

JMR

JUNIOR MEDICAL RESEARCH JOURNAL

A QUARTERLY JOURNAL FOR YOUNG AND TALENTED

VOLUME 1 NUMBER 2

2018



Editorial

A futuristic educational perspective for health practioners.

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Continuing Medical Education (CME) is a concept developed and used since the 1970's in. Its efficiency in ensuring medical practioners up-to-date is no more discussable nowadays. The terms Continuing Professional Development (CPD) and Continuing Medical Education (CME) are usually used interchangeably. However, the CME should be considered as a component of CPD. The CPD system establishes a balance between group learning, self-directed learning, and assessment activities.

The educational goals of CPD includes expanding knowledge, acquiring skills, developing new competencies, improving performance and patient care outcomes, as well as supporting multiple transitions as a natural part of career development.

Adults learning is a more complex process. This may be due to many unpredited factors; such as the lack of information, reduced motivation, time unavailability, and the wrong perception of scientific education necessity. Different objective studies have proven that continuous learning process is multi-dimensional and could be held in different approaches for adults. Continuous education for health practioners is a more sophisticated process. The clinical practice requires a specific orientation for self- learning; system thinking management; and team working ability.



CME initiative reinforces active information seeking, and sets the best learning atmosphere for health practioners. who are struggling to be up-to-date either due to lack of necessary information or sometimes due to its excess.

It makes an environment of collective decision-making and contribute to the elaboration of standards and guidelines. CPD includes a focus on discipline-specific knowledge and embraces learning across a wide range of content. It is a lifelong learning process which enables health professionals to maintain and improve different skills such as communication; leadership and management; evidence based practice and clinical guidelines; and quality improvement.

The educational goals will not only provide measureable outcomes for healthcare practitioners but also establish the value and commitment of each one.



In order to ensure health practioners engagement in CPD, a credit points system is established. For each self-learning, group learning, and assessment activities; the points are assigned according to the aims fulfilled and the number of hours spent to complete the target. The renewal of the parctionner license belong to a sufficient number of credit points in any countries.

Unfortunately, in Tunisia this concept is not yet developed. This could revolutionize the health practice once established.



Original Article

Non tumoral portal vein thrombosis during cirrhosis: Should anticoagulation be proposed?

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Publication data:

Submitted: January 17,2018

Accepted: March 4,2018

Available Online: June 22,2018

This article was subject to full peer-review.

Abstract

Background:

Portal vein thrombosis (PVT) is considered as infrequent and pejorative event in cirrhosis. Up to date, many questions remain about therapeutic management.

Aim:

The objectives of this study were to assess the impact of the PVT on the progression of liver disease, to review the indications for anticoagulation and its repercussions.

Materials and methods:

A case-control study was conducted over a period of 12 years (2002-2013). It included 484 cases of cirrhosis. Among these patients, 41 had non tumoral portal vein thrombosis (case group). The control group included the remaining 443 patients.

Results:

In our study, there was no impact of PVT on the natural history of cirrhosis both in terms of complications or survival. Only the early introduction of anticoagulant therapy was associated with a re-permeabilization of portal vein at one year (OR1.6; 95% CI [1.10-2.01]). Prolonged anticoagulation was inversely correlated with recurrent PVT after treatment. However, obtaining a portal vein re-permeabilization was not correlated to a significant gain in terms of prevention of complication related to cirrhosis and survival.

Conclusions:

results suggest that portal vein thrombosis in patients with cirrhosis is not a formal indication for anticoagulant therapy. It should be reserved for candidates of liver transplantation, those with an extension of the PVT to mesenteric vessels or with severe prothrombotic status.

Key words:

portal vein thrombosis, cirrhosis, anticoagulation.

Non tumoral portal vein thrombosis during cirrhosis: Should anticoagulation be proposed?

Introduction:

Non tumoral Portal vein thrombosis (PVT) during cirrhosis is considered as an uncommon and pejorative event [1]. The causes of PVT belong usually to local and / or general factors, including cirrhosis [2]. However, the impact of PVT on the cirrhosis mortality and liver disease progression remains questionable. Therapeutic management of PVT remains difficult due to the lack of national and international guidelines and the absence of objective tools for benefit-risk balance assessment.

Patients and methods:

In our work, we first investigated the indications of anticoagulants in a group of cirrhotic patients with non-tumoral PVT. We studied efficiency as well as complications occurring during anticoagulation. In a second step, we studied the effect of PVT on the progression of liver disease and the impact of the re-permeabilization on survival.

A case-control study including all adults with cirrhosis hospitalized in the Gastroenterology department of the Habib Thameur Hospital during 12 years (January 2002 May 2013) was performed.

The case group consisted in patients with:

-Cirrhosis diagnosed most often on the association of clinical, biological, morphological and endoscopic arguments;

-Acute or chronic PVT diagnosed by Doppler ultrasound or by tomodesitometry with intravenous contrast;

-A minimum follow-up of 3 months;

The control group was composed of patients with the same inclusion criteria but without PVT.

Patients with a history of neoplastic pathology in remission, or hepatocellular carcinoma (HCC) were not included in our study.

Exclusion criteria were:

-Patients who received anticoagulation for another indication than the PVT before their inclusion;

-Patients with a follow-up of less than 3 months;

- patients who developed a HCC within a period of 6 months next to PVT diagnosis.

The diagnosis of PVT was made by ultrasound coupled with the Doppler or by a tomodesitometry with contrast injection. The main objective of imaging was to establish the diagnosis of PVT, to determine its partial or total character, to specify its extension in particular to splanchnic vessels and to eliminate mesenteric venous ischemia

Imaging aimed also to eliminate neoplastic causes for PVT as well as septic pylephlebitis.

Endoscopic monitoring was performed for all patients according to the last Baveno VI guidelines. Primary or secondary prophylaxis of gastrointestinal bleeding was established according to endoscopic data. Each time a treatment for PVT has been established, the following data have been specified: the therapeutic indication, the modalities of the treatment, the delay in initiating the treatment with respect to the diagnosis of PVT and its duration. Clinical and radiological follow-up of the patients were recorded.

We studied the spontaneous radiological evolution or under anticoagulant treatment, as well as the evolution of the hepatic function according to the re-permeabilization or not of the portal vein. When a radiological follow-up was carried out during the year following the diagnosis of PVT, the reversal of the PVT was qualified as total, partial or absent.

The success of the treatment instituted was confirmed by a total re-permeabilization of the portal vein.

Hemorrhagic complications (digestive or extra-digestive) under anti-coagulation were recorded, as well as their time of appearance and their evolution. At the end of the study survival was compared in both groups.

Non tumoral portal vein thrombosis during cirrhosis: Should anticoagulation be proposed?

The statistical analysis was carried out by SPSS.21.

The averages were compared using the Student T test and the Mann and Whitney nonparametric test. The comparison of percentages on independent series was carried out by the Pearson Chi-square test and the Fisher test. The survival analysis was performed according to the Kaplan-Meier method. The analysis of the prognostic factors was based on the Log-Rank test for the univariate analysis. A logistic regression according to the Cox model was used for the multivariate analysis. A p value was considered statistically significant if <0.05.

Results:

A total of 548 cirrhotic patients hospitalized in the department were recorded. In total, 484 cirrhotic patients have no HCC, in which 41 cases with PVT and 443 controls were included. The prevalence of non tumoral PVT in cirrhosis was thus of 8.5% in our study. Twenty-three patients (56.1%) received anticoagulant therapy.

The indications of anticoagulation were:

- Extension of the PVT to the mesenteric vessels with or without signs of intestinal ischemia: 12 patients (one died before the beginning of anticoagulation).
- Severe prothrombotic status (protein C deficiency, anti-thrombin III deficiency): 3 patients (1 case of one extension of the PVT to the mesenteric veins).
- When the benefit-risk balance was in favor of anticoagulant treatment: 10 patients with mild cirrhosis (CHILD A and B7 score).

All patients treated (n=23) received Antivitamin K (AVK)-based anticoagulation. The 11 patients with extension of the PVT to the mesenteric vessels with or without signs of intestinal ischemia as well as the 2 patients with a severe prothrombotic status initially received an anticoagulant treatment based on low molecular weight heparin (LMWH) then relayed by the AVK.

The average time to introduce AVK was 2.6 days. LMWH was introduced immediately in case of mesenteric ischemia. The mean duration of anticoagulation was 8.65 months (1-24).

The average duration of follow-up was 26.4 months (1-120). Seven patients had no radiological control of their PVT. For the others, Doppler monitoring was performed every 3 to 6 months.

Among the 34 patients followed over 3 months, re-permeabilization was obtained in 19 cases (55.8%). It was total in 29% of cases. In patients with anticoagulant therapy (n=23), portal re-permeabilization was obtained in 69.5% (n=16) and was total in 10 (43.5%). In the 11 untreated patients, re-permeabilization was obtained in only 27.2% (n=3) and no case of total re-permeabilization of the portal vein was noted. The difference was statistically significant (p=0.025) (Figure 1). In the treated group re-permeabilization was obtained within a year in 79% of cases. The average duration of re-permeabilization was 7.9 months. All patients treated for 12 months (n=10) had complete re-permeabilization of their portal vein. On the other hand, in 9 patients treated for less than 6 months, a re-permeabilization was obtained in 44.4% of the cases (n=4), and a PVT reappeared in one case. (Figure 2)

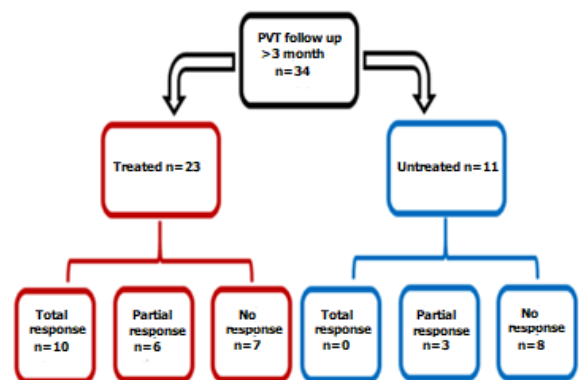


Figure 1: Evaluation of the re-permeabilization of the portal vein with vs without anticoagulation

Non tumoral portal vein thrombosis during cirrhosis: Should anticoagulation be proposed?

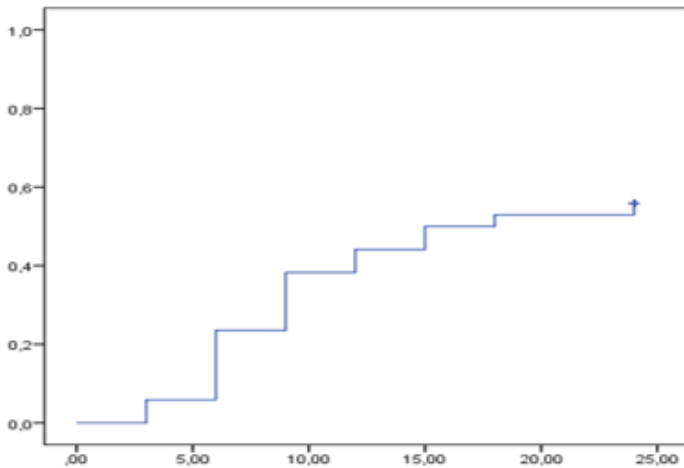


Figure 2: time to PVT re-permeabilization (months)

During the follow-up, 2 patients presented extra-digestive hemorrhage (epistaxis) and one case of gastrointestinal hemorrhage due to varicose rupture. Two cases of gastrointestinal hemorrhage were recorded in untreated patients (6.5%).

Eleven (32.3%) patients developed a non-hemorrhagic complication following the diagnosis of PVT including 2 cases of refractory ascites, 5 cases of hepatic encephalopathy and 4 cases of spontaneous bacterial peritonitis. Overall survival at 1 year and 2 years were respectively 68.3% and 34.1%.

The median survival was 24 months. At two years, 4 of the 27 patients who died were in the successful group. The remaining 23 were among patients with failure or absence of the treatment. Liver disease progression was the cause for all the patients of the treated group and for 20 patients from the other group.

Thus, at two years, the overall mortality rates in the two groups were 40% and 74.2% respectively. If only specific mortality is considered, the respective rates increase to 40% and 64.5%.

The introduction of an anticoagulant treatment but especially its early character (within 30 days after the diagnosis of the PVT) represented decisive factors in the obtaining of a portal re-permeabilization in our study. Thus, 3 factors appeared to be correlate with portal re-permeabilization in univariate analysis: initiation of anticoagulant therapy ($p = 0.025$), initiation of treatment within one month after diagnosis of PVT ($p = 0.005$), and a partial PVT ($p = 0.027$). However, in multivariate analysis, only the rapid onset of treatment within 7 days was significantly correlated with re-permeabilization of the portal vein with an OR of 1.6; 95% CI [1.10-2.01] (Table 1)

The introduction of effective anticoagulant therapy (with complete portal re-permeabilization) does not seem to have any effect on the evolution of cirrhosis. Thus, there was no significant difference between the two groups of patients in case of regression or persistence of PVT concerning complications such as spontaneous bacterial peritonitis ($p = 0.912$), refractory ascites ($p = 0.263$), hepatic encephalopathy ($p = 0.748$), gastrointestinal hemorrhage ($p = 0.421$). Moreover, the overall rate of complications was comparable between the two groups ($p = 0.452$).

Twenty-four liver-related deaths occurred in the first two years, of which 4 were successfully treated for the PVT. There was no significant difference between the patients in whom total permeability was obtained comparing specific mortality rate at 1 year ($p = 0.282$) and at 2 years ($p = 0.171$) (Figure 3).

Non tumoral portal vein thrombosis during cirrhosis: Should anticoagulation be proposed?

<i>Variable</i>	<i>p (univariate analysis)</i>	<i>p (multivariate analysis)</i>
Treatment	0.025	NS
Partial PVT	0.027	NS
Initiation of the treatment within 30 days	0.005	0.038; OR=1.6 [1.1 – 2.01]

Table 1: Predictive factors of re-permeabilization of the portal vein

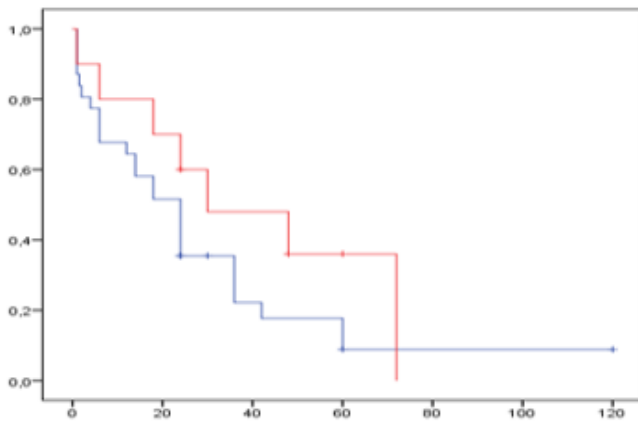


Figure 3: Survival after total re-permeabilization obtained (blue curve) vs not obtained (red curve)

Three hemorrhagic events (2 cases of epistaxis and 1 case of gastrointestinal hemorrhage due to varicose rupture) occurred under AVK. The mean time to bleeding complication was 1.7 months and the 3 hemorrhagic events occurred during the first quarter of treatment. Two cases of gastrointestinal hemorrhage occurred in untreated patients (6.5%). The patients presenting hemorrhagic events were minor and the AVK treatment was not discontinued.

Discussion:

The lack of consensual guidelines for the management of PVT in cirrhotic patients is may be due to the difficult assessment its impact on the cirrhosis natural history. However, the basic concept of any proposed treatment is the safety and the positive impact on the evolution of the liver disease [3].

Regarding primary prevention, Villa et al demonstrated that the use of Enoxaparin 4000 IU once daily for 48 weeks in CHILD B7-C10 cirrhotic patients prevented both the onset of PVT and the decompensation of the cirrhosis ($p < 0,0001$ compared to controls) and improved mortality ($p=0.02$) with a benefit maintained between 2 and 4 years [4]. This suggests the action of anticoagulation both on PVT and progression of hepatopathy. Thus, alteration of hepatic and / or intestinal microcirculation seems to be under the direct influence of coagulation abnormalities.

Previously in the literature; 6 studies including a total of 199 patients treated this topic (Table 2).

Our results about the effectiveness of anticoagulation for PVT in cirrhotic patients are limited. Heterogeneous data; the lack of precision in the assessment of the PVT extension; and unavoidable selection bias in some situations decreased the specificity of the analysis. However, the tolerance and the absence of interference with the mortality due to digestive bleeding are well demonstrated now. This was also remarkable in many other studies [4,6,9,10]. For spontaneous or induced (secondary to paracentesis for example) extra-digestive hemorrhage, the only established risk factor is severe thrombocytopenia $< 50,000 / ml$ [5-10].

Non tumoral portal vein thrombosis during cirrhosis: Should anticoagulation be proposed?

	Type of study	n (controls)	Severity of the cirrhosis	Type and duration of anticoagulation	Type of PVT (patial/total)	Re-permeabilization (total/partial)	Stabilization/ progression	Bleeding complications
Werner and al (3)	Retrospective	28	MELD 7-29	Warfarin 10 months	-	11 (39.3%) / 17 (60.7%)	10(53%)/1(5%)	1 vaginal
Villa and al (4)	Controlled randomized essay	34 (36)	Child Pugh 7-10	Enoxaparin 12 to 24 months	Primary prevention	-	7/0	4 (2 digestive)
Delgado and al (6)	Retrospective	55	MELD 12.8	Warfarin 6.8 months	41 (75%) / 14 (25%)	25 (45%) / 30 (55%)	0/0	10 (8 digestive)
Amiltrano and al (7)	Prospective	28	-	Enoxaparin 6months	23 (82%) / 5 (18%)	21 (75%) / 5 (18%)	0 (treated)/10(28%) untreated	0
Senzolo and al (12)	Cases, controlled, Prospective	33 (21)	MELD 12.6	Nadraparin 6 months	24 (69%) / 11 (31%)	12 (34%) / 9 (26%)	0/2(7%)	4 (1 digestive)
Francoz and al (16)	Cases, controlled, Prospective	19 (10)	MELD 12.8	Warfarin 8 months	18 (95%) / 1 (5%)	8 (42%) / 0	7(20%)/5(15%)	1 (after band ligation)
Our study	Cases, controlled, retrospective	41 (434)	MELD 15.9	Antivitamin K 8.7months	30 (73%) / 11 (27%)	Treated (n=23) 10/6 Untreated (n=11) 0/3	3/5	3 (1 digestive)

Table 2: review of previous reports regarding anticoagulation for PVT in cirrhotic patients

About the curative treatment, data from the literature agreed with our results for total or partial re-permeabilization (rates are 40% and 15% respectively) [11]. Complete re-permeabilization of the portal vein is obtained for almost all patients with treatment duration >1 year [7,12] early discontinued treatment is associated with recurrence in 25% of cases [6]. Some other authors support the fact that 40% of the PVT decreases in size spontaneously. The only predictive factor of re-permeabilization under anticoagulation is, such as found in our study, the early introduction of anticoagulant. The relationship between permeability and complications, described in some series, was not confirmed by comparative studies [13]. Many new oral anticoagulants have been commercialized, Rivaroxaban® proved its efficacy and safety [14,15]. These molecules have many advantages such as easy route of administration and the absence of interaction with the INR and MELD score. Therefore, no continuous monitoring is required. Their disadvantage includes the absence of antidote and frequent drug interactions.

Finally, in light of the above publications and our results, anticoagulant therapy is recommended in the following situations:

1. In patients with advanced cirrhosis who are considered for short-term or medium-term therapy, an anticoagulant treatment, preceded by a preventive treatment of gastrointestinal bleeding should be proposed. An easier access for hepatic transplantation and the improvement of post-operative survival are rational behind this recommendation. The aim is to solve the portal obstruction or at least to limit its extension.
2. In the presence of a PVT extended to the mesenteric vessels with or without signs of intestinal ischemia. The aim of the treatment is to prevent mesenteric infarction.
3. A strong prothrombotic status associated with PVT in a cirrhotic patient is also an indication for anticoagulant therapy alone or in combination with TIPS.

Non tumoral portal vein thrombosis during cirrhosis: Should anticoagulation be proposed?

Conclusions:

Our study showed that untreated PVT has no impact on the progression of cirrhosis neither on the overall survival. the only complication correlated with the portal obstruction was the gastrointestinal hemorrhage with a higher incidence and a more complicated management. From a therapeutic point of view, only the early introduction of anticoagulant therapy was associated with portal re-permeabilization at one year and prolonged anticoagulation was inversely correlated with recurrence of PVT after discontinuation of treatment

Conflict of interest: none

Non tumoral portal vein thrombosis during cirrhosis: Should anticoagulation be proposed?

References:

- [1] Ponziani FR, Zocco MA, Garcovich M, D'Aversa F, Roccarina D, Gasbarrini A. What we should know about portal vein thrombosis in cirrhotic patients: a changing perspective. *World J Gastroenterol*. 2012 ;18(36):5014-20.
- [2] Zocco MA, Di Stasio E, De Cristofaro R, Novi M, Ainora ME, Ponziani F and al. Thrombotic risk factors in patients with liver cirrhosis: correlation with MELD scoring system and portal vein thrombosis development. *J Hepatol*. 2009 ;51(4):682-9.
- [3] Werner KT, Sando S, Carey EJ, Vargas HE, Byrne TJ, Douglas DD and al. Portal vein thrombosis in patients with end stage liver disease awaiting liver transplantation: outcome of anticoagulation. *Dig Dis Sci*. 2013 Jun;58(6):1776-80.
- [4] Villa E, Camma C, Marietta M, Luongo M, Critelli R, Colopi S and al. Enoxaparin prevents portal vein thrombosis and liver decompensation in patients with advanced cirrhosis. *Gastroenterology*. 2012;143(5):1253-60.
- [5] Wanless IR, Liu JJ, Butany J. Role of thrombosis in the pathogenesis of congestive hepatic fibrosis. *Hepatology*. 1995;21(5):1232-7.
- [6] Delgado MG, Seijo S, Yepes I, Achecar L, Catalina MV, Garcia-Criado A and al. Efficacy and safety of anticoagulation on patients with cirrhosis and portal vein thrombosis. *Clin Gastroenterol Hepatol*. 2012;10(7):776-83
- [7] Amitrano L, Guardascione MA, Menchise A, Martino R, Scaglione M, Giovine S and al. Safety and efficacy of anticoagulation therapy with low molecular weight heparin for portal vein thrombosis in patients with liver cirrhosis. *J Clin Gastroenterol*. 2010;44(6):448-5
- [8] Francoz C, Valla D, Durand F. Portal vein thrombosis, cirrhosis, and liver transplantation. *J Hepatol*. 2012 ;57(1):203-12.
- [9] Romero-Gomez M, Gutierrez-Tous R, Delgado-Mije D. Anticoagulation therapy for recent portal vein thrombosis in a patient with liver cirrhosis suffering from variceal rebleeding. *Gastroenterology*. 2002;122(7):2095.
- [10] Huard G, Bilodeau M. Management of anticoagulation for portal vein thrombosis in individuals with cirrhosis: a systematic review. *Int J Hepatol*. 2012; 2012:672986.
- [11] Valla D. Place des anticoagulants au cours de la cirrhose [Internet]. 2014. [cited January 21]. Available from: <http://www.fmcgastro.org/textes-postus/postu-2014/place-des-anticoagulants-au-cours-de-la-cirrhose>
- [12] Senzolo M, T MS, Rossetto V, Burra P, Cillo U, Boccagni P and al. Prospective evaluation of anticoagulation and transjugular intrahepatic portosystemic shunt for the management of portal vein thrombosis in cirrhosis. *Liver Int*. 2012;32(6):919-27
- [13] Luca A, Caruso S, Milazzo M, Marrone G, Mamone G, Crino F and al. Natural course of extrahepatic nonmalignant partial portal vein thrombosis in patients with cirrhosis. *Radiology*. 2012;265(1):124-32.
- [14] Martinez M, Tandra A, Vuppalachchi R. Treatment of acute portal vein thrombosis by nontraditional anticoagulation. *Hepatology*. 2014;60(1):425-6.
- [15] Intagliata NM, Northup PG. Anticoagulant Therapy in Patients with Cirrhosis. *Semin Thromb Hemost*. 2015;41(5):514-9.
- [16] Francoz C, Belghiti J, Vilgrain V, Sommacale D, Paradis V, Condat B, et al. Splanchnic vein thrombosis in candidates for liver transplantation: usefulness of screening and anticoagulation. *Gut*. 2005;54(5):691-7.



Original Article

Traumatic versus non traumatic spinal cord injury: Characteristics and functional outcome in a Tunisian rehabilitation centre

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Publication data:

Submitted: February 5, 2018

Accepted: April 24, 2018

Available Online: June 22, 2018

This article was subject to full peer-review.

Abstract

Background:

Understanding of the underlying mechanisms of Spinal cord injury (SCI) would help in the development of treatment strategies and enhance neurological recovery.

Aim:

The aim of this study was to describe clinical and demographic data of SCI in a physical medicine department and to compare neurological and functional outcome in Traumatic Spinal Cord Injury group (TSCI) and Non Traumatic Spinal Cord Injury group (NTSCI) during two years of follow up.

Materials and methods:

This study was conducted in a physical medicine and rehabilitation department of a tertiary hospital (January 2008-December 2014). Medical records of 177 patients with spinal cord injury (SCI) were reviewed. Two groups were defined: traumatic (TSCI) and non-traumatic (NTSCI) spinal cord injury. Characteristics and functional outcome were analyzed and compared.

Results:

Patients of NT group were significantly older. Most of injuries in both groups had a cervical level. ASIA scale scores and MIF scales were significantly higher in NT group at admission and after two years of follow up. The impairment was more remarkable in this group.

Conclusions:

Our study suggests that non traumatic SCI represent a considerable proportion of SCI rehabilitation admissions. Although different characteristics and injury patterns, functional outcomes maybe comparable to traumatic SCI.

Key words:

spinal cord injury, epidemiology, etiology, rehabilitation

Introduction:

Spinal cord injury (SCI) is an event that results in a disturbance to normal sensory, motor, or autonomic nervous function. It may also lead to several disorders of organ systems, such as respiratory, joint, and urinary system. SCI usually affects also the patient's psychological, and social well-being. The annual global incidence of SCI is 10.4 to 83 cases per million [1]. It may arise from traumatic and non-traumatic causes. In both types of injury, the damage suffered can progress unpredictably. The management of severe cases is difficult due to the lack of guidelines and the high cost of the consensual procedures. Implementing an appropriate prevention strategy require an established knowledge on injury mechanisms, disease pathophysiology, and disability characteristics [2].

Patients and methods:

This is a retrospective study (2008-2014) conducted in the physical medicine and rehabilitation department of Sahloul university hospital, Sousse, Tunisia.

Medical records of patients with SCI admitted were reviewed. Patients were divided into two groups: T group (for TSCI) and NT group (for NTSCI). Patients diagnosed with traumatic Cauda equina syndrome were excluded from group T. Cases of Myelopathy cervicarthrosis majored by a trauma were not included in group NT. The variables studied were associated with the social demographic profile of patients (age, gender, marital status, personal income, social care, occupation and comorbidities). In addition, the cause, type and level of spine injury were specified in the physical examination. Neurological levels of SCI were classified using the American Spinal Injury Association Impairment Scale (AIS)(Appendix1). Functional status at admission and after two years of follow up was assessed by functional independence measure (FIM) (Appendix 2). Concomitant injuries, length of stay (LOS) and different treatment options were recorded.

Recordings were made at the time of admission in rehabilitation department as well as after two years

Statistical analysis was performed using SPSS software (version 17.0). Descriptive statistics were used to represent data as average, range, median and percentages. Ordinal data were expressed as medians, inter-quartile ranges, and percentages. For this normal distribution, Chi-square (χ^2) tests of comparison was applied. Independent t-tests were used to compare parametric variables. A p value < 0.05 was considered as significant.

Results:

During the study, 177 patients with SCI were included. Defined groups were: TSCI (T group; n =108) and NTSC (NT group; n=69). Sociodemographic data is represented in Table1. Patients of NT group were significantly older ($p < 0.001$). however sociodemographic profiles of the two groups were comparable ($p > 0.05$).

Road traffic accidents (RTA) were the main cause of TSCI. Main concomitant injuries observed were brain injuries in 19 patients (17.6%), rib fracture in 13 cases (12.0%) and pelvis fracture in 9.3 % of cases. Regarding NT group, degenerative disease was the main cause of NTSCI including discal hernia and myelopathy in 30.4 % and 20.1% respectively. Mechanisms of SCI in both groups are summarized in table 2.

Regarding baseline evaluation, the cervical level was the most frequently affected region in both groups. AIS scores were significantly higher in NT group at admission ($p < 0.001$). In T group, most of patients were AIS A. However, in NT group, most of lesions were classified as AIS D. Thirteen patients of T group were diagnosed with conus medullaris versus 5 cases in NT group. Patients with TSCI showed a significant lower functional status at admission than NT group (96.0% vs 76% of T and NT group respectively had FIM scores lower than 100/126). Details of baseline evaluation are represented in table 3.

Table1: SCI Sociodemographic characteristics

	T	NT	P
Mean age	34	48.5	<0.001
Gender:			
M	77(71.3%)	37(53.6%)	0.17
F	31(28.7%)	32(46.4%)	
Insurance	54(50%)	59(85%)	0.085
Education:			
Primary	56(52%)	47(69%)	0.19
High	39(37%)	20(29.6%)	
University	13(12%)	2(1.9%)	
Occupation			
Manual	70	52	
Office	22	2	0.06
None	6	2	
Student	10	11	

Table2: Spinal cord injury mechanisms

	Mechanism	n (%)
T	RTA	52(48.1)
	Falls	27(25.0)
	Work accident	15(13.9)
	Diving	6(5.5)
	Violence	4(3.7)
	Suicide attempt	4(3.7)
	NT	Degenerative disease
	Neoplastic disease	14(20.3)
	Infection	13(18.8)
	Vascular disease	4(5.8)
	Inflammatory disease	3(4.3)

Regarding the operative management; surgical decompression was earlier in T group. Medical management of SCI depended on the etiology. It included antibiotics (infectious spondylodiscitis), anti-tubercular agents and corticosteroids (tuberculosis), embolization, chemotherapy, radiation (neoplastic diseases). Regarding urinary dysfunctions, treatment strategies were adapted to bladder disorder types.

Treatment of overactive bladder was based on anticholinergic drugs and self-intermittent catheterization (76.9% and 44.9% of T and NT group, respectively). Five patients in T group had suprapubic catheter for urinary retention in case of urethral trauma or penile sores.

Table3: Baseline evaluation

Admission	T	NT	P
Cervical level	46	32	
Thoracic level	34	30	
Lumbar Level	12	22	
Multifocal lesions	16	49	<0.001
ASIA « A / B »	64	13	
ASIA « C »	21	24	
ASIA « D / E »	10	27	
Urinary incontinence	78	16	<0.001
Anal incontinence	58	15	<0.001
Mean FIM score	52.7	78.8	<0.001
DOS(days)	40	24	<0.001
Time to surgery	7	180	<0.001
Surgical procedure	n=92	n=48	<0.001
Laminectomy	8	19	
Laminectomy fixation	70	8	
Reduction	4	0	
Discectomy	0	8	
Excision	0	14	

Requirement of assistance devices was significantly higher in T group (92.6% versus 62.3% in NT group; P <0.001).

Readmissions in rehabilitation department characteristics were analyzed and compared between the two groups. The rate of readmission was significantly higher in T group (33.6% of T group, 12.8 % of NT group; P=0.01).

Characteristics of SCI readmissions are summarized in table 4.

Table4: Characteristics of readmissions in SCI

Readmission	T	NT
%	33.6	12.9
Average time to readmission	432	404
Mean inpatient days	19	7
FIM score	73/126	95/126
% Scheduled /complications	55.6/44.4	70 /30

A variety of complications was diagnosed during the follow up of patients with clear difference between the two groups. In fact, all types of complications were significantly more frequent in T group. However, the comparative study could not be independent from postoperative courses factors. Managed complications are detailed in table 5.

Table 5: Major complications

Complications	T (n)	NT(n)	P
Spasticity	44	22	0.008
Neuropathy	41	13	0.05
Urinary tract infection	63	6	< 0.001
Sepsis	35	2	< 0.001
Thrombosis	14	2	0.03
Pressure ulcer	55	8	< 0.001
Osteoma	23	0	< 0.001
Constipation	40	4	< 0.001

ASIA scale scores and MIF scales were significantly higher in NT group at admission and after two years of follow up as compare with T group. Details of final evaluation are represented in table 6.

Table 6: final assessment

Final assessment (n)	T	NT	P
ASIA A/ B	53	6	< 0.001
C/D/E	42	57	
Non walkers	66	7	< 0.001
Walkers	42	62	
Spontaneous urination	25	42	0.05
Urinary symptoms	27	7	< 0.001
Mean FIM score	87.5	98.6	0.05
Gain MIF	27.02	18.27	0.04

On the basis of the present findings neurological and functional impairment was higher in T group as compare with NT group, not only at admission in rehabilitation department, but also after two years of follow.

Discussion:

Spinal cord injury is a devastating condition. In addition to organic and psychological disorders; SCI management represents substantial financial challenge on patients and society [3,4]. A comprehensive study of the leading factors and the pathological behaviour of SCI has simplified the management and improved the prognosis. Trauma contributes to the largest proportion of SCI. The demographic data, etiology, and functional outcome have been well codified for traumatic SCI in the previous published literature [5]. Male predominance is usually noticed for traumatic SCI. In our study, patients in T group were male in 71.3% of cases. This was concordant with earlier studies results [5,6]. Regarding non-traumatic SCI; Citterio and al have also reported a male predominance (58%) [7]. However, most of the other authors found a female predominance independent from the etiology [6-8]. Traumatic SCI affect more young adults. In our study, mean age of patients in T group was 34 years (21-30). However a remarkable increase of traumatic SCI incidence is noticed in older population [9,10]. This can be explained by the progress of demographic assessment and a higher accident rate beyond the age of 65 [11].

In our study, patients of NT group were significantly older (49 years vs 34 years). This finding is widely described in the literature [4,7,11].

Moutquin and al found a significant higher rate of associated comorbidities in non-traumatic SCI [12]. That was the case of diabetes (6%), cancer (57%) and chronic obstructive pulmonary disease (2%).

As previously reported; the most two common causes of traumatic SCI are Road traffic accidents and falls (respective incidence are 48.1% and 25.0%) [11,12,13]. However, in non-traumatic SCI; degenerative diseases remain the most common cause (50.7%) [13].

Most of injuries in both the groups are located in a cervical level. Gupta and al reported most frequent thoracic and lumber injuries especially in non-traumatic SCI [14].

Regarding AIS scale at admission, we found a significant difference between the two groups. The majority of the T group patients (61.1%) presented with an AIS "A", however in the NT group most of patient's AIS were "C" or "D". Our results are similar to those described in the literature. Table 6 summarizes recent works dealing with this subject.

Recent epidemiological studies reported that patients diagnosed with traumatic SCI have more complete lesions. In our study, comparable findings could be seen (61.1% of the T group had complete lesions compared to 11.5% in the NT group, P <0.001). This can be explained by the high velocity and sudden mechanisms in traumatic injuries [12,14].

Length of stay in rehabilitation department is considered as indicator in the outcome assessment. A significant difference was found between the groups in our study.

Patients in NT group had a shorter rehabilitation than those in T group (24 days vs 40 days). Several factors may contribute to a longer rehabilitation for traumatic SCI patients. These factors include the treatment of concomitant injuries and the management of non-specific complications which are more frequently observed [15].

Even consensual and well codified; the management of SCI is still difficult. A multidisciplinary team management approach is mandatory in the rehabilitation of SCI. In addition to the managing physicians; the team should include by a physiotherapist, a dietician, and a psychologist. Training and education of the patient's family improve always the treatment outcome [16].

Table 6: Literature review

		T Group					NT Group				
		total	ASIAA	ASIAB	ASIAC	ASIAD	total	ASIAA	ASIAB	ASIAC	ASIA D
Current study	2018	108	61.1%	6.3%	22.1%	10.5%	69	14.1%	6.3%	38%	40.6%
Anghelescu [22]	2016	346	62.7%	13.9%	13.9%	9.5%	87	24.13%	19.54%	14.94%	41.33%
Derakhshanrad [23]	2016	1137	53.5%	18.7%	17.6%	9.6%					
Rinkaewkan [24]	2015	85	57.6%	12.4%	16.4%	7.5%	115	22.4%	16.9%	21.4%	36.3%
Noreau [6]	2014	1137	42.8%	9.1%	18.3%	15.0%	412	19.9%	3.2%	22.8%	35.9%
Shin[10]	2013	481	51.4%	15.2%	18.1%	15.4%	148	12.2%	6.8%	30.4%	50.7%
Scivoletto [25]	2011	144	51.3%	8.3%	27.8%	12.5%	236	20.3%	7.2%	43.6%	28.8%
Gupta [14]	2008	38	50%	13.1%	13.1%	5.2%	38	28.9%	15.7%	23.6%	31.5

Early inpatient rehabilitation program aims to teach the patient the daily tasks achievement. This may include the wheelchair use skills, bowel and bladder management, and skin care. The prevention and the management of late complications is considerable part of the treatment.

Urinary tract disorders, pressure ulcers, deep venous thrombosis, spasticity, and depression are frequent and delay patient autonomy recuperation [17].

The use of specific scores simplify the assessment and make from physical examination findings a measurable entity that could be followed up. In our study; FIM scores at the time of admission and after two years were recorded and used as functional outcome measurement tool. The mean MIF was 52.7/126 in T group versus 78 in NT group ($P < 0.001$). The significant difference in traumatic SCI patients is attested by all the authors and highlights the severity of pathological lesions as well as the delayed healing in these cases. [18-20]. According to Ditunno; most asked questions asked by patients and their relatives are related to motility function "Will i be able to walk?" [20]. Social and psychological assistance is capital during the walking recovery period [21].

In our study, 38.9% of T group and 89.9% of NT group were walkers. These patients were initially classified AIS "C" or "D". Actually the chance of walking recovery after a SCI can be predicted from the admission time. Patients with complete lesions have very limited chance for full recovery. The prognosis is better for partial lesions in young patients and in the absence of severe associated comorbidity or late complications. The prevention and early diagnosis improve the treatment results in both types of SCI [22].

The WHO recommended three levels prevention strategy to improve functional prognosis of SCI. Primary consist in the control of the leading factors such as road traffic accident for trauma SCI. Secondary prevention aims to ensure an early diagnosis of the injury and an efficient management (complete initial neurological examination, quick screening and early decompressive surgery).

Tertiary prevention aims to minimize durable side effects and to improve patient's re-integration [23-25].

Conclusions:

Understanding of the underlying mechanisms and the control of the leading factors would help in the development of SCI treatment strategies and enhance neurological recovery.

This report corroborates many previously evident facts; especially the difficulty of the management of traumatic cases. However it showed a comparable treatment results in both types of lesions in an area of very high accidents rate. The rehabilitation is as important as the first given care. It should be driven in a well codified scientific way to ensure a maximum of recuperation. A larger study may allow to avoid statistical bias and give more objective results.

Conflict of interest: none

References:

- [1] Osterthun, R, Post M W M, Van Asbeck F W A. Characteristics, Length of Stay and Functional Outcome of Patients with Spinal Cord Injury in Dutch and Flemish Rehabilitation Centres. *Spinal Cord*.2009; 47(4): 339-44.
- [2] Guilcher SJ, Munce SE, Couris CM, Fung K, Craven BC, Verrier M, Jaglal SB. Health care utilization in non-traumatic and traumatic spinal cord injury: A population-based study. *Spinal Cord*.2010 ;48(1): 45-50.
- [3] St Andre JR, Smith BM, Stroupe KT, Burns SP, Evans CT, Ripley DC et al. A comparison of costs and health care utilization for veterans with traumatic and nontraumatic spinal cord injury. *Top Spinal Cord Inj Rehabil*. 2011;16(4):27-42.
- [4] Yang R, Guo L, Wang P, Huang L, Tang Y, Wang W, et al. Epidemiology of spinal cord injuries and risk factors for complete injuries in Guangdong, China: a retrospective study. *PLoS One*. 2014; 9(1): e84733.
- [5] Zárate-Kalfópulos, B, Jiménez-González A, Reyes-Sánchez R, Robles-Ortiz R, Cabrera-Aldana E, Rosales-Olivarez L. Demographic and clinical characteristics of patients with spinal cord injury: a single hospital-based study. *Spinal Cord*.2016; 54(11): 1016-19.
- [6] Noreau L, Noonan V, Cobb J, Leblond J, Dumont F. Spinal Cord Injury Community Survey: A national, comprehensive study to portray the lives of Canadians with spinal cord injury. *Topics in Spinal Cord Injury Rehabilitation*.2014; 20(4): 249-64.
- [7] Citterio A, Franceschini M, L Spizzichino L, Reggio A, Rossi B, Stampacchia G. Nontraumatic spinal cord injury: An Italian Survey. *Archives of Physical Medicine and Rehabilitation*.2004; 85(9): 1483-87.
- [8] New P W. Functional outcomes and disability after nontraumatic spinal cord injury rehabilitation: Results from a retrospective study. *Archives of Physical Medicine and Rehabilitation*.2005; 86(2): 250-61.
- [9] Shihao Z, Wadhwa R, Haydel J, Toms J, Johnson K, Guthikonda B. Spine and Spinal Cord Trauma. *Neurologic Clinics*.2013; 31(1): 183-206.
- [10] Shin, J C, Kim DH, Yu SJ, Hea Eun Yang HE, Yoon SY. Epidemiologic Change of Patients with spinal cord injury. *Annals of Rehabilitation Medicine*.2013; 37(1): 50-56.
- [11] McKinley WO, Seel RT, Gadi RK, Tewksbury MA. Nontraumatic vs. traumatic spinal cord injury. *Am J Phys Med Rehabil* .2001 ; 80 : 693-99.
- [12] Moutquin J M, Larouche K, Mayot M H, Rossignol M. Lésions médullaires traumatiques et non-traumatiques : analyse comparative des caractéristiques et de l'organisation des soins et services de réadaptation au Québec : l'Institut national d'excellence en santé et en services sociaux ; Fev 2013. *Rapport ETMIS*. 2013 ;9(1).
- [13] Kay E, Deutsch A, Chen D, Larry Manheim L, Rowles D. Effects of etiology on inpatient rehabilitation outcomes in 65- to 74-year-old patients with incomplete paraplegia from a nontraumatic spinal cord injury. *PM&R*.2010; 2(6): 504-13.
- [14] Gupta A, Taly AB, Srivastava A, Vishal S, Murali T. Traumatic vs non-traumatic spinal cord lesions: Comparison of neurological and functional outcome after in-patient rehabilitation. *Spinal Cord*.2008; 46(7): 482-87.
- [15] Majdan M, Brazinova A, Mauritz W. Epidemiology of traumatic spinal cord injuries in Austria 2002-2012. *Eur Spine J*.2015; 25: 62-73.
- [16] Bauchet L, Lonjon N, Perrin FE, Gilbert C, Privat A, Fatta C. Strategies for spinal cord repair after injury: A review of the literature and information. *Annals of Physical and Rehabilitation Medicine*.2009; 52(4): 330-51.
- [17] Perrouin-Verbe B. Rehabilitation of spinal cord injury patients. *Bulletin De l'Academie Nationale De Medecine*.2005; 189(6): 1159-74.
- [18] Kemal N, Yazmalar L, Sah V, Aydin A, Ones K. Rehabilitation of Spinal Cord Injuries. *World J Ortho*.2015; 6(1): 8-16.
- [19] Lofvenmark I, Norrbrink C, Nilsson-Wikmar L, Hultling C, Chakamdinakira S, Hasselberg M. Traumatic spinal cord injury in Botswana: characteristics, aetiology and mortality. *Spinal Cord*. 2015; 53: 150-54.
- [20] Ditunno JF. Predicting recovery after spinal cord injury: A rehabilitation imperative. *Archives of Physical Medicine and Rehabilitation*.1999; 80(4): 361-64.
- [21] Scivoletto G, Tamburella F, Laurenza L, Torre M, Molinari M. Who is going to walk? A review of the factors influencing walking recovery after spinal cord injury. *Front Hum Neurosci*.2014; 8:141.

[22] Anghelescu A, Onose LV, Popescu C, Andone I, Octaviana DC, Magdoiu AM, et al. Evolution of traumatic spinal cord injury in patients with ankylosing spondylitis, in a Romanian rehabilitation clinic. *Spinal Cord Ser Cases*. 2016; 2:16001.

[23] Derakhshanrad N, Yekaninejad M, Vosoughi F, Fazel FS, Saberi H. Epidemiological study of traumatic spinal cord injuries: experience from a specialized spine center in Iran. *Spinal cord*. 2016; 54:901-7.

[24] Rinkaewkan P, Kuptniratsaikul V. The effectiveness of inpatients rehabilitation for spinal cord patients in Siriraj Hospital. *Spinal Cord*.2015; 53(8): 591-97.

[25] Scivoletto G, Farchi S, Laurenza L, Molinari M. Traumatic and non-traumatic spinal cord lesions: An Italian comparison of neurological and functional outcomes. *Spinal Cord*.2011; 49(3): 391-96.

Appendix 1

ASIA INTERNATIONAL STANDARDS FOR NEUROLOGICAL CLASSIFICATION OF SPINAL CORD INJURY (ISNCSCI) **ISCS**

Patient Name _____ Date/Time of Exam _____
 Examiner Name _____ Signature _____

RIGHT

MOTOR KEY MUSCLES

Elbow flexors C5
 Wrist extensors C6
 Elbow extensors C7
 Finger flexors C8
 Finger abductors (5th finger) T1

Elbow flexors L2
 Knee extensors L3
 Ankle dorsiflexors L4
 Long toe extensors L5
 Ankle plantar flexors S1

(VAC) Voluntary anal contraction (Yes/No)

RIGHT TOTALS (MAXIMUM) (50) (56) (56)

MOTOR SUBSCORES
 UER + UEL = UEMS TOTAL (28) (28)
 LER + LEL = LEMS TOTAL (28) (28)

Key Sensory Points

SENSORY KEY SENSORY POINTS
 Light Touch (LT) P-Pink (PP)

C2
C3
C4
C5
C6
C7
C8
T1
T2
T3
T4
T5
T6
T7
T8
T9
T10
T11
T12
L1
L2
L3
L4
L5
S1
S2
S3
S4-5

SENSORY SUBSCORES
 LTR + LTL = LT TOTAL (112) (112)
 PPR + PPL = PP TOTAL (50) (50)

LEFT

MOTOR KEY MUSCLES

Elbow flexors C5
 Wrist extensors C6
 Elbow extensors C7
 Finger flexors C8
 Finger abductors (5th finger) T1

Elbow flexors L2
 Knee extensors L3
 Ankle dorsiflexors L4
 Long toe extensors L5
 Ankle plantar flexors S1

(DAP) Deep anal pressure (Yes/No)

LEFT TOTALS (MAXIMUM) (50) (56) (56)

MOTOR SUBSCORES
 UEL + UER = UEMS TOTAL (28) (28)
 LEL + LER = LEMS TOTAL (28) (28)

NEUROLOGICAL LEVELS (Steps 1, 2 for classification as on muscle)

1. SENSORY

R	L
<input type="text"/>	<input type="text"/>

2. MOTOR

R	L
<input type="text"/>	<input type="text"/>

3. NEUROLOGICAL LEVEL OF INJURY (NLI)

4. COMPLETE OR INCOMPLETE? Incomplete = Any sensory or motor function in S4-5

5. ASIA IMPAIRMENT SCALE (AIS)

ZONE OF PARTIAL PRESERVATION (In complete injuries only)
 Sensory

R	L
<input type="text"/>	<input type="text"/>

 Motor

R	L
<input type="text"/>	<input type="text"/>

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Appendix 2

FIM™ instrument

LEVELS	7 Complete Independence (Timely, Safely) 6 Modified Independence (Device)	NO HELPER		
	Modified Dependence 5 Supervision (Subject = 100%+) 4 Minimal Assist (Subject = 75%+) 3 Moderate Assist (Subject = 50%+) Complete Dependence 2 Maximal Assist (Subject = 25%+) 1 Total Assist (Subject = less than 25%)	HELPER		
		ADMISSION	DISCHARGE	FOLLOW-UP
Self-Care				
A. Eating		<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
B. Grooming		<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
C. Bathing		<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
D. Dressing - Upper Body		<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
E. Dressing - Lower Body		<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
F. Toileting		<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Sphincter Control				
G. Bladder Management		<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
H. Bowel Management		<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Transfers				
I. Bed, Chair, Wheelchair		<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
J. Toilet		<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
K. Tub, Shower		<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Locomotion				
L. Walk/Wheelchair		<input type="checkbox"/> <input type="checkbox"/> W Walk C Wheelchair B Both	<input type="checkbox"/> <input type="checkbox"/> W Walk C Wheelchair B Both	<input type="checkbox"/> <input type="checkbox"/> W Walk C Wheelchair B Both
M. Stairs		<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Motor Subtotal Score		<input type="text"/>	<input type="text"/>	<input type="text"/>
Communication				
N. Comprehension		<input type="checkbox"/> <input type="checkbox"/> A Auditory V Visual B Both	<input type="checkbox"/> <input type="checkbox"/> A Auditory V Visual B Both	<input type="checkbox"/> <input type="checkbox"/> A Auditory V Visual B Both
O. Expression		<input type="checkbox"/> <input type="checkbox"/> V Vocal N Nonvocal B Both	<input type="checkbox"/> <input type="checkbox"/> V Vocal N Nonvocal B Both	<input type="checkbox"/> <input type="checkbox"/> V Vocal N Nonvocal B Both
Social Cognition				
P. Social Interaction		<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Q. Problem Solving		<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
R. Memory		<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Cognitive Subtotal Score		<input type="text"/>	<input type="text"/>	<input type="text"/>
TOTAL FIM Score		<input type="text"/>	<input type="text"/>	<input type="text"/>
NOTE: Leave no blanks. Enter 1 if patient not testable due to risk				

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Case report

L'hépatothorax : Une complication rare des plaies diaphragmatiques droites.

The hepatothorax: An unusual complication of right sided diaphragmatic injuries.

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Publication data:

Submitted: February 24 ,2018

Accepted: April 17,2018

Available Online: June 22 ,2018

This article was subject to full
peer-review.

Abstract

Right diaphragmatic post-traumatic rupture with liver herniation is an extremely rare condition. The diagnosis is mainly radiological and the rupture may go unnoticed in the acute setting.

Depending on the size of the right diaphragmatic defect, the initial herniation can be partial and the total hepatothorax is established progressively.

The diagnosis may be delayed and made with the onset of the first symptoms. Hepatothorax leads usually to severe right lung atelectasis with respiratory and cardiac impairment.

Definitive treatment consists in surgical repair of the diaphragm. We present hereby the case of an hepatothorax diagnosed 4 years after a penetrating thoracoabdominal trauma.

Key words: Trauma, diaphragmatic rupture, hepatothorax.

Introduction :

La rupture de la coupole diaphragmatique droite est un incident rare résultant d'un traumatisme de haute énergie. Elle est plus fréquente en cas de plaies thoraco-abdominales pénétrantes mais pouvant aussi résulter de traumatismes fermés [1]. La hernie du foie dans le thorax est exceptionnelle et souvent progressive. Le diagnostic tardif majore la morbidité liée aux conséquences respiratoires et hémodynamiques [2].

Observation:

Il s'agit d'une dame âgée de 37 ans ayant été victime d'une agression par une arme blanche 4 ans auparavant occasionnant une plaie de 3 cm dans la face postérieure du thorax en regard du 7^{ème} espace intercostal droit. La radiographie du thorax était sans anomalies et la plaie était suturée.

La patiente a reconulté en urgence pour une dyspnée d'installation brutale associée à des douleurs basithoraciques droites. L'examen a noté une polypnée et une diminution des murmures vésiculaires droits.

La radiographie du thorax a montré une opacité basithoracique droite effaçant la coupole diaphragmatique (figure1). Le scanner thoraco-abdominal pratiqué a objectivé une hernie diaphragmatique droite à collet large de 9 cm contenant le foie et l'angle colique droit (Figure2).

La chirurgie a été indiquée. A l'exploration par voie sous costale droite ; le foie droit ainsi que l'angle colique droit étaient ascensionnés en intra thoracique à travers une brèche diaphragmatique droite faisant 12 cm de grand axe.

Une réduction du contenu en intra-abdominal a été réalisée avec réparation de la brèche diaphragmatique par une plaque suivie de la mise en place d'un drain thoracique. Les suites opératoires ont été marquées par l'apparition d'une pleuro-pneumopathie droite qui ayant favorablement évoluée sous antibiothérapie.

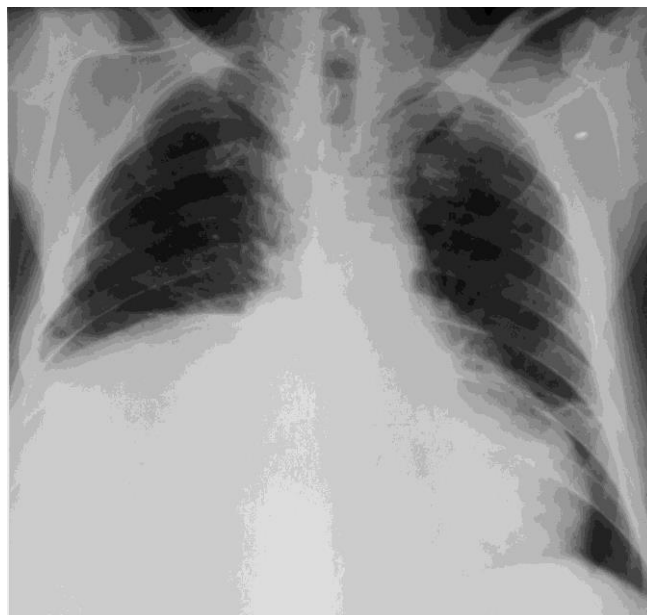


Figure 1 : une opacité basithoracique droite de tonalité hydrique effaçant la coupole diaphragmatique droite



Figure 2 : Coupes sagittales d'une TDM Thoraco-abdominale objectivant une hernie diaphragmatique droite de 9 cm de diamètre contenant le foie et l'angle colique droit.

Discussion :

Les ruptures des coupes diaphragmatiques sont présentes dans 0,8 à 5% des traumatismes thoraco-abdominaux. L'atteinte diaphragmatique droite est trois fois moins fréquente que celle à gauche [3]. Une hernie secondaire des viscères abdominaux dans la cavité pleurale n'est observée que dans 19% des cas [4]. L'atteinte diaphragmatique est souvent asymptomatique et peut passer inaperçue [4].

La lésion du diaphragme est secondaire à une plaie pénétrante 10 à 15% des cas. Les plaies sont souvent initialement de petite taille pouvant s'agrandir progressivement et causer la hernie viscérale. Les lésions droites sont plus rares en cas de traumatisme fermé et souvent le résultat d'un impact à haute énergie grevé d'une lourde morbidité [5].

L'hépatothorax est une complication grave de la rupture diaphragmatique droite. Son évolution est souvent progressive et insidieuse. La symptomatologie respiratoire ou digestive reste non spécifique et l'hépatothorax est découvert fortuitement sur l'imagerie dans 18 à 24% des cas [6].

Les circonstances de découverte sont parfois aiguës surtout en cas d'évolution longues. Il peut s'agir d'une détresse respiratoire aiguë ; une défaillance cardiaque droite ou encore une occlusion intestinale aiguë en cas de composante digestive herniée associée [7].

La suspicion diagnostique conduit à la demande d'une radiographie du thorax. Cet examen confirme rarement le diagnostic surtout dans un contexte d'urgence. La sensibilité n'est que de 27 à 62% pour les lésions diaphragmatiques gauches et 18 à 33% pour les lésions droites souvent colmatées par le foie. Les signes radiologiques sont indirects tel que l'ascension de la coupole ; l'épanchement pleural ; ou plus rarement une déviation controlatérale des structures médiastinales [8].

La tomographie assistée par ordinateur (TDM) est l'examen de référence pour établir le diagnostic positif de la lésion diaphragmatique et pour évaluer les lésions associées. Elle a une sensibilité de 71-100% et visualise directement le défaut musculaire [9].

Le traitement de l'hépatothorax est chirurgical. L'objectif de cette chirurgie est de repositionner le foie ; de réduire les viscères herniés dans la cavité abdominale ; et de réparer le défaut afin d'éviter la récurrence. La laparotomie exploratoire reste toujours envisageable en cas d'urgence compte tenu du taux élevé de lésions abdominales associées. Les hernies diaphragmatiques constituées peuvent être réparées par voie thoracique ou abdominale. Cependant, une thoracotomie est préférable pour les grandes hernies [10]. L'utilisation de matériaux synthétiques est recommandée dans les cas de perte significative de substance diaphragmatique. La morbidité spécifique semble avoir diminué avec les nouvelles générations de plaques.

La morbi-mortalité des lésions diaphragmatiques dépend essentiellement de la gravité des lésions associées. Son taux varie de 18 à 40%. L'hépatothorax bien qu'impressionnant parfois ; n'a pas d'impact direct sur le pronostic du traumatisme en cause [11,12].

Conclusion :

Notre observation met en relief l'importance du diagnostic précoce de plaies diaphragmatiques droites et l'indication indiscutable d'une imagerie de l'abdomen pour toute plaie située en de ça du 5^{ème} espace intercostal. En effet les complications des plaies thoracoabdominales sont fréquentes et peuvent nécessiter une chirurgie lourde en cas de prise en charge tardive.

Conflict of interest: none

References:

- [1]Rashid F, Chakrabarty MM, Singh R, Iftikhar SY. A review on delayed presentation of diaphragmatic rupture. *World J Emerg Surg.* 2009; 4:32.
- [2]Baek SJ, Kim J, Lee SH. Hepatothorax due to a right diaphragmatic rupture related to duodenal ulcer perforation. *World journal of gastroenterology.* 2012; 18:5649-52.
- [3]Kozak O, Mentés O, Harlak A, Yigit T, Kilbas Z, Aslan I, et al. Late presentation of blunt right diaphragmatic rupture (hepatic hernia). *Am J Emerg Med.* 2008;26(5):638. e3-5.
- [4]Lugarinho-Monteiro MT, Pereira L, Seco C. Chronic hepatothorax due to right diaphragmatic rupture: an anesthetic challenge in a rare case. *Rev Bras Anesthesiol.*2018;68(2):190-93.
- [5]Gao J, Du D, Li H, Liu C, Liang S, Xiao Q, et al. Traumatic diaphragmatic rupture with combined thoracoabdominal injuries: Difference between penetrating and blunt injuries. *Chinese Journal of Traumatology.*2015;18:21-26.
- [6]Robustelli U, Noschese G, Armellino MF, Gagliardi N, Festa P, Scardi F, Catuogno F. Diaphragmatic rupture with intrathoracic hepatic dislocation. Two cases report. *G Chir.* 2009;30(6):294-98.
- [7]Topuz M, Cihat Ozek M. Right ventricle collapse secondary to hepatothorax caused by diaphragm rupture due to blunt trauma. *Ulus Travma Acil Cerrahi Derg.* 2014; 20(6) :463-65.
- [8]Hanna WC, Ferri LE, Fata P, Razeq T, Mulder DS. The current status of traumatic diaphragmatic injury: lessons learned from 105 patients over 13 years. *The Annals of thoracic surgery.* 2008;85(3):1044-48.
- [9]Turmak M, Deniz MA, Ozmen CA, Aslan A. Evaluation of the multi-slice computed tomography outcomes in diaphragmatic injuries related to penetrating and blunt trauma. *Clinical Imaging.*2018; 47:65-73.
- [10]Li Tserng T, Gatmaitan MB. Laparoscopic approach to the management of penetrating traumatic diaphragmatic injury. *Trauma case reports.*2017;10:4-11.
- [11]Kastanakis M, Anyfantakis D, Kokkinos I, Petrakis G, Bobolakis E. Delayed post-traumatic diaphragmatic rupture complicated by total hepato-thorax: a case report. *Int J Surg Case Rep.* 2013;4(6):537-39.
- [12]Vilallonga R, Pastor V, Alvarez L, Charco R, Armengol M, Navarro S. Right-sided diaphragmatic rupture after blunt trauma. A unusual entity. *World J Emerg Surg.* 2011; 6:3.



Case report

Aneurysmal Bone Cyst of D2 in a Child complicated with paraplegia.

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Publication data:

Submitted: May 9,2018

Accepted: June 10,2018

Available Online: June 22,2018

This article was subject to full peer-review.

Abstract

Aneurysmal bone cysts (ABCs) are benign osteolytic lesion representing 15% of all primary spine tumors. We report a case of a 9-year-old girl who had an ABCs localized in D2.

Symptoms involved back pain and paraplegia. Radiology investigations showed osteolysis of D2 and anterolisthesis of C7 and D1.

The patient had a posterior decompression and laminectomy of D2, D3 and D4 without neurological improvement. Surgical biopsy confirmed the diagnosis.

Computed tomography scan showed tumor remnants. An embolization of the tumor and an anterior liberation associated with bone graft were performed.

The result was a spectacular neurological improvement with disappearing of all neurological symptoms. Radiology investigations follow up showed only spine instability but no residual tumor.

Key words: Tumor; Cyst; Bone; Spine.

Introduction:

Aneurysmal bone cysts (ABCs) are benign and locally aggressive osteolytic lesions. They represent 1.4% of primary bone tumors; 9.1% of all bone tumors; and only 15% of primary spine tumors [1]. These lesions occur either in thoraco-lumbar or cervical spine. These locations are problematic due to the frequency of spine instability. The reconstructive surgery is always challenging [2].

Case presentation:

Our case is a 9-year-old girl with no previous medical history. She first presented with back pain and progressive paraplegia. There was no history of trauma or fever. Physical examination showed neck stiffness, flaccid paraplegia and abolition of tendon reflexes. There was no sensory neither sphincterian disorders.

The X-ray showed osteolysis of D2 and anterolisthesis of C7 and D1. The CT scan showed an osteolytic lesion in the posterior arch of D2 vertebrae (Figure 1). The lesion was compressing the spinal cord. The MRI showed hypersignal of the vertebrae D2 in T1 and T2 weighted sequences with vascular enhancement and medullary compression (Figure 2).

The patient first had a posterior decompression and laminectomy of D2, D3 and D4, stabilized by a halo cast. A surgical biopsy was also performed, and it was in favor of ABC (Figure 3).

There was no neurological improvement after the surgery. Spasticity of lower appeared one month later. A CT scan showed tumor remnants in the medullar cavity (Figure 4). An embolization of the tumor was performed (figure 5), associated with anterior liberation, bone graft and stabilization with halo cast for three months.

6 months later, the neurological defect has disappeared. The muscular testing was normal.

CT scan follow up showed that there is no recurrence of the tumor (Figure 6). At 9 years after the surgery, the patient is living a normal life and has no complaints. No recurrence of the tumor was observed.

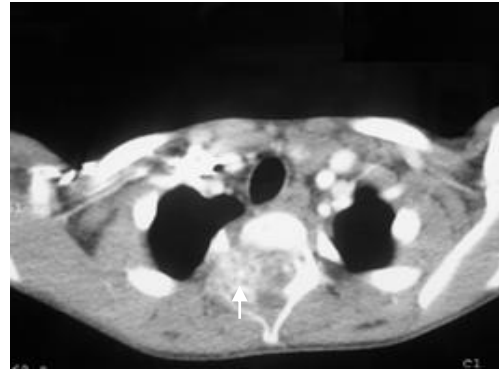
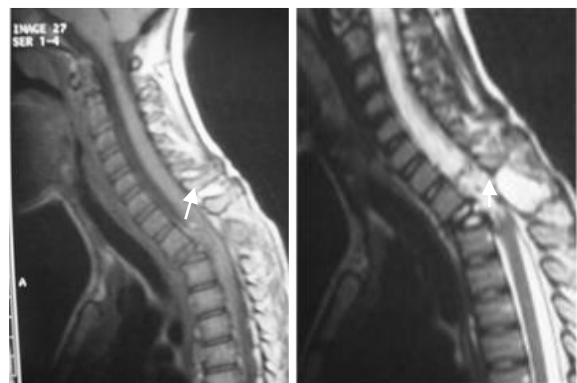


Figure 1: CT axial images show an expansive and lytic lesion in the vertebral body, right pedicle, transverse and spinous process of D2 which enhance after contrast injection with moderate canal compromise



(a)



(b)

Figure 2: a) T1, T2 tumoral appearances b) T2 with injection of gadolinium contrast: hypersignal of the vertebrae D2 in T1 and T2 weighted sequences with vascular enhancement and medullary compression

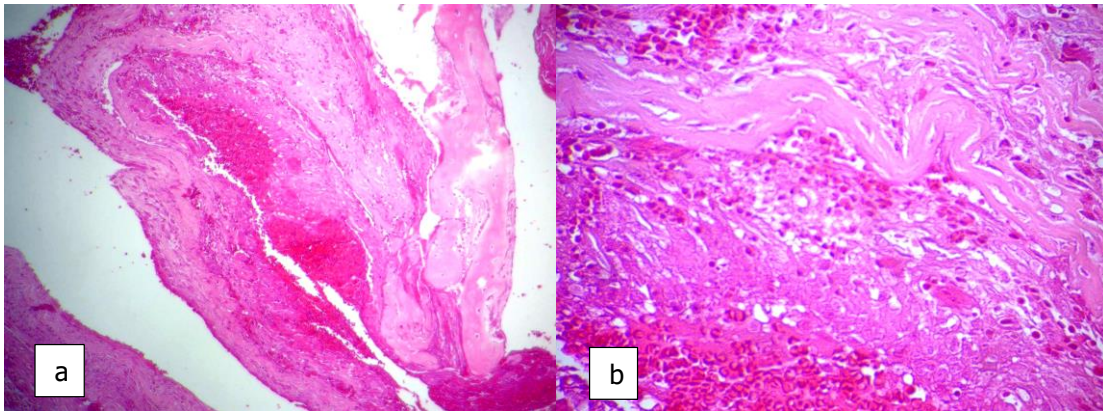


Figure 3: Histological study a: cavities separated by septa of various thickness. These cavities were filled with red blood cells. b: Thin osteoid hyaline bands close of the borders of the cavities

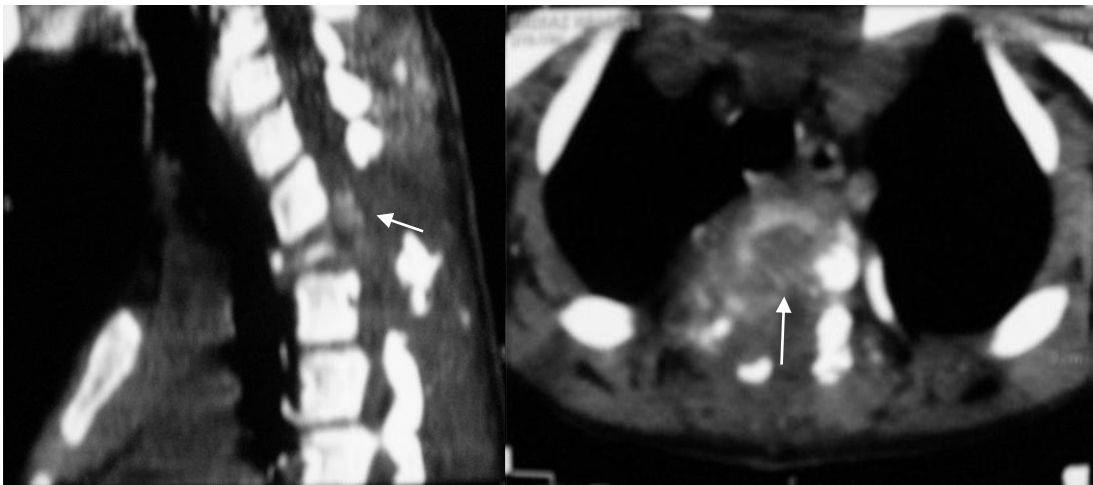


Figure 4: Tumor remnants in the medullary cavity

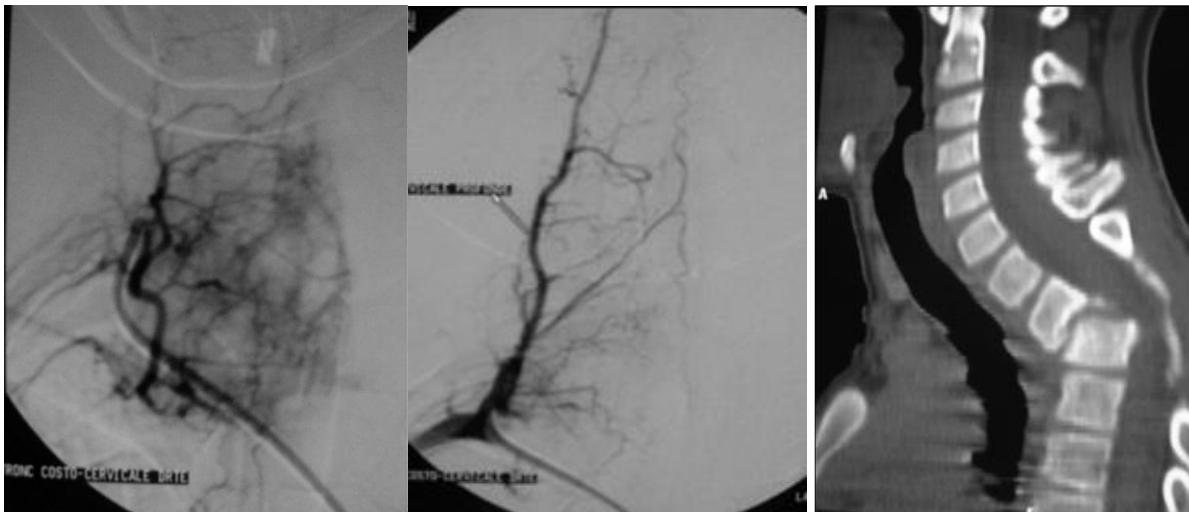


Figure 5: Pre-operative embolization of the tumor

Figure 6: follow up CT scan at 6 months

Discussion:

ABC was first considered as a variety of giant cell tumors, then as an isolated tumor-like bone dystrophy. But its real nature is still unknown. ABC was first described in 1942 by Jaffe and Lichtenstein [3]. This terminology was, since then, world widely used, even ABCs are neither cysts, nor aneurysms. It is a tumor-like lesion and can be individualized in two forms: primitive ABC, which is an independent entity (70% of the cases), and secondary ABC (30% of the cases), which is a reactional and developed on a preexisting lesion [4].

Pathogenic mechanisms of ABC are still discussable. Recent researches, particularly genetic and immuno-histochemical, are tending to prove that ABC is more a tumor than a tumor-like lesion. However, pathogenesis of ABC is probably multifactorial. In an epidemiological multicentric study about 411 children with primitive ABC, femur (22%), tibia (17%), spine (15%), humerus (10%), pelvis (9%) and fibula (9%) were the most frequent localizations [5]. Lumbar region is the most frequently affected in the spine. ABC is first found in the posterior arc (40% of unique lesions), then fills the vertebral body in the front side via the pedicle, adjacent vertebrae in the up and downside via the articular processes, and the ribs laterally. Isolated localization in the body of the vertebrae is very rare [6]. ABC can present in a form of stiff and painful scoliosis, with an important functional disability. A neurological syndrome is found in 50% of the cases. Neurological signs, like radicular compression, are either progressive due to the growth of the size of the tumor, or brutal due to the damage caused in the vertebral body. X-rays and CT scan findings depend on the stage of ABC. During the osteolytic stage, radiological images are usually zones of eccentric bone depletion. The active growth stage shows a sub-periosteal eruption. Healing stage is characterized by progressive calcification and ossification of the cyst [7].

MRI can localize the lesion and its extension, confirm its sub-periosteal situation and analyze the surrounding vessels and noble structures. Some images are very revealing, like a well-limited expansive bone lesion, a decrease of signal in T1 associated with increase of the signal in T2 (liquid compound), a peripheral border of low signal enhanced by the injection of gadolinium, multiple small cavities confined by septa and the presence of liquid-liquid levels.

Association of X-ray and MRI is helpful for the diagnosis of ABC, but biopsy is mandatory before the treatment for histological confirmation [8].

Selective arterial embolization, used as the only treatment, or during the pre-operative phase (an uncontrollable bleeding in this region can be fatal) is admitted by all the authors. It is widely used when the ABC affects the spine and the pelvis where we can't use a pneumatic tourniquet. Complications as ischemia of neurological structures or other organs are possible [9].

If surgery is indicated, it must fulfill three obligations: the complete excision of the tumor, decompression of the spinal cord and reconstruction and stabilization of the spine. It is essential, especially in this localization, to treat the lesion in only one surgical procedure. further surgeries are challenging and always complicated [10].

Surgical curettage is the most appropriated treatment for ABC of the spine. It consists in accessing the cyst via a window, performing a careful curettage of its cavity and excising its lining. We can combine this technique with bone graft. Most of the recurrences occur during the first months after the treatment (3 to 6 months). There are usually less chances of recurrence in the vertebral localizations [11,12].

Conclusion:

ABCs are benign and rare tumors of the child. A stiff and painful back is the most frequent warning sign. This tumor can be severe when it is localized in the spine because of its neurological risks. Surgical treatment is essential when neurological symptoms are present.

Conflict of interest: none

References:

[1]Burch S, Hu S, Berven S. Aneurysmal bone cysts of the spine. *Neurosurg Clin N Am.*2008; 19: 41-47.

[2]Mascard E, Gomez-Brouchet A, Lambot K. Bone cysts: unicameral and aneurysmal bone cyst. *Orthop Traumatol Surg Res.* 2015 ;101(1): S119-27.

[3]Jaffe HL, Lichtenstein L: Solitary unicameral bone cysts with emphasis on the roentgen picture, the pathologic appearance and the pathogenesis. *Arch Surg.* 1942; 44: 1004-25.

[4]Tsagozis P, Brosj O. Current Strategies for the Treatment of Aneurysmal Bone Cysts. *Orthop Rev.* 2015 28 ;7(4) :106-10.

[5]Cottalorda J, Kohler R, Sales De Gauzy J, Chotel F, Mazda K, Lefort G, et al: Epidemiology of aneurysmal bone cysts in children: a multicenter study and literature review. *J Pediatr Orthop B,* 2004, 13, 389-94.

[6]Zileli M, Isik HS, Ogut FE, Is M, Cagli S, Calli C. Aneurysmal bone cysts of the spine. *Eur Spine J.* 2013 ;22 (3):593-601.

[7]Riahi H, Mechri M, Barsaoui M, Bouaziz M, Vanhoenacker F, Ladeb M. Imaging of Benign Tumors of the Osseous Spine. *Journal of the Belgian Society of Radiology.* 2018; 102(1): 1-11.

[8]Chan MS, Wong YC, Yuen MK, Lam D. Spinal aneurysmal bone cyst causing acute cord compression without vertebral collapse: CT and MRI findings. *Pediatr Radiol.* 2002 ;32(8):601-4.

[9]Terzi S, Gasbarrini A, Fuiano M, Barbanti Brodano G, Ghermandi R; Bandiera S; et L. Efficacy and Safety of Selective Arterial Embolization in the Treatment of Aneurysmal Bone Cyst of the Mobile Spine: A Retrospective Observational Study. *Spine.*2017; 42(15):1130-38.

[10]Park HY, Yang SK, Sheppard WL, Hegde V, Zoller SD, Nelson SD, et al. Current management of aneurysmal bone cysts. *Curr Rev Musculoskelet Med.* 2016; 9(4): 435-44.

[11]Hauschild O, Ludemann M, Engelhardt M, Baumhoer D, Baumann T, Elger T, et al. Aneurysmal bone cyst (ABC): treatment options and proposal of a follow-up regime. *Acta Orthop belg.* 2016; 82(3):474-83.

[12]Ulici A, Nahoi C, Carp M, Fodor I, Dinu C. Surgical Treatment of an Aneurysmal Bone Cyst with Avascular Bone Graft. *Chirurgia (Bucur).* 2017 ;112(2):172-77.

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