Spinal Epidural Abscess in COVID-19 patients

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Short Report

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Abstract

Objective: To report the peculiarities of spinal epidural abscess in COVID-19 patients, because we observed an unusually high number of these patients following the outbreak of SARS-Corona Virus-2.

Methods: We reviewed the clinical documentation of six consecutive COVID-19 patients with primary spinal epidural abscess that we had to surgically manage during a two-month period. These cases were analyzed for what concerns both the viral infection and the spinal abscess.

Results: Abscess was primary in all cases meaning that no evident infective source was found. Primary abscess represents the rarest form of spinal epidural abscess, which is usually secondary to invasive procedures or spreading from adjacent infective sites, such as spondylodiscitis and generally occurs in patients with diabetes, obesity, cancer, or other chronic disease. In all cases, there was mild lymphopenia but the spinal abscess occurred regardless the severity of the viral disease, the immunologic state, and the presence of bacteremia. Obesity was the only risk factor and was reported just in two patients. All patients but one were hypertensive. The preferred localizations were cervical and thoracic, whereas the classic abscess generally occurs at lumbar level. No patient had history of pyogenic infection, even though previous asymptomatic bacterial contaminations were reported in three cases.

Conclusion: We wonder about the concentration of this uncommon disease in a so short period. To our knowledge, cases of epidural spinal abscess in COVID-19 patients have been not yet reported. Accordingly, we ignore if the SARS-Corona Virus-2 may really predispose to spinal epidural abscesses. However, we hypothesize that, in our patients, the spinal infection could have depended on the coexistence of an initially asymptomatic bacterial contamination. The well-known COVID-19-related endotheliitis might have created the conditions for retrograde bacterial invasion of the correspondent spinal epidural space. Anyway, the spinal epidural abscess carries significantly high morbidity and mortality. It is difficult to diagnose, especially in compromised COVID-19 patients but it should be kept in mind because early diagnosis and treatment are crucial.

Introduction

On February 21, the first case of Coronavirus Disease 2019 (COVID-19) was diagnosed in Lombardy. Afterwards, the contagion rapidly spread throughout the region. Up to today (June 12,

2020), 90,932 Lombards contracted the COVID-19 and 16,374 died, which makes Lombardy (a region of about 10 million inhabitants) one of the most plagued areas of the world. The Lombard Health System had to be reorganized to face the breakout: our department suspended all the elective procedures and managed only neurosurgical emergencies. During the last three months, six patients with SARS-Corona Virus-2 (SARS-COV-2) were referred to us for acute spinal cord syndrome due to primary spinal epidural abscess (SEA) [1]. No adjacent spondylodiscitis or other infection sources were evident. This time-concentrated incidence of primary SEA is quite unusual in our practice, and a relationship with the COVID-

19 is conceivable. Anyway, to our knowledge, to date there are no published cases of SEA in patients with COVID-19.

Material And Methods

During the peak of epidemic (March to May 2020), we had to surgically manage six COVID-19 patients with SEA. Their clinical charts, radiological documentation, and surgical reports were carefully reviewed looking for possible peculiarities for what concerns both the viral infection and the neurological disease. The main clinical features are summarized in Table 1.

Four men and two women (mean age = 58.5 years) were referred to us because of progressively worsening spinal cord dysfunction. All these patients presented with few day histories of back pain and had diagnosis of COVID-19.

Viral infection: On admission to our Department, the diagnosis of COVID-19 had been already ascertained in three patients and was highly suspected in a fourth one.

In particular, one patient was still hospitalized because of interstitial pneumonia (IP), whereas two patients had been recently discharged after recovery from IP. These 3 individuals had had positive nasopharyngeal swabs for SARS-COV-2 and their chest-computed tomography (CT) had shown the typical ground-glass appearance. Therapy had included tocilizumab, antibiotics, corticosteroids, anticoagulants, and antiviral agents. The fourth patient had been admitted to another department because of dyspnea, fever and anosmia. He had known history of deep vein thrombosis and chest-CT had shown pulmonary infarction and IP. Nasopharyngeal swabs were negative. Serologic tests were initially not available. Subsequently, such tests were planned but never performed because this patient deteriorated to a pre-agonic state owing to new pulmonary infarction. These four patients had required respiratory assistance (CPAP, NIMV, or intubation), but two had completely overcome the disease. The remaining two patients had histories of recent flu episodes but never presented clear symptoms of COVID-19. They had negative nasopharyngeal swabs, but serologic tests revealed exposure to SARS-COV-2.

Spinal Epidural Abscess: On admission to our Department, three patients were quadriparetic, two were paraparetic, and one was paraplegic. Neurological conditions were rapidly worsening in all cases. Magnetic resonance imaging (MRI) (Figure 1) showed epidural abscesses at cervical (3 patients), thoracic (2 patient), and lumbar (1 patient) levels. In no case, the MRI showed spondylodiscitis or any other possible sources of infection. Mild fever was reported only in one patient. Slight neutrophil increase and lymphopenia were present in all cases. Procalcitonin was increased in 3 cases and C-reactive Protein (CRP) was increased in all cases but one.

All patients underwent emergency emi-laminectomy with pus drainage and spinal cord decompression.

Results

In all cases, cultural assay of the surgical material revealed methicillin-sensible Staphylococcus Aureus (MSSA). During the following days, MSSA was also found in the blood of 2 patients despite no sign of sepsis was evident. In all cases, antibiogram allowed adequate and effective antibiotic therapy. Postoperatively, the infectologist consultant carefully searched for possible hidden sources of infection, but clinical examination, total body CT-scan, orthopantomography, and cultural assays of several fluids and secretions were all negative. The previous clinical histories were carefully examined too. All these 6 patients had never presented clinical evidence of pyogenic infection. However, when we managed them for the SEA, we retrospectively reconstructed that, some weeks before, staphylococcus aureus (SA) had been found in the expectoration and in the oropharyngeal swab respectively of two and one patients. At that time, patients were asymptomatic for bacterial infection and these findings were interpreted as sample contamination. No patient was intravenous drug abuser. Predisposing factors for SEA (obesity and obesity plus diabetes) were reported only in two cases.

Repeated MRI documented the abscess resolution in all cases. Within a couple of weeks all patients were referred to rehabilitation units. One month later, that patient with known deep vein thrombosis was still quadriplegic, suffered a new pulmonary infarction and rapidly deteriorated. Two months after surgery, partial recovery was evident in four patients, whereas one was completely paraplegic. Anyway, no patient completely recovered the initial spinal cord syndrome.

Discussion

SEA

Risk factors for SEA include diabetes, immunosuppressed state, intravenous drug abuse, chronic renal or liver failure, spinal surgery, obesity, and bacteremia [2-9]. Despite the incidence of SEA progressively increased during the last decades [3-7,9], indeed SEA remains uncommon ranging from 1.2 to 3 per 10,000 hospitalized patients [3,7,9], with less than 1000 cases published over a 40 years period [5,10]. Cases of primary SEA that are cases without any evident infective source are even rarer accounting for 20% [1,2].

The bacteria may enter the epidural space spreading from adjacent local infections (such as discitis), contaminating invasive procedures (such as spinal surgery or spinal anesthesia), or through the hematogenous route [3-6,9]. SA, usually the MSSA, is responsible for 70% of cases [3,4,6,7,9].

The classic triad of SEA consists of back pain, fever, and neurologic deficits but it is just reported in 8% of cases [3-7,9]. Pain is constant, fever is present in less than 50% of cases, true neurological deficits affect a minority of patients [4-6,9,10]. Onset of symptoms may be sudden, or slowly progressive but back pain often evolves to paraplegia within few days [7]. Accordingly, early diagnosis is crucial, but it is difficult, and half of cases are initially misdiagnosed [3-7,9].

White blood cell (WBC) count is normal in about half of patients, while erythrocyte sedimentation rate (ESR) and CRP are generally elevated [4,5,7,9-11]. Bacteremia causing or arising from SEA is detected in

60% of patients [5-7].

MRI with contrast is the diagnostic method of choice: SEA is generally seen as a T1 hypointense, T2 hyperintense mass with enhancing capsule in the epidural space [4-7]. Most of SEAs are lumbar and posterior because infections are more likely in larger fatty epidural spaces [5].

Decompressive laminectomy and drainage together with systemic antibiotics are mandatory in patients with neurological symptoms [5,9]. Spinal fusion is to be considered in secondary SEA when spondylodiscitis causes structural compromise to the spinal column [6,12]. Pending the identification of the causative organism, empiric therapy should start using broad spectrum antibiotics [5]. Therapy usually include vancomycin, last generation cephalosporin, and sometimes aminoglycoside, and/or metronidazole [3,4,6,7]. There are no guidelines for the duration of therapy, but patients typically require 4-8 weeks of therapy [3-7,9]

Postoperative recovery depends on age, health status, comorbidities, and history duration [13-15] but above all on the patient's neurologic status immediately before surgery [2,4,5,7]. Despite recent improvements, outcomes of SEA remain poor, with mortality ranging from 5% to 23% and neurological morbidity ranging from 4% to 55% [4-7,9,16].

COVID-19

COVID-19 primarily is a respiratory tract infection with significant systemic impaction on cardiovascular, neurologic, gastrointestinal, hematopoietic, hemostatic, and immune systems [17-19]. It is now widely accepted that a sort of disseminated intravascular coagulation (DIC) may develop with platelet consumption and hemorrhagic risk [19-21]. Recent studies show that the SARS-COV-2 provokes a diffuse damage to the vascular endothelium [20,21]. In a large autoptic series [21], all cases presented more or less degree of endothelial damage and arteriolar thrombosis was evident in 87% of cases. DIC and microembolisms are caused by this sort of endotheliitis and the IP can be *de facto* considered as a diffuse micro-infarction of the lung. The diffuse cellular damage would trigger autoimmune response with further cellular destruction. Lymphocytes and monocytes often decrease with possible impaired immune response to exogenous infective agents [17,22,23]. Bacterial infections have been reported in half of patients [18-20,22,23]. From a practical point of view, COVID-19 may consist of a complex clinical situation including disseminated microembolisms, bleeding diathesis, diffuse vasculitis, and autoimmune aggression with decreased antibacterial defence. However, asymptomatic or paucisymptomatic cases are common. Apart from direct antiviral therapy, treatment of COVID-19 mainly consists of anti-inflammatory drugs, anticoagulants, immunomodulators and antibiotics [18,19,23].

COVID-19 and primary SEA

During the last 10 years, we surgically treated a total of 7 patients with primary SEA that means without spondylodiscitis or evident infective source. Following the outbreak of COVID-19, we received 6 patients in a couple of months. Three of these patients complained of full-blown severe COVID-19. In another

patient, the diagnosis was only based on clinical symptoms, but we think it was highly probable. In two patients, the viral infection was almost asymptomatic and just revealed by the serologic tests. Accordingly, the clinical severity of COVID-19 was not correlated to the SEA occurrence. Moreover, when SEA occurred, only two patients were still fighting against active COVID-19.

These six patients presented some differences from the classical SEA patients: they were relatively younger; none was drug-abuser; only two were obese and only one of these was diabetic; only one harboured lumbosacral SEA. All these patients had lymphopenia and three had previously received immunomodulators to face the viral infection. Mild immunodeficiency cannot be excluded even in those two patients who were asymptomatic for COVID-19. Nonetheless, we do not believe that immunodeficiency played a major role in the SEA development. As aforementioned, immunocompromised state represents a risk factor for developing SEA and lymphopenia is quite common in COVID-19 patients. Accordingly, an increased SEA incidence could be expected in COVID-19 patients. Conversely, to our knowledge, cases of SEA in COVID-19 patients have been not yet published. Theoretically, SEA could have been underdiagnosed in comatose or severely compromised COVID-19 patients. Otherwise, SEA simply could have been not reported because physicians focused on other aspects of the disease.

In two patients, the MSSA was also subsequently found in blood cultures. Perhaps, bacteremia could have caused the SEA, but it is also possible that the bacterium secondarily entered the blood from the SEA [5-7]. Both these patients were apyretic, never presented signs of sepsis, and WBC and neutrophils were just moderately increased.

All patients had never presented clinical evidence of pyogenic infection. Nosocomial infections may be perhaps suspected in the two hospitalized patients, but these presented no sign of sepsis or other infections. Three patients had history of recent asymptomatic SA presence in pharynx and expectoration, that had been interpreted as sample contamination. When SEA occurred, neither pharyngeal nor pulmonary infections were evident. Interestingly, within few weeks, the two patients with contaminated expectoration developed thoracic SEA, the one with contaminated swab suffered from cervical SEA. A retrograde spinal invasion is thus conceivable.

As aforementioned, the coronavirus is typically responsible of a diffuse endothelial damage [20,21]. In two of our patients with IP, the chest-CT-scan (Figure 1) also showed the “atoll-sign”, which is a well-known expression of inflammation and granulomatous reaction in organizing pneumonia and is classically associated to angioinvasive agents [24]. All patients but one had history of arterial hypertension. We wonder if this could have had an effect on the vascular wall. Anyway, some degree of damage to the vascular endothelium is presumable in all our patients. This could have favoured the vascular penetration of SA even in absence of a clear SA infection. By this way, SA could have retrogradely reached the correspondent spinal epidural space causing progressive cellulitis of the epidural fat with the ultimate formation of SEA.

If this was the case, the higher than normal SEA incidence in this population might be explained. Of course, we are not stating that COVID-19 was responsible for SEA development but a role can be hypothesized. The viral infection might create the conditions for spinal invasion in subjects that are predisposed owing to the presence of a bacterium in a given location. This might also account for the cervical and thoracic SEAs that are relatively uncommon in classic SEA patients. Even the late onset of SEA following the recovery from the COVID-19 might be explained by the time to retrogradely invade the epidural space.

COVID-19 patients may present problems that can seriously hamper surgeries [25]. However, we did not encounter particular surgical problems in these 6 patients whose platelets were normal and immunological, respiratory, and circulatory states were acceptable. Despite relatively prompt treatment, no patient completely recovered from the spinal cord damage. Since the preoperative status is the main determinant of favourable outcome [2,4,5,7] and SEA may be encountered at unexpected rates, careful neurological examination of COVID-19 patients is mandatory.

Conclusion

Following the outbreak of COVID-19, we noticed an unusual high incidence of primary SEA which indeed should be quite uncommon in non-drug-abuser patients. In our mind, if the SEA development were just related to immunocompromised state or to simple nosocomial superinfections, we should encounter much more patients with SEA considering the magnitude of the pandemic. Instead, no case has been yet published even though we suspect that the incidence might be higher.

We hypothesize that SEA may develop because an asymptomatic bacterial colonization co-exists with damage of the vascular endothelium induced by the COVID-19 at the same level. This could favour the retrograde spinal invasion at the correspondent level.

Since the outcome of SEA remains poor in many patients mainly because of delayed diagnosis and treatment, physicians should be aware that COVID-19 patients may have some more risk of SEA than the general population.

Declarations

Funding: The Authors declare that they received no funding

Conflicts of interest/Competing interests: The Authors declare no conflicts of interest associated with this study.

Availability of data and material: All the data supporting our findings are contained within manuscript.

Code availability: Not applicable

Ethical approval: This study was approved by the ASST Niguarda Ethics Committee

Informed consent: On behalf of all the authors, I hereby certify that all our patients consented to be reported in our paper “Spinal Epidural Abscess in COVID-19 patients”.

Consent for publication: All authors have approved publication of the manuscript.

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Table

Table 1. Patient's Data

	COVID-19 diagnosis	COVID-19 Treatment	Previous Medical History	Symptoms of Spinal Abscess	MRI	Blood Assay		Cultural Assay	Previous MSSA localizations	Months after surgery	Two-month Outcome
Male 48 yrs	Positive Swab Chest-CT: IP	CPAP lopinavir/topinavir hydroxychloroquine tocilizumab enoxiparin	Hypertension Obesity	Pain, Paraplegia Mild Fever	Th1-Th7	WBC Neutrophils Lymphocytes RBC Platelets Procalcitonin CRP	+ + - norm. norm. + norm	MSSA in Abscess & more lately in Urine	Expectoration (1 month before)	2	Healed Infection No Pain Unchanged Paraplegia
Male 57 yrs	Positive Swab Chest-CT: IP	INTUBAT./VENTIL. tocilizumab, azithromycin, ceftriaxone, corticosteroid, enoxiparin	Hypertension Dyslipidemia	Pain, Quadripar. No Fever	C4-C6	WBC Neutrophils Lymphocytes RBC Platelets Procalcitonin CRP	+ + - norm norm. n.a. +++.	MSSA in Abscess & Blood	Oropharyngeal Swab (15 days before)	1.5	Healed Infection No Pain Improved Quadripar.
Male 55 yrs	Positive Swab Chest-CT: IP	CPAP tocilizumab, amoxicillin/ clavulanic acid, heparin	Hypertension NH Lymphoma Myocardial Infarction	Pain, Ascending Quadripar. No Fever	C5-Th1	WBC Neutrophils Lymphocytes RBC Platelets Procalcitonin CRP	- + --- norm. norm. n.a. n.a.	MSSA in Abscess	-	1	Healed Infection No Pain Improved Paraparesis
Fem., 78 yrs	Negative Swab Positive Serology	-	Hypertension Diabetes Obesity	Worsening Paraparesis No Fever	Th7-Th12	WBC Neutrophils Lymphocytes RBC Platelets Procalcitonin CRP	+ + - norm. norm. + +	MSSA in Abscess	Expectoration (15 days before)	2	Healed Infection No Pain Improved Paraparesis
Male 56 yrs	Negative Swab High Clinical Suspicion	NIMV, corticosteroid, oxacillin, gentamicin, heparin	Hypertension Deep Vein Thrombosis	Pain, Ascending Quadripar. No Fever	C1-C2 & C7-D1	WBC Neutrophils Lymphocytes RBC Platelets Procalcitonin CRP	++ + --- - norm. + ++	MSSA in Abscess	-	2.5	Healed Infection No Pain Unchanged Quadriplegia Muribund
Fem., 57 yrs	Negative Swab Positive Serology	-	Iron Deficency Anemia,	Pain, Paraparesis No Fever	Th12-L5	WBC Neutrophils Lymphocytes RBC Platelets Procalcitonin CRP	+ + --- -- - norm. +	MSSA in Abscess & Blood	-	2	Healed Infection No Pain Improved Paraparesis

COVID-19 = Corona Virus Disease 2019, IP = Interstitial Pneumonia, CPAP = Continuous Positive Airway Pressure, NIMV = Non Invasive Mechanical, Ventilation, NH = Non-Hodgkin WBC = White Blood Cells, RBC = Red Blood Cells, CRP = C-reactive Protein, MSSA = Methicillin-Sensible Staphylococcus Aureus

Figures

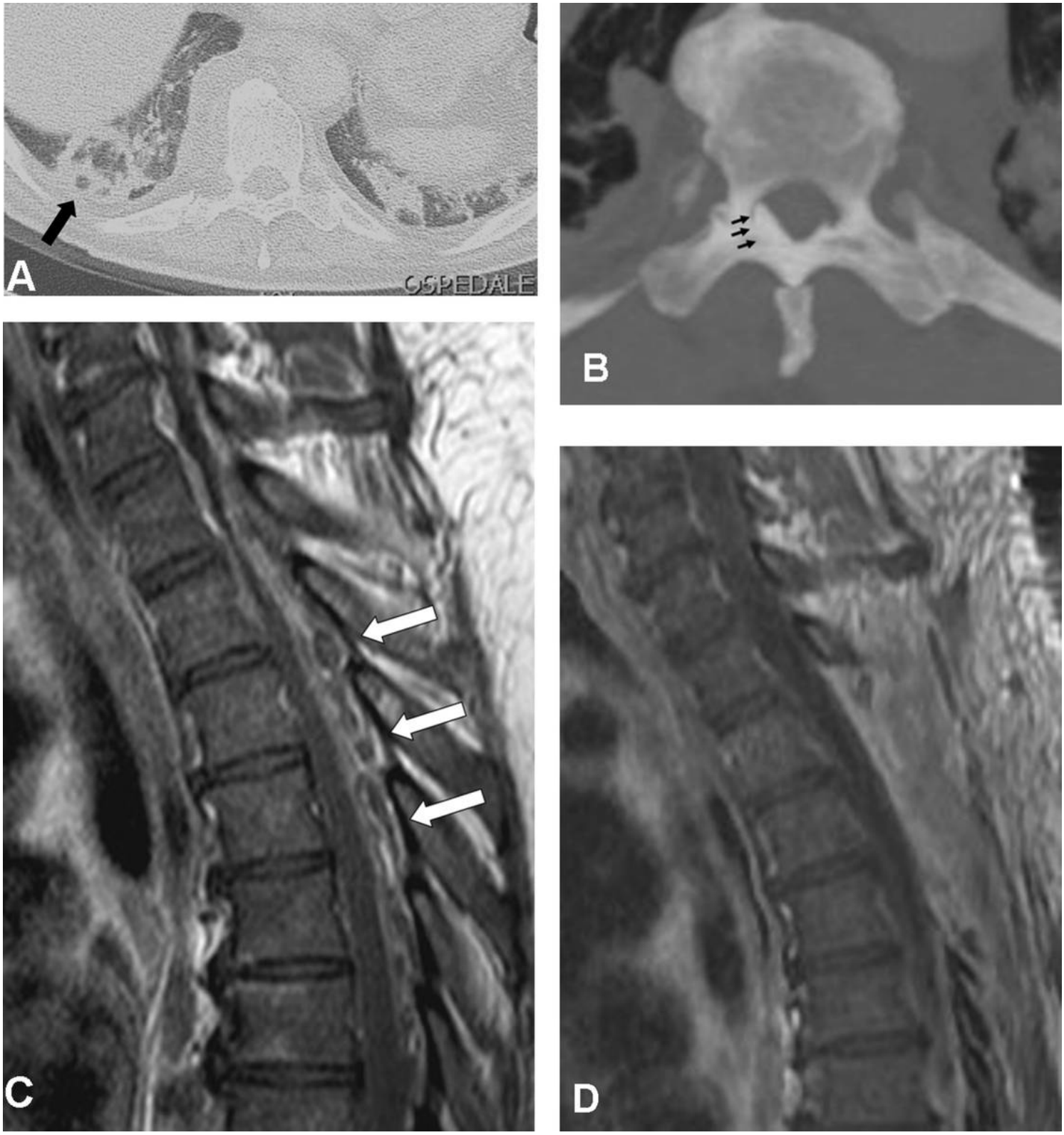

Figure 1

A) Chest CT-scan obtained in a 48-year-old man to control the evolution of Interstitial Pneumonia due to COVID-19. The “atoll sign” (arrow), that is expression of organizing pneumonia, is evident. B) Particular of the enhanced chest-CT scan focusing on the thoracic spine: an enhancing lesion is visible in the epidural space (arrows). C) Spinal MRI obtained the following day showing an epidural abscess extending from

Th1 to Th7 (arrows). D) Postoperative MRI showing the good drainage of the abscess with three-level emi-laminectomy.