Review on Acute Cardio-Cerebral Infarction: a Case Report

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Abstract

Objective: To describe a case of a Cardio-Cerebral Infarction (CCI) male patient presented with a history of chest pain recognized using electrocardiography, brain computed tomography, and Percutaneous Coronary Intervention (PCI).

Methods: A 69 years old man came with history of chest pain since 13 hours before to the emergency room. Electrocardiography, brain computed tomography, and PCI were performed, leading to the diagnosis of CCI.

Results: The electrocardiography showed ST Elevation in Antero-lateral, atrial fibrillation and left-sided hemipharesis, which occurred on the second day. Brain computed tomography demonstrated acute infarct stroke, while the Percutaneous Coronary Intervention (PCI) showed one vessel disease with severe stenosis in LAD and implanted stent in proximal-mid LAD. Therapy prescribed was providing antiplatelet and anticoagulation.

Discussion: Acute Myocardial Infarction (AMI) and Acute Infarct Stroke (AIS) have a narrow therapeutic time-window and a delayed intervention may results in morbidity and death. Antiplatelet and anticoagulant used in PCI for AMI increase the risk for hemorrhagic, and AIS with thrombolytic increase the risk of cardiac wall rupture in AMI. Direct Oral Anticoagulant (DOAC) treatment should reduce ischaemia and lower bleeding. The optimal time point to start anticoagulant treatment might be between 4-14 days after the onset of stroke. Duration of post-PCI triple therapy should be minimized depending on bleeding and risks of ischemia.

Keywords: Infarction, magnetic resonance, relapse, spinal cord

Introduction

Spinal cord infarction (SCI) is a rare disease and constitutes one of the acute spinal emergencies. In comparison to brain ischemic stroke, the spinal cord infarction it presents an extremely low incidence, perhaps due to the abundance of the arterial anastomosis and low evidence of atherosclerosis in the spinal arteries. Since the first spinal cord infarction reported in early nineteenth century, there have been remarkable progresses in the understanding of this disease entity.[1]

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However, the fact that there is no established standard of care treatment as of today highlights the complexity and challenging nature of SCI.[1] Here we report the case of 84 years-old man who suffered from spine infarction with relapse after initial improvement.

Case

This case of 84 years-old Caucasian man who was in hospitalized due to sudden appearance, since about one month, of lumbar pain, bilateral lower limbs weakness, paresthesias from the knees to feet, associated with urge incontinence. Computed tomography (CT) without contrast was normal.

His vital signs upon arrival at the emergency

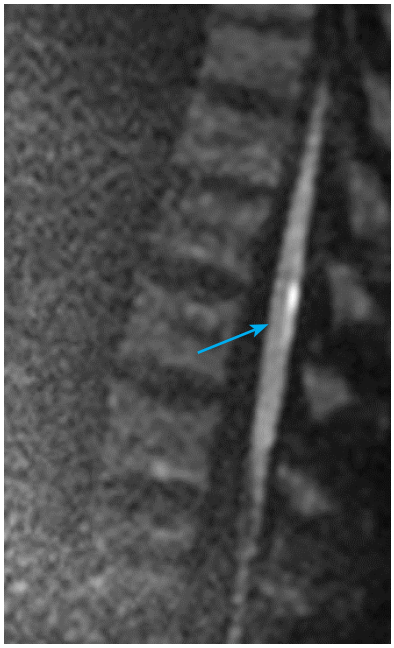

Fig. 1 DWI Sequences Showing D10-D12 Hyperintensity

room were as follows: body temperature, 36.2°C; pulse rate, 67 beats per minute; respiratory rate, 16 breaths per minute; blood pressure, 140/80 mm Hg; and oxygen saturation, 98% on ambient air.

Neurological examination at in-hospitalization showed bilateral weakness (4/5 Medical Research Council at the right lower arm, 3/5 at the left lower arm), with persistent paresthesias from the knees with distal spreading. Osteo-tendineous reflexes were bilaterally moderately brisk at the lower limb with mild prevalence at the left al while Babinski resulted mildly positive at left arm.

His medical history was positive for chronic anemia due to iron deficiency and colon cancer about three years before, surgically treated with left hemicolectomy. He was previously a heavy smoker (he used to smoke about one package of cigarettes in a day for about forty years, at the time of in-hospitalization, used to smoke 8 cigarettes in a day).

He performed spine magnetic resonance imaging (MRI) (Fig. 1) showing hyperintensity at posterior lateral columns of D10-D12 at diffusion- weighted imaging (DWI), without bleedings or expansive lesions and without enhancement after Gadolinium. Digital subtraction angiography was not performed and no sign of radiculomedullary artery was detected.

Heart and abdomen ultrasounds resulted all negative except for mild aortic valve stenosis and aortic calcifications. Brain MRI showed subcortical ischemic lacunae. Carotid ultrasound showed calcific plaques involving internal carotid arteries bilaterally the internal carotid artery (ICA), which resulted in a bilateral stenosis of about 30–35%. 24-hour holter monitoring resulted normal. Blood exams resulted negative except for triglycerides (263 mg/dL normal value <150), cholesterol (238 mg/dL normal value<190). Routine immunodeficiency virus and Syphilis screening, antinuclear antibodies, extractable nuclear antigen antibodies, cytoplasmic neutrophil antibodies resulted all normal.

He was discharged and rehabilitation

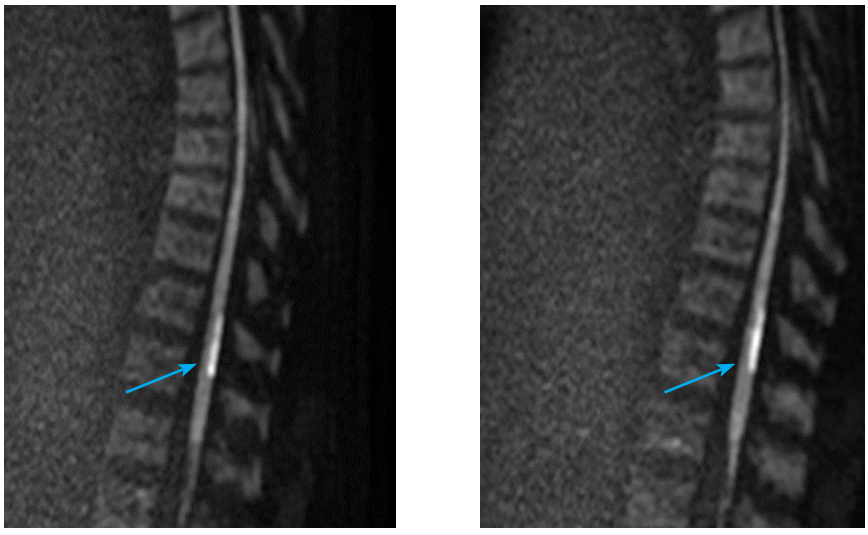

Fig. 2 DWI Sequences Showing Relapse of Stroke Involving Also the Medullary Cone

was promptly started. Urinary catheter was inserted due to persistent urinary incontinence. Antiaggregation with acetylsalicylic acid 100 mg 1/day was started, together with subcutaneous low molecular weight heparin (LMWH 4000 UI/day) and rosuvastatin 10 mg/day.

Neurological weakness improved in the first ten days, but, suddenly, he developed lower limbs paraplegia with bilateral hypoesthesia from the thighs with paresthesias at left limb from the knee to foot. Neurological examination showed lower limbs areflexia with negativity of Babinski. Contemporary lone atrial fibrillation with rapid ventricular response (160 beats per minute) was detected at monitor, so intravenous Diltiazem was promptly started together with contemporary anticoagulation oral therapy with Warfarin (maintaining INR between 2 and 3). Central pain at the lower limb was controlled with oxycodone-naloxone and pregabalin.

Spine MRI showed, at DWI sequences, relapse of stroke involving also the medullary cone (Fig. 2). Both magnetic resonances had to be quickly interrupted due to patient's intolerance with continuous movements. Axial sequenced resulted full of artifacts and unreliable.

Clinical course was complicated by left orchitis urinary tract infection (UTI) due to Klebsiella Pneumoniae. Lower limbs movement didn't improve and urinary catheter was maintained. Ethics approval and informed consent to participate were both given.

Discussion

Spinal cord ischemia (SCI) is rare and represents only 1.2% of strokes.[1] Given the rarity, it may be misdiagnosed as other pathologic processes such as transverse myelitis.[1]

Sudden reduction of blood flow to spinal cord gray matter or white matter, which results in disrupted oxygen and glucose delivery, causing metabolic failure of affected spinal cord tissue, constitute the main pathogenetic mechanism for SCI.[2]

Various etiologies that cause either global flow insufficiency or selective occlusion of a radiculomedullary artery.[2,] among which we can include atherosclerosis, coagulation disorders, vasculitis, infections, systemic hypotension, emboli, aortic dissection, decompression sickness.[2] Iatrogenic causes of spinal cord infarction include aortic stent-grafting, vascular surgery with aortic cross clamping, open hiatal hernia repair, lumbar sympathectomy, chest and abdomen surgery, adrenalectomy, and anesthetic procedures, such as epidural anesthesia, intercostal and celiac plexus block.[2]

The spinal cord receives its vascular support from three arteries: one anterior spinal artery (ASA) and two posterior (PSA) that span the length of the cord longitudinally. They originate from the vertebral arteries at the level of the craniocervical junction, anastomose via the vasocorona and transverse radicular branches forming the pial plexus, and in the end they originate perforating branches that can enter the spinal cord and supply different levels.[3] The ASA gives origin to the sulcocommissural artery, which is also responsible for providing blood supply to the anterior of the spinal cord.[4], while the PSA can generate posterior inferior cerebellar arteries (PICA) and can also rely on posterior radicular arteries (originating from a vertebral artery) for vascular supply.[4] Five to eight of the radicular arteries can be actively involved in supplying the ASA. In 90% of people one of the thoracolumbar arteries, known as the artery of Adamkiewicz,[4]provides vascular perfusion to the lower thoracic, lumbar spinal cord and to the conus medullaris.[1,2] High level of collateral circulation in spinal cord decrease its susceptibility to vascular injury[4]. Perfusion of the anterior two-thirds of the spinal cord and the anterior portion of the posterior column occurs via the anterior spinal artery (ASA), while the posterior remnant region is provided by the two posterior spinal arteries (PSA).[4] The PSA supplies the posterior columns, posterior dorsal horns, portions of the corticospinal and spinothalamic tracts.[4]

Each radicular artery supplies a different functional region of the spinal arteries, particularly the anterior spinal artery. The first region extends from C1 until T3.[4] The second region extends from T3 until T7 and sometimes receives a branch from the intercostal artery (usually at T7 level).[4] The third region extends from T8 to the cone and receives a branch (Adamkiewicz artery) from the intercostal artery.[4] There is sometimes a cone artery originating from the internal iliac artery (Desproges-Gotteron artery) at the L27 or L58 level.[4]

The most common clinical presentation of a spinal cord infarction is characterized by anterior spinal artery syndrome. Anterior spinal artery infarct typically presents with bilateral weakness and pain/temperature hypo-anesthesia, with relative sparing of

proprioception and vibration below the level of the lesion.[5] The acute stages are characterized by sudden appearance of flaccidity and loss of deep tendon reflexes; spasticity and hyper-reflexia develop during ensuing days and weeks. Autonomic dysfunction may be also observed and can manifest as hypotension, sexual dysfunction, and/or bowel- bladder dysfunction. If the lesion is in the rostral cervical cord, breathing can be compromised.[5]

Ischemia may be localized at the level of the anterior horns; in this case, clinical presentations may be characterized by sudden paraplegia (pseudopoliomyelitic form) without sensory abnormalities or sphincter dysfunction, painful bilateral brachial diplegia (the man-in-the-barrel syndrome in cervical SCI), progressive distal amyotrophy due to chronic lesions of the anterior horns.[7]

Clinical features are variable, and include loss of vibratory sensation and proprioception, suspended global anesthesia and segmental areflexia due to posterior horn involvement, with paresis below the level at which the posterior portion of the lateral column containing the crossed corticospinal tract is affected.[5]

Central sulcal artery occlusion can cause Brown-Sequard syndrome, which typically presents with ipsilateral weakness and posterior column sensory deficit (vibration and proprioception) below the lesion and contralateral decrease in pain and temperature sensation a few segments below the lesion.[6]

Central cord syndrome may theoretically be a manifestation of spinal cord ischemia, but commonly result from cervical spondylosis, trauma, syringomyelia, or other tumors. Location is the cervical or upper thoracic regions, involving the central portions of the spinal cord. The hallmark sign weakness in both the upper arms, with relative sparing of the legs. Affected patients also have varying degree of sensory loss below the lesion and urinary retention.[7-9]

Less common manifestations can be constituted by spinal cord transient ischemic attack, infarct of the conus medullaris, transverse medullary infarctions.

Laboratory examinations can include: blood cell count, copper, zinc, human T-lymphotropic virus 1 serology, vitamin B12, folate, homocysteine, cholesterol and tryglicerides, anti-cyclic citrullinated peptide, antiphospholipid antibodies, complement level essay, glucose, thyroid function, antinuclear antibody, anti-neutrophil cytoplasmic antibody, angiotensin-converting enzyme, aquaporin-4-IgG, myelin oligodendrocyte glycoprotein, hypercoagulable profile, and paraneoplastic autoantibody assessment, erythrocyte sedimentation rate, C reactive protein, protein C, S, electrolytes, Lyme disease, herpes virus, human immunodeficiency virus, human T-cell leukemia type 1, hepatitis B and syphilis serology.[7]

CT and conventional angiography are not considered as the first choice for the evaluation of SCI, The gold standard for diagnosis is MR imaging, which can be performed with both 1.5T and 3T scanners: sagittal spin-echo T2 or T2 STIR, sagittal spin-echo T1, axial gradient-echo T2 at the cervical and dorsal levels, spin-echo T2 at the medullary conus, and diffusion-weighted images in the sagittal or axial planes.[6]

The typical MRI findings in SCI diagnosis include isolated pencil-like area of T2 hyperintensity mostly confined to centro-medullary region with sparing of the anterior rim, often involving more than two vertebral segments on sagittal images.[11] Bilateral hyperintense T2 alterations that are mostly confined to the anterior horn area, leading to the axial "snake eyes or owl's eyes" configuration, can be observed among 90 % of patients with SCI, they are not specific or sensitive (in the acute phase, only 50 % of the cases shows a demarcation of T2 hyperintensity of the spine within the first 24 hours from symptoms appearance).[1]

Differential diagnosis of spinal cord infarction is broad and holds multiple entities, usually included among"acute nontraumatic myelopathy." These can be categorized into (a) inflammation, (b) infection, (c) compressive myelopathy, (d) venous congestion related to vascular malformations such as spinal dural arteriovenous fistula (SDAVF), and (e) tumor. Distinction of SCI from "mimics" is challenging especially in subacute stage. Hyperintensities on T2-WI can also be seen in inflammatory diseases and tumors.[1]

While the short-term mortality rate is 20-25% in the first month from symptoms onset, any spinal cord infarction patients experience significant improvement with time, perhaps due to better cares and improvement in rehabilitation techniques. Up to half of the patients who were unable to walk one week after spinal cord infarction reveal great improvement in the following months; particularly two-thirds are able even to walk without aids. In the long-term, almost all surviving patients <60 years with SCI onset are able to work, consequently long term prognosis is favourable.[8]

Up to 79% of spinal cord infarction patients report chronic pain on follow-up, and this is more frequent than in cerebral infarction patients where less than half report pain on follow-up.[8] Pain is not associated with functional state in spinal cord infarction patients.[9]

Prompt surgical management is necessary in cases of vascular compression and acute aortic event in addition to maintaining hemostatic equilibrium.[11] In the setting of aortic surgery, lumbar drainage and blood pressure augmentation are often options for management.[11] A review of spinal manifestations of patients with vertebral artery dissection revealed the use of anticoagulation was the sole form of therapy in spinal ischemia; antiplatelet therapy was also administered in conjunction with anticoagulants.[11] Another investigation used antiplatelet therapy in all 36 participants, including aspirin and clopidogrel.[11]

We reported the case of an 84 years old Caucasian man who was heavy smoker with dyslipidemia, who also developed lone atrial fibrillation. The particularity of the case was the relapse of stroke after one month with relative improvement of weakness, after which he was confined to wheelchair with paraplegia, urinary catheter.

The possible presentation of SCI in in 2 acute stages has been already considered. In the study performed by Novy in 20,065 patients presented with a biphasic stroke. Two of these 5 patients had a deficit that was initially unilateral and then became bilateral, while in another patient the deficit increased secondarily. The 2 remaining patients experienced transient ischemic attacks (TIAs) before spinal cord infarction (1 patient experienced several TIAs per year for 6 years). The TIAs had the same motor features as the definitive stroke. In these cases of progressive stroke or TIAs, the infarct was frequently localized at the cervical level (4 of 5 cases).

Etiologies of SCI are very heterogeneous and include thromboembolic events, aortic dissection, mechanical injury and trauma.

Cheng *et al.*[12] reported the case of one patient, who had had 2 similar episodes 3 weeks apart. The first attack was tingling sensation and slight weakness in one lower extremity followed by the second on the opposite side. The spinal cord MRI revealed a posterior spinal arterial infarction in the thoracic region.

We didn't find in literature a relapsing case after one month of acute stroke, not preceded by TIAs. We considered the possible role of lone atrial fibrillation in the pathogenesis of SCI. In a retrospective cohort study performed by Mir in 2017 atrial fibrillation was associated with increased risk of subsequent SCI. The association between AF and SCI persisted or grew stronger after excluding those with concomitant diagnoses of spinal cord injury, spinal abscess, and spinal or aortic surgery.[13] However, Wang *et al* in 2016 determined that SCI could predispose also to atrial fibrillation, but when strokes occurred above T6 level, due to the abnormal disruption of the communication between the brainstem and the autonomic nervous system.[14]

That's not the case of patients, but it is difficult to find the causes of the first stroke (he presented to emergency department after 20 days from the beginning of symptoms). Atrial fibrillation could have been in the trigger factor (undiagnosed at home).

Intravenous tissue plasminogen activator is currently considered the only therapy that can improve the outcome of acute ischemic stroke if administered within 3 hours (or 4.5 if we consider a recent randomized controlled trial).

Hyperbaric oxygen therapy has been proven to be neuroprotective when administered before or after spine stroke, through various mechanisms such as. improved spinal cord oxygen tension, reduced apoptosis and inflammation, improvement of oxidative stress and angiogenesis.

In conclusion this case reported a very rare event in literature. We think that it should be useful to perform a strict follow - up of risk factors for SCI, expecially among older patients and to consider the risk of relapse, even rare, which could have even more devastating effects on patients' disability and outcome.

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