphonates are probably the most frequently used calcium-lowering agents. They (etidronate, pamidronate) prevent osteoclastic bone resorption, and they may directly inhibit osteoclasts. Etidronate can also impair bone formation. For prompt response, bisphosphonates are given intravenously. Pamidronate is more potent compared with etidronate, and is probably used more often, although its clear superiority is not well documented. The recommended dose of pamidronate is 60–90 mg, intravenous. With

either bisphosphonate, calcium decreases in about 48 h and may fall to normal over the next 2–3 days. Although commonly recommended for administration over 24 h, pamidronate has been safely administered by short infusion (e.g. 0.5–3 h), making it attractive for outpatient use. Bisphosphonates are relatively free of side effects. There are reports of elevations of serum creatinine with etidronate in large doses. The clinical importance of mild reversible elevations is not clear. Because of the reports, it is recommended to avoid the use of etidronate in patients with serum creatinine greater than 5, and to decrease the dose when creatinine is 2.5 or greater. Clinically, however, bisphosphonates may be safe to use even before patients are completely rehydrated (e.g. while their creatinine may still be elevated).

Oral etidronate has been recommended for maintenance of normocalcemia. Its efficacy is less well established than that of parenteral bisphosphonates. Doses up to 20 mg/kg/day have been used. Although inhibition of bone formation is a potential concern, many patients with cancer-associated hypercalcemia have limited survival, and will be unlikely to suffer long-term consequences.

Plicamycin (mithramycin) is commonly used to treat cancer-associated hypercalcemia after hydration. The dose is 25 mcg/kg and should be reduced in patients with renal dysfunction (e.g. by 50%). Plicamycin may be given as an intravenous bolus or infusion. Most side effects of plicamycin are associated with higher or repeated doses (e.g. 25–50 mcg/kg/day´5). These side effects include thrombocytopenia, coagulopathy, and hepatitis. The 25 mcg/kg dose given once and perhaps repeated in 48–72 h if necessary is well tolerated. Although calcium is lowered to normal levels in majority of patients, the duration of normocalcemia is variable. When plicamycin provides normocalcemia for 7 days or more, it can also be a useful agent for maintaining lowered calcium.

Gallium nitrate is given as a continuous intravenous infusion (200 mg/m^2/day´5 days). It appears to be effective in a large proportion of patients. In a comparative study with etidronate, the median duration of normocalcemia was 8 days. Overall, gallium was reported to be more effective compared with etidronate. Gallium should be used after rehydration. Higher doses have been associated with nephrotoxicity, and it is recommended that gallium not be used in patients with serum creatinine greater than 2.5. Its relative place in therapy is yet to be defined, keeping in mind factors such as cost, duration of effect and duration of treatment, potential for nephrotoxicity, etc.

Calcitonin is also used to lower serum calcium. It works within several hours and may be useful in lowering calcium acutely. Calcitonin may also be used in patients with renal insufficiency or before rehydration. Resistance develops quickly to calcitonin, and although some investigators suggest that steroids prolong the effectiveness, others have not found that to be the case. Calcitonin requires frequent parenteral administration (intravenous or SQ). These latter factors make calcitonin a less than optimal choice for maintenance of normocalcemia.

Finally, cisplatin has been used to treat hypercalcemia associated with certain malignancies. It may provide several weeks of normocalcemia. However, it should not be used in patients with renal insufficiency. Cisplatin should only be given to patients who have been well hydrated.

Maintenance of normocalcemia may be achieved with intermittent administration of some of the agents described above to lower calcium initially (e.g. plicamycin, pamidronate). Maintenance with oral etidronate was discussed above. Oral phosphate is also used to maintain normocalcemia, provided patients are not hyperphosphatemic. Neutral phosphate capsules should be emptied and mixed with liquid. The starting dose of phosphate is about 1 g/day in divided doses. Diarrhea frequently impairs dose escalation and/or compliance, thus minimizing effectiveness.

Malignant pericardial effusions

Introduction

Malignant pericardial effusions (MPEs) are a rare complication of advanced cancer, but are associated with high morbidity and mortality. This fast fact discusses the diagnosis and management of MPEs.

Epidemiology and prognosis

Approximately 10% of patients with cancer develop cardiac metastases, with ~75% of these affecting the epicardium. Only a third of these, however, will develop clinically significant MPEs. Lung and breast cancers are the most common causes. MPEs are associated with a poor prognosis. Studies suggest a median survival of 2–3 months after a MPE is diagnosed, with a mean survival of 5 months for solid tumors and 20 months for hematologic malignancies.

Physiology and symptoms

The pericardial space is normally filled with less than 50 ml of serous fluid. As this volume increases due to epicardial or pericardial metastases or lymphatic obstruction, both right and left ventricular failure can occur due to inadequate filling. Signs and symptoms include peripheral and pulmonary edema, chest

discomfort, cough, shortness of breath, and orthopnea. Severity of symptoms depends on the volume of the MPE as well as the rapidity of its accumulation; severe cases can present with cardiac tamponade and shock. An echocardiogram is indicated whenever an MPE is suspected. Not only does it confirm the presence of an effusion but also its findings can dictate whether or not urgent treatment is indicated (e.g. if signs of tamponade are evident). A diagnostic pericardiocentesis or pericardial biopsy is sometimes needed to confirm the cause of the effusion.

Treatment options

(1) *Systemic chemotherapy or radiotherapy* are effective for chemosensitive or radiosensitive tumors such as previously untreated breast cancer and many lymphomas. Reaccumulation rates for both modalities are about one-third overall, depending on the patient's overall course and response to therapy.
(2) *Pericardiocentesis* results in immediate symptom relief in most patients; however, the effusion may rapidly reaccumulate in many patients, requiring repeat pericardiocentesis (within 1–2 weeks in some series).
(3) *Pericardial sclerosis* involves instilling a sclerosing agent with the intention of scarring the pericardium to the epicardium, preventing reaccumulation of the MPE. Multiple agents have been studied, including doxycycline, minocycline, and bleomycin. Success rates (no reaccumulation at 30 days) are about 70–90%. Longer term success rates have not been defined because of the poor survival of study patients. The major side effects are chest pain (50–70%), cardiac arrhythmias, and fever. In head-to-head comparisons with doxycycline, bleomycin has been shown to have fewer side effects and to lead to shorter hospitalizations.
(4) *Surgical decompression* therapies range from less invasive (balloon pericardiotomy, subxiphoid or thoracoscopic pericardiectomy) to more extensive (open thoracotomy with pericardial stripping). A pericardial 'window' (which allows ongoing drainage of fluid externally or internally such as into the pleural cavity) is often created. Case series have suggested that reaccumulation rates with surgical therapies are low (<15% up to 10 months out).

Decision-making

The treatment of MPEs depends on how urgently treatment is needed, the likelihood of the tumor responding to antineoplastic treatments, and the anticipated survival of the patient. A multidisciplinary approach to decision-making, involving input from medical and radiation oncology, cardiology, and thoracic surgery is recommended. Simple pericardiocentesis may be appropriate for patients with short prognoses (<1 month), particularly if their MPE is not expected to reaccumulate in their remaining life-span. A symptomatic patient with no signs of tamponade and a chemotherapy-sensitive tumor such as untreated breast cancer may receive a durable response from a pericardiocentesis for symptom relief, followed by chemotherapy. Patients with longer prognoses (>1 month) who are expected to reaccumulate their MPEs will likely benefit most from sclerosis or surgical decompression; there is no clear evidence currently suggesting that one strategy is superior to the other. Symptom-directed care without specific intervention for the MPE is an appropriate option for patients with very short prognoses and for those who decline more invasive treatments.

Malignant spinal cord compression

Malignant spinal cord compression (MSCC) is an uncommon condition that affects people with certain cancers that have spread to the bones in the spine, or have started in the spine.

MSCC happens when cancer cells grow in, or near to, the spine and press on the spinal cord and nerves. This causes swelling and a reduction in the blood supply to the spinal cord and nerve roots. The symptoms of spinal cord compression are caused by the increasing pressure (compression) on the spinal cord and nerves.

Any type of cancer can spread to the bones of the spine (vertebrae), which may lead to spinal cord compression. It is more common in certain cancers including breast, lung, or prostate and in individuals who have lymphoma or myeloma.

Symptoms

(1) Back or neck pain: the first symptom is usually any unexplained back or neck pain, which may be mild to begin with but becomes severe. The pain may feel like a 'band' around the chest or abdomen and can radiate over the lower back, into the buttocks, or legs. The pain can also spread down the arms. Quite often this pain is worse when lying down and it may affect sleeping.
(2) Numbness or pins and needles in toes and fingers, or over the buttocks.
(3) A new feeling of being unsteady on your feet, having difficulty climbing stairs or walking, or your legs giving way.
(4) Difficulty controlling your bladder, passing very little urine, or passing none at all.
(5) Constipation or problems controlling your bowels.

These symptoms can also be caused by a number of other conditions. It is very important to let the doctor know presence of any of these symptoms so that they can be investigated.

The earlier MSCC is diagnosed, the better the chances are of treatment being effective.

Diagnosis

Magnetic resonance imaging scan

This scan uses magnetism instead of X-rays to build up a detailed picture of areas of the body. The scanner is a powerful magnet, so individuals may be asked to complete and sign a checklist to make sure it is safe for them.

Before undergoing the scan, individuals are asked to remove any metal belongings including jewellery. Some people are given an injection of dye into a vein in the arm. This is called a contrast medium and can help the images from the scan to show up more clearly. During the test, individuals are asked to lie very still on a couch inside a long cylinder (tube) for about 30 min. Some people feel a bit claustrophobic during the scan. Earplugs or headphones are given as it is noisy.

Computed tomography scan

A CT scan takes a series of X-rays, which build up a three-dimensional picture of the inside of the body. The scan takes 10–30 min. CT scans use small amounts of radiation, which is unlikely to harm individuals undergoing the scan or anyone they come into contact with.

They may be asked not to eat or drink for at least 4 h before the scan.

They may be given a drink or injection of a dye, which allows particular areas to be seen more clearly. For a few minutes, this may make them feel hot all over. It is important to let the doctor know allergies to iodine or presence of asthma, because individuals with such conditions could have a more serious reaction to the injection.

Having an MRI or CT scan is painless; however, individuals may find lying on a hard surface for a long time uncomfortable. Individuals can ask for a painkiller before the scan if required.

Bone scan

This scan does not diagnose MSCC but may be performed to check whether there are any abnormal areas inside the bone.

Rarely, MSCC is the first symptom of cancer. The doctor may recommend a biopsy of the spine to give an exact diagnosis.

Treatment

Treatment should be started as soon as possible after diagnosis, with the aim of minimizing permanent damage to the spinal cord. Treatment will also help to reduce pain by shrinking the tumor and relieving the pressure on the nerves. The damage to the spinal cord means that some individuals will have some paralysis at the time of diagnosis. This may be permanent in some people.

The choice of treatment depends on several factors including the type of cancer, the area of the spine affected, and general fitness. The most common treatment is radiotherapy, although surgery and chemotherapy are also used sometimes.

Treatment usually involves a combination of the following.

Assessing mobility

The doctor usually advises the patient to lie flat on the back until tests have shown whether the individual has a spinal cord compression or not. This is to reduce movement of the affected area of the spine and to prevent an increase in symptoms.

If the tests confirm the presence of spinal cord compression, the doctor and physiotherapist decide what movement is safe for the individual and explain the do's and don'ts.

During and after treatment, individuals will have regular physical examinations with the doctor and physiotherapist, when they will carry out a detailed check of the nervous system. This includes examining range of movement, muscle strength, co-ordination, and sensation to touch, which helps them to see any improvement in symptoms.

Collars and braces

Some individuals may be given a collar or brace to wear that can help to support their neck or spine, which the physiotherapist will discuss with the patient.

Steroids

Steroids are chemicals naturally produced in the body that help control and regulate how the body works. High doses of a steroid called dexamethasone are usually started immediately if spinal cord compression is suspected. The steroid helps reduce pressure and swelling around the spinal cord and can quickly relieve symptoms such as pain. The dose is gradually reduced over time and then stopped depending on the improvement of symptoms and after starting other treatments such as radiotherapy and surgery.

Radiotherapy

Radiotherapy is the use of high-energy rays to destroy cancer cells. It is the most common way to treat spinal cord compression. It is usually used on its own, or occasionally alongside other treatments such as surgery. It is given by directing radiotherapy rays at the tumor from outside the body, known as external radiotherapy. Radiotherapy is usually given as a short course of treatment. This can range from one single treatment to one treatment a day for 2 weeks. It may be given for up to 4 weeks for myeloma and lymphoma. Radiotherapy will be started as soon as possible after MSCC is diagnosed.

Surgery

Surgery is only suitable to treat a small number of people for their spinal cord compression. This depends on several factors, including the type of tumor, where it is situated, and how unstable the spine may be.

The aim of surgery is to remove as much of the tumor as possible and relieve pressure within the spinal canal.

Surgery may involve removing several parts of the vertebrae, as well as removing as much of the tumor as possible, without weakening the spine. The common surgical techniques used in this situation are called anterior stabilization and debulking of tumor, or decompression laminectomy.

This surgery may also involve stabilizing the spine further by means of metal rods or bone grafts. The doctor or nurse will explain the operation in more detail if surgery is appropriate.

If some of the tumor cannot be removed by surgery, or if the tumor comes back after initial treatment, radiotherapy is sometimes given.

Chemotherapy

Chemotherapy is the use of anticancer (cytotoxic) drugs to destroy cancer cells. It is occasionally used to treat spinal cord compression. It may be used for tumors that are sensitive to chemotherapy, such as lymphoma or small-cell lung cancer.

Chemotherapy and hormonal therapy can also be used after radiotherapy/surgery for certain cancers, such as breast and prostate cancers.

Coping with symptoms

Pain control

In case of pain, the doctor or nurse discuss ways of controlling pain. Different drugs may be given to help with pain, and these will be assessed regularly to make sure they are effective. Bisphosphonate drugs can be used to treat pain and weakened vertebrae in breast cancer and myeloma. They can also be used in prostate cancer if painkillers are ineffective.

Loss of mobility

Mobility may be affected by changes in muscle strength and ability to feel and control the movement in muscles. The physiotherapist will help individuals to adjust to these changes. An occupational therapist can give practical advice and supply aids to help the individual to be as independent as possible.

Bladder changes

The doctor and nurse monitor how well the bladder is working, and a thin flexible tube (catheter) may be used to help drain urine from the bladder in patients having problems in passing urine.

Bowel changes

Medication may be given to help with constipation or to help patients having difficulty controlling bowel.

After treatment have finished

Spinal cord compression can affect people differently. The care required after treatment depends on the result of treatment and level of mobility. Before leaving the hospital, the staff should organize any care the patient might require when at home.

Individuals who have lost the ability to walk or have lost movement before treatment, may not get this back. Further care may be available at cancer center, local hospital, or hospice. This involves a team of healthcare professionals who work closely with the patients and their family to organize a plan of care and rehabilitation to suit your needs.

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Conflicts of interest

None declared.

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