International Journal of Medical and Health Research
ISSN: 2454-9142, Impact Factor: RJIF 5.54
www.medicalsciencejournal.com
Volume 3; Issue 3; March 2017; Page No. 82-85

Venous thromboembolic diseases to emergencies reporting other diseases

[1] H Kechna, [2] Ali Zinebi, [3] Abdellatif Ez-zouak, [4] Mohamed Matous, [5] Nabil Jbili, [6] Lotfi Bibiche, [7] Jaouad Loutid, [8] Khalid Chkoura, [9] Moulay Ahmed Hachimi

[2] Service of Internal Medicine, Moulay Ismail Hospital, Faculty of Medicine and Pharmacy, Sidi Mohamed Ben Abdellah University, Fez, Morocco

[3] Cardiology service, Moulay Ismail Hospital, Faculty of Medicine and Pharmacy, Sidi Mohamed Ben Abdellah University, Fez, Morocco

[4] Emergency Service, Moulay Ismail Hospital, Faculty of Medicine and Pharmacy, Sidi Mohamed Ben Abdellah University, Fez, Morocco

[1, 5-9] Moulay Ismail Hospital, Faculty of Medicine and Pharmacy, Sidi Mohamed Ben Abdellah University, Fez, Morocco

Abstract
Thromboembolic venous disease is a frequent and severe disease, unfortunately still too often diagnosed late due to a polymorphism of its clinical presentation. Etiological research is necessary because it is often a revealing mode of other pathologies whose treatment can sometimes control the thromboembolic process or limit its consequences. Our work focuses on the thromboembolic syndrome encountered in the emergency department of our training. Out of 3110 hospitalizations through the emergency department, 41 patients were treated with venous thrombosis from different sites, including 18 patients, or 44% with unusual sites related to various pathologies.
Thromboembolic disease is a medical emergency requiring the use of anticoagulant therapy in the shortest possible time. However, apart from a context which favors patent, the search for an etiological diagnosis is necessary.

Keywords: thrombophlebitis, venous thrombosis, thrombophilia

1. Introduction
Thromboembolic venous disease (VTE) is a common, frequent and serious disease. Unfortunately, this pathology is still too often underestimated. The polymorphism of its clinical presentation is probably partly responsible for such a state of affairs: of the "superficial phlebitis" taken over by the general practitioner, with severe pulmonary embolism, not always having time to the intensive care of cardiology, to post-operative deep phlebitis after general surgery.
In half of the cases, a favorable factor is found. Thrombophilia is defined as a coagulation state favoring the onset of VTE may be constitutional or acquired.
Etiological research is necessary because it is often a revealing mode of other pathologies whose treatment can sometimes control the thromboembolic process or limit its consequences. The primum movens of our work is the diagnosis of a recto colitis ulcero hemorrhagic (RCH) in a young patient hospitalized in intensive care for cerebral thrombophlebitis the month of January 2015. We have been interested since the thromboembolic syndrome encountered in the service of Emergencies of our hospital.

2. Material and methods
This is an open-ended prospective work spread over two years since 1/1/2015. In collaboration with several departments, a register was created where the various patients hospitalized in the various departments of the hospital were recorded through the Emergency Department for thromboembolic diseases regardless of the location (limbs, abdomen, lung, brain ...). In addition to the demographic data, there was the referral service and especially the evolution of the patients and the final diagnosis.

3. Results
During the study period (23 months), there were 3110 emergency department admissions, including 41 patients with venous thrombosis, a percentage of 1.31. These were 24 men and 17 women, a sex ratio of 1.41. The average age of patients is 47.46 years ± 16.5 with extremes between 22 years - 75 years.
Table 1 lists the sites of thrombophlebitis diagnosed during the study period. It appears that this is most often phlebitis of the lower limbs and / or pulmonary embolism (Table 2). Elsewhere, they are unusual localizations At the level of upper limb vein, mesenteric vein, portal vein, Superior cave vein (Table3), at level of right ventricle (Table 4) or cerebral veins (Rable 5) ...

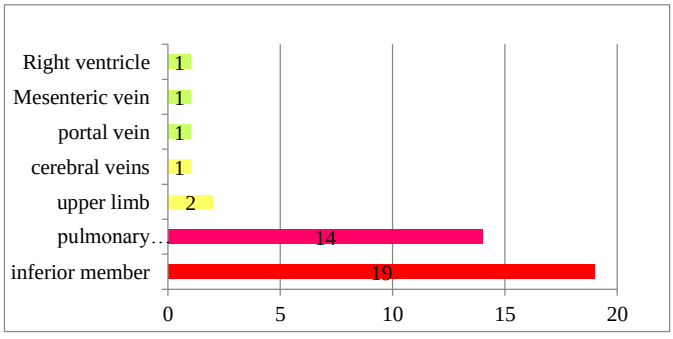

Table 1: Localization of Collected Thrombophlebitis

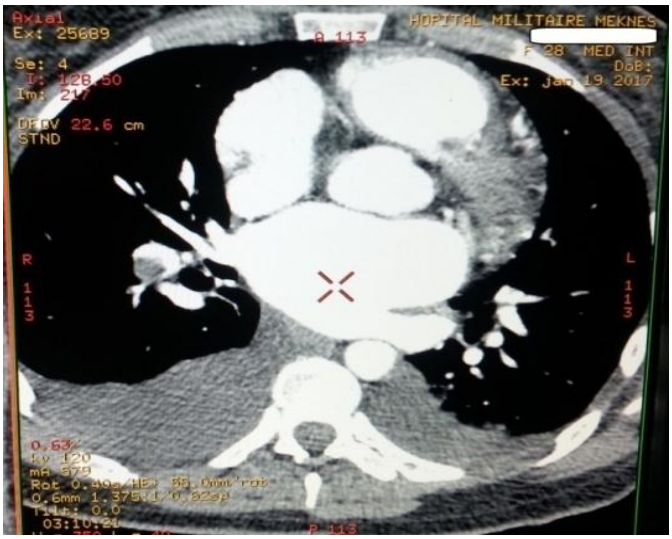

Fig 2: Massive pulmonary embolism especially right

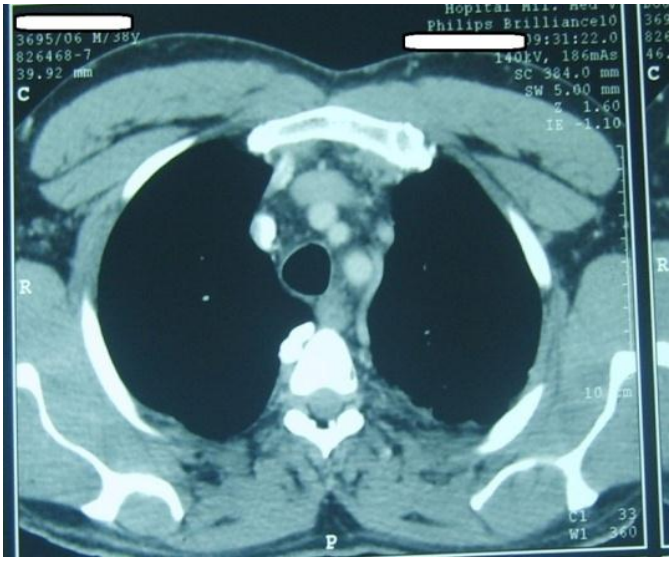

Fig 3: Thrombosis of the superior vena cava

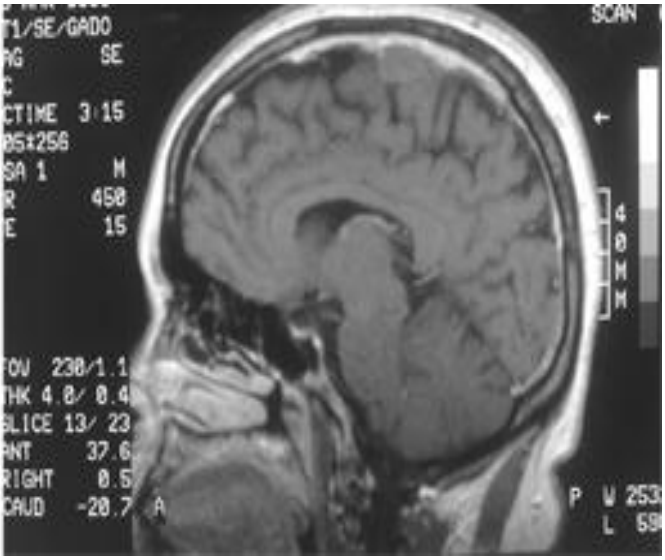

Fig 4: Thrombosis of the upper longitudinal sinus

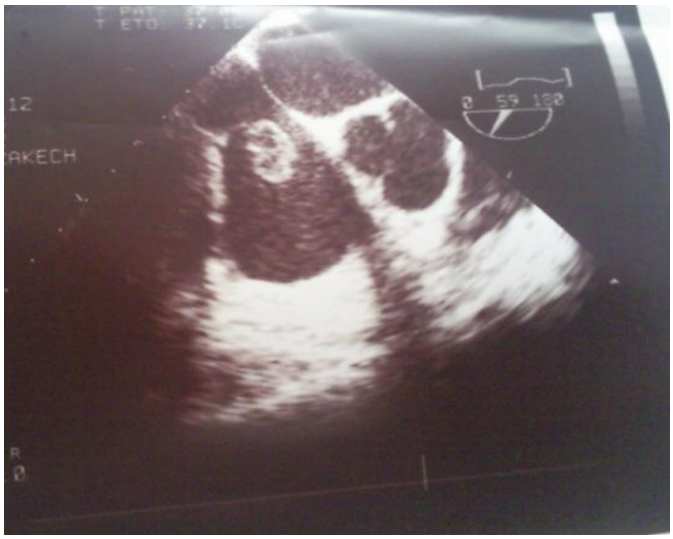

Fig 5: Right intraventricular thrombus

The etiological analysis and research of the different cases find that:

- In 19 patients, 46% of cases were circumstantial thromboembolic disease
- In 4 patients there was no evidence of a risk factor
- In 18 patients, 44% of the cases found that the venous thrombotic phenomenon is related to favorable pathologies.

Figure 6 illustrates the etiologies diagnosed and retained as a clear risk factor for thromboembolic disease. These are usually paraneoplastic syndrome or behcet disease. Elsewhere, two cases of lupus disease have been diagnosed and one case has been diagnosed for the following pathologies: ulcerative haemorrhagic retinitis, hyperhomocysteinemia, C protein deficiency and leaden mutation

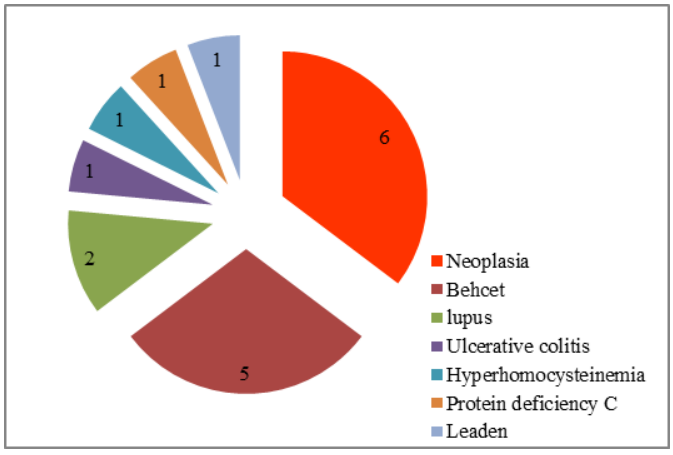

Fig 6: Graphical representation of the etiologies diagnosed in the course of a venous thrombotic episode.

Patient development was generally favorable, however, there were 6 deaths (14.63%) related to venous thromboembolic disease:

- 4 pulmonary embolisms on advanced neoplastic grounds
- Cerebral thrombophlebitis which revealed an inflammatory enteropathy type ulcerative-hemorrhagic ulcerocolitis having posed a therapeutic dilemma.

- Venous mesenteric ischemia is very likely secondary to a disease of behcet.

4. Discussion

Venous thromboembolic disease (VTE) is frequent and severe, with an incidence of 1.1 to 1.8 per 1000 population [1] and an early mortality rate ranging from 2 to 15% [2].

The numerous risk factors for VTE can be classified according to their constitutional or acquired, transient or permanent character [3, 4]. There are three main categories of risk factors: those related to the field (age, cancer ...), those linked to the circumstances leading to the occurrence of VTE (surgery, pregnancy, contraception ...) and, lastly, Thrombophilia or abnormalities of coagulation constitutional or acquired most often permanent.

There is no consensus to define patients in whom thrombophilia should be sought. Recommendations from the British Society of Hematology have been published [5, 6]. An unusual location of thrombosis, a combination of thrombosis and miscarriages, cutaneous necrosis with the introduction of antivitamin K are very evocative situations of underlying anomalies, but exceptional in practice.

The occurrence before the age of 45, the existence of a family history of thromboembolism, and even more so of familial thrombophilia, makes thrombophilia more likely. However, given the multifactorial nature of this pathology and the frequency of favorable circumstances, a familial antecedent is a relatively common element that does not necessarily reflect the existence of a hereditary anomaly. Conversely, the frequency in the general population of asymptomatic heterozygotes does not eliminate the existence of a constitutional thrombophilia in the absence of a history of VTE. Finally, the existence of transient factors favoring thrombophilia, associated in 50% of cases with thrombophilia [7], does not eliminate thrombophilia. However, the severity of the thromboembolic event does not in itself constitute an indication to perform a thrombophilia assessment.

The search for thrombophilia, when recommended, should be concerned with all the abnormalities responsible, as there is no clinical characteristic that makes it possible to focus on this or that anomaly. This assessment will be carried out ideally before starting the anticoagulant treatment or at a distance (one month) of the cessation of the anticoagulant. Indeed, the antivitamins K disrupt the assays of the proteins C and S, and the heparin decreases the AT. Abnormalities observed during hormonal treatment or gestation should be controlled at a distance.

In first intention several dosage and or research are possible. In order of frequency, a deficiency of antithrombin III (activity), protein C (activity) and / or protein S. will be sought. A factor VIII assay will be performed, the G20210A mutation or a protein C The metabolism of the V-factor Leiden, an assay for homocysteinemia (± dose of methionine loading), antiphospholipid antibodies (anticardiolpine / antiphospholipid antibodies, anti-beta2-glycoprotein, detection of circulating lupus anticoagulant) are sought.

Although rare, the discovery of thrombophilia has therapeutic implications. Thus, a pregnant woman without MCDV ATCD with coagulation abnormality requires preventive anticoagulation with low molecular weight heparin (LMWH) during pregnancy and / or for 6 weeks after delivery. A thrombophilia discovered in a woman against it indicates a hormonal treatment (contraceptive, substitutive or antineoplastic) [8]. The controversy remains large for the asymptomatic subjects carrying thrombophilia in situation at risk (plane, surgery, immobilisation)

In the treatment of thrombophilia, the presence of thrombophilia changes little or little over the duration of treatment, which is a function of the risk of recurrence and the risk of haemorrhage. A treatment of 6 to 12 months is recommended for idiopathic thrombosis and those associated with an isolated coagulation abnormality [9]. Nevertheless, in case of deficiency in Anti thrombin III, heparin doses must be increased or even supplemented with antithrombin in order to achieve effective anticoagulation [8]. Protein C deficiency; There is a risk of cutaneous necrosis (especially homozygotes) secondary to the introduction of Anti vitamin k (AVK). It makes sense to achieve a less early, progressive relay in these patients. Supplementation with folate and vitamin B12 helps normalize homocysteinaemia. However, the effectiveness of this measure does not prevent the risk of recidivism [10].

Whether or not it is decided to conduct a search for constitutional thrombophilia, it is essential to look for other risk factors for VTE that are more frequent and whose diagnosis could be the cause of a particular management (neoplasia, Myeloproliferative syndrome, nephrotic syndrome) [11].

5. Conclusion

Venous thromboembolic disease is a medical emergency requiring the use of anticoagulant therapy as soon as possible. Constitutional thrombophilia are rare in their relatively modest therapeutic implications. Apart from a favorable context, there is a certain interest in seeking more frequent factors (neoplasia, myeloproliferative syndrome, nephrotic syndrome), which have a more important therapeutic effect.

6. References

1. Oger E. Incidence of venous thromboembolism: a community based study in western France. EPI-GETBP Study Group. West Britain Thrombosis Study Group. Thromb Haemost. 2000; 83(5):657-60.
2. Quinlan DJ, McQuillan A, Eikelboom JW. Low-molecular-weight heparin compared with intravenous unfractionated heparin for treatment of pulmonary embolism: a meta-analysis of randomized, controlled trials. Ann Intern Med. 2004; 140(3):175-83.
3. Heit JA, *et al*. Risk factors for deep vein thrombosis and pulmonary embolism: apopulation-basedcase-controlstudy. Arch Intern Med. 2000; 160(6):809-15.
4. Goldhaber SZ. Pulmonary embolism. Lancet 2004; 363(9417):1295-305.
5. British Society for Haematology. Investigation and management of heritable thrombophilia. Br J Haematol. 2001; 114(3):512-28.
6. Emmerich J. How to assess thrombophilia and which one? Rev Prat. 2007; 57(7):714-5.
7. Seligsohn U, Lubetsky A. Genetic susceptibility to venous thrombosis. N Engl J Med. 2001; 344(16):1222-31.
8. Nicolaides AN, *et al*. Thrombophilia and venous thromboembolism. International consensus statement. Guidelines according to scientific evidence. Int Angiol 2005; 24(1):1-26.

9. Buller HR, *et al*. Antithrombotic therapy for venous thromboembolic disease: the seventh ACCP conference on antithrombotic and thrombolytic therapy. Chest 2004; 126(3 Suppl):401S-28S.
10. Ray JG, *et al*. Homocysteine-lowering therapy and risk for venous thromboembolism: a randomized trial. Ann Intern Med. 2007; 146(11):761-7.
11. Roux A, Sanchez O, Meyer G. What is a thrombophilia assessment in a patient with venous thromboembolic disease? Resuscitation. 2008; 17:355-362.