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Embolisation artérielle versus traitement médical dans la prise en charge des hématomes musculaires abdomino-pelviens spontanés sous traitement anticoagulant

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par

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Né le 09/05/1993 à Cholet (*Maine-et-Loire*)

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Embolisation artérielle versus traitement médical dans la prise en charge des hématomes musculaires abdomino-pelviens spontanés sous traitement anticoagulant.

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Abréviations

ANSM : Agence Nationale de Sécurité du Médicaments et des produits de santé

APTT : Activated Partial Thromboplastin Time

AVK : Anti-Vitamine K

CT : Computed Tomography

DOAC : Direct Oral Anticoagulant

eGFR : estimated Glomerular Filtration Rate

INR : International Normalized Ratio

PT : Prothrombin Time

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Introduction

A. Les hématomes musculaires spontanés

1. Définitions

Les hématomes musculaires spontanés sont définis comme une extravasation de sang au sein d'un groupe musculaire, sans cause mécanique (traumatique, chirurgicale, iatrogène) ou comorbidité favorisante identifiée (1, 2). Ils surviennent majoritairement chez des patients âgés sous traitement anticoagulant (3, 4).

Leur physiopathologie est complexe, basée sur une micro-angiopathie multifactorielle (1, 4, 5). Leur survenue est rapportée dans les suites de microtraumatismes (efforts à glotte fermée, contractions musculaires isométriques), et différents facteurs favorisants ont été identifiés dans la littérature (âge, athérosclérose, hypertension artérielle, diabète) (1, 4, 5).

Ces hématomes peuvent rester contenus dans le compartiment musculaire concerné, ou se rompre et diffuser dans les espaces de la cavité abdominale (intra-péritonéal, rétro-péritonéal, sous-péritonéal) levant alors l'effet de tamponnade anatomique des fascia musculaires (6).

Le traitement médical est souvent efficace pour les hématomes de petit volume dont l'extension est limitée par le fascia musculaire, mais un traitement invasif peut être nécessaire en cas d'hémorragie abondante responsable d'hématomes de volume important et/ou rompus (7–11).

Toutes les classes de traitement anticoagulant (anti-vitamine K, héparines et leurs dérivés, anticoagulants oraux directs) sont concernées par les complications hémorragiques parmi lesquelles figurent les hématomes musculaires, favorisées par un surdosage (1–3, 12).

2. Épidémiologie

L'incidence des hématomes musculaires spontanés sous traitement anticoagulant est croissante et corrélée à l'augmentation du nombre de patients traités (1, 13).

L'utilisation des anticoagulants ne cesse d'augmenter ces dernières années (12, 14–16). Un rapport de l'Agence Nationale de Sécurité du Médicament et des produits de santé publié en avril 2014 estime que 13,7% des sujets de plus de 65 ans ont été exposés au moins une fois à un traitement anticoagulant en France en 2013 (12).

La littérature rapporte des taux de complications hémorragiques compris entre 2.0% et 4.0% par an chez les patients traités par anticoagulant, au sein desquelles les hémorragies des tissus mous figurent parmi les plus fréquentes (15, 16, 17).

3. Rappels anatomiques

Les hématomes musculaires liés aux anticoagulants surviennent le plus souvent dans quatre régions anatomiques : la paroi abdominale antérieure, le compartiment ilio-psoas, la région fessière et la racine de cuisse.

La paroi abdominale antérieure est constituée des muscles grand droit, oblique interne, oblique externe, et transverse, tous pairs et symétriques. Leur vascularisation est assurée par l'artère épigastrique inférieure, branche de l'artère iliaque externe dont les rameaux distaux s'anastomosent avec l'artère épigastrique supérieure, branche de l'artère thoracique interne, et par l'artère circonflexe iliaque profonde branche de l'artère iliaque externe.

Les muscles iliaque, psoas et les muscles érecteurs du rachis constituent la paroi musculaire postérieure de l'abdomen et du pelvis, ils sont également pairs et symétriques. Ils sont vascularisés par les artères lombaires, branches pariétales de l'aorte abdominale au nombre de cinq de chaque côté, et par l'artère iliolumbaire, première branche ascendante du tronc postérieur de l'artère iliaque interne.



Figure 1 : radio-anatomie des muscles la paroi abdominale antérieure et postérieure. Scanner sans injection, plan axial. e-Anatomy, IMAIOS 2021.

La région fessière comprend les muscles grand glutéal, moyen glutéal, petit glutéal, piriforme, et obturateur interne, tous pairs et symétriques. Ils sont vascularisés par les artères glutéale supérieure, branche terminale du tronc postérieur de l'artère iliaque interne, et glutéale inférieure, branche terminale du tronc antérieur de l'artère iliaque interne.

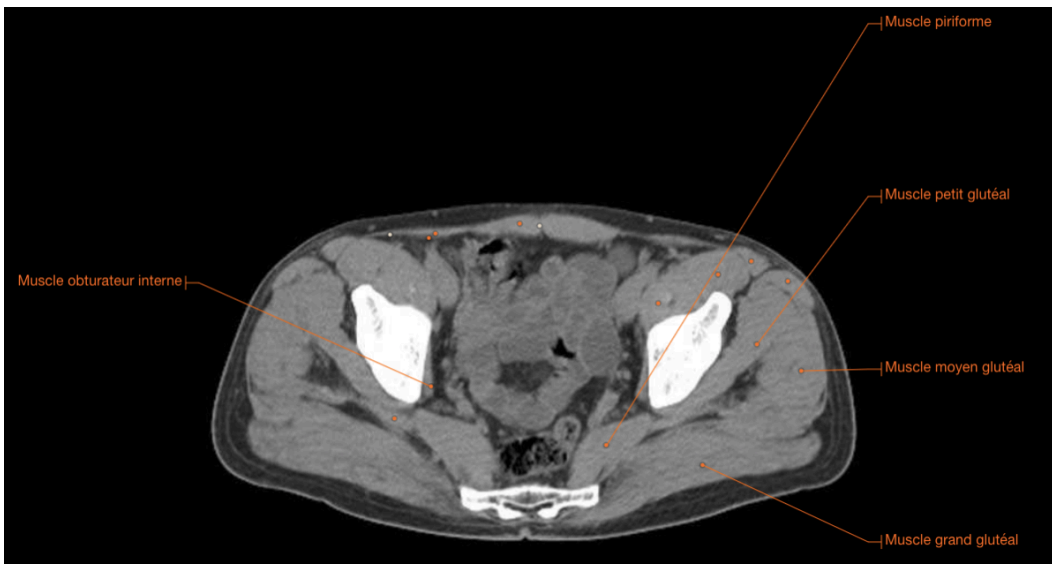


Figure 2 : radio-anatomie des muscles de la région fessière. Scanner sans injection, plan axial. e-Anatomy, IMAIOS 2021.

Les muscles de la racine de cuisse sont répartis en trois loges de chaque côté : antérieure qui comprend les quatre vastes du quadriceps (droit fémoral, vaste latéral, vaste médial, vaste intermédiaire), interne qui comprend les muscles adducteurs (grand adducteur, court adducteur, long adducteur), et postérieure qui comprend les muscles ischio-jambiers (biceps fémoral, semi-tendineux, semi-membraneux). Ces muscles sont vascularisés par les artères circonflexes médiale et latérale de cuisse, et les artères perforantes de cuisse, branches de l'artère fémorale profonde, et par l'artère obturatrice, branche du tronc antérieur de l'artère iliaque interne.



Figure 3 : radio-anatomie des muscles de la racine de cuisse. Scanner sans injection, plan axial. e-Anatomy, IMAIOS 2021.

La connaissance de l'anatomie vasculaire de ces groupes musculaires est indispensable pour l'identification de l'artère responsable de l'hémorragie au scanner et lors de l'artériographie.

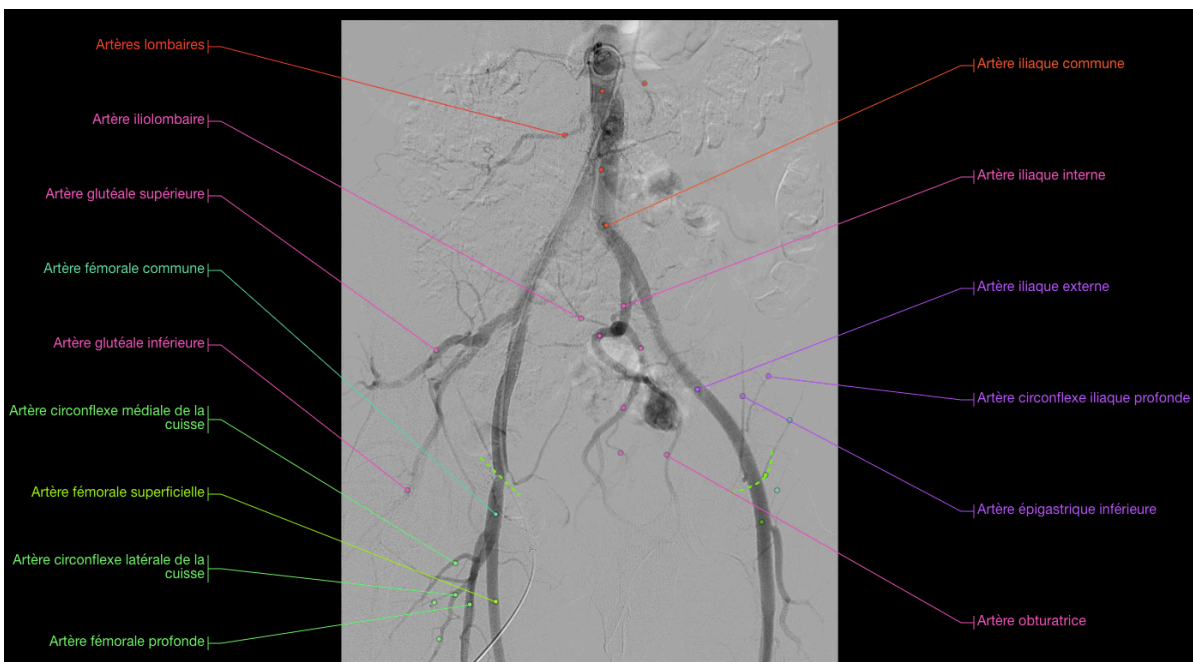


Figure 4 : artériographie de l'aorte abdominale, des axes ilio-fémoraux et de leurs branches. Vue axiale oblique. e-Anatomy, IMAIOS 2021.

4. Diagnostic

La présentation initiale est variée et les symptômes sont aspécifiques. Les hématomes musculaires spontanés sont le plus souvent révélés par des douleurs abdominales, une masse pariétale, des ecchymoses cutanées, ou des nausées et des vomissements. L'examen physique peut retrouver une défense abdominale et des signes d'irritation péritonéale (1–4, 18).

Des signes généraux doivent être recherchés : tachycardie, hypotension artérielle, oligo-anurie, pâleur cutanée, allongement du temps de recoloration cutanée, afin de ne pas méconnaître un état de choc hémorragique.

L'anamnèse s'attache à rechercher la prise d'un traitement anticoagulant et un éventuel surdosage.

Le diagnostic de certitude est apporté par l'imagerie. La tomodensitométrie est supérieure à l'échographie dans ces indications, avec des sensibilité et spécificité proches de 100% dans la plupart des études, et la possibilité de rechercher des signes d'hémorragie active (5, 6, 19). Le protocole d'acquisition comprend un premier passage en contraste spontané, puis après injection de produit de contraste iodé plusieurs acquisitions aux phases artérielle, portale, voire tardive.

L'hélice sans injection de produit de contraste permet le diagnostic positif d'hématome musculaire spontané, qui se présente comme une image intramusculaire spontanément hyperdense (classiquement 40 à 60 UH), et d'en définir la taille et l'extension anatomique.

La phase artérielle permet de rechercher des signes d'hémorragie active (extravasation de produit de contraste) et de localiser le vaisseau responsable du saignement. Elle est utile pour obtenir une cartographie vasculaire avant un éventuel traitement par embolisation.

Les phases portale et tardive permettent de rechercher des saignements dont le débit est plus faible, ou qui n'apparaîtraient pas dès la phase artérielle chez des patients en état de choc du fait de l'insuffisance circulatoire.

Un diagnostic précoce permet une prise en charge rapide et adaptée et une réduction de la mortalité (2, 11, 20).

La détection d'une extravasation de produit de contraste au temps artériel, traduisant un saignement actif, est un élément clef de la prise en charge. En effet plusieurs études ont montré qu'un saignement actif retrouvé au scanner constituait un critère de gravité et un facteur prédictif d'échec du traitement médical seul (7, 21, 22).

B. Stratégie de prise en charge

1. Traitement médical

Le traitement médical, également appelé traitement conservateur, est recommandé en première intention chez les patients stables et pour lesquels le scanner n'a pas montré de signe de saignement actif (8, 20–23). Il comprend plusieurs volets : expansion volémique associée si besoin à des transfusion de culots de globules rouges, plasma frais congelés, et concentrés plaquettaires. Arrêt du traitement anticoagulant et antagonisation en cas de surdosage (sulfate de protamine pour les héparines, concentrés de complexes prothrombiniques pour les anti-vitamine K). Le recours à des amines est parfois nécessaire et doit faire discuter l'intérêt d'un traitement invasif.

2. Embolisation artérielle

L'embolisation artérielle constitue le traitement de référence des patients instables ou chez lesquels le traitement médical est jugé insuffisant ou inefficace (13, 23, 24).

L'abord vasculaire s'effectue le plus souvent par ponction percutanée de l'artère fémorale commune, permettant ensuite l'introduction de guides et sondes adaptés pour naviguer dans le réseau artériel.

Sur la base des constatations scanographiques, le radiologue explore les différentes artères vascularisant le groupe musculaire concerné à la recherche d'extravasations de produits de contraste traduisant un saignement actif. Des micro-cathéters (2,7 – 2,8F) permettent un cathétérisme super-sélectif des pédicules vasculaires qui augmente la sensibilité de la détection des fuites de produit de contraste.

Après identification et traitement de la cible artérielle, il est nécessaire de rechercher des branches collatérales qui pourraient reprendre en charge l'hémorragie en cas d'embolisation trop proximale, ou à l'inverse représenter un risque de complication en cas de reflux de l'agent d'embolisation.

Le choix de l'agent d'embolisation est fonction de l'expérience de l'opérateur, de l'anatomie vasculaire, et de la gravité de l'hémorragie, en prenant en compte les caractéristiques propres à chaque agent (taille, composition, mécanisme d'action, temps de résorption). Les agents particuliers comprennent la gélatine (résorbable) et les microsphères calibrées (non résorbables). Les agents mécaniques comprennent les coils ou microspires, et plugs. Les agents liquides comprennent les colles acryliques (cyanoacrylate) et l'éthylène vinyl alcool (Onyx).

Un cathétérisme sélectif ayant permis une embolisation de la cible avec absence de saignement actif sur l'angiographie finale de contrôle définit le succès technique de l'embolisation.

3. Traitement chirurgical

Le traitement chirurgical n'est pas recommandé en première intention dans la prise en charge des hématomes musculaires spontanés, notamment rétropéritonéaux. En effet l'ouverture du compartiment musculaire lève l'effet de tamponnade anatomique pouvant majorer l'hémorragie, et l'identification peropératoire de l'artère responsable du saignement est difficile (8, 11).

La chirurgie doit être envisagée chez les patients instables en cas d'échec de l'embolisation artérielle ou si l'embolisation n'est pas disponible (11, 24). Elle est également indiquée en cas de compression nerveuse symptomatique ou de syndrome du compartiment abdominal (11).

Il n'existe pas à ce jour de consensus concernant la stratégie optimale de prise en charge des hématomes musculaires abdomino-pelviens spontanés sous traitement anticoagulant. Le traitement dépend souvent des habitudes locales et de la disponibilité d'un plateau technique de radiologie interventionnelle. L'objectif de notre étude était de comparer la présentation clinique, biologique et les caractéristiques tomodensitométriques des patients à leur admission, et de comparer l'efficacité et la sécurité du traitement médical et de l'embolisation artérielle.

Article :

Arterial Embolization vs Conservative Treatment in the Management of Spontaneous Abdominal and Pelvic Muscle Hematomas due to Anticoagulation Therapy

Abstract

Objectives: To compare the clinical presentation, biological and imaging findings of patients admitted for anticoagulant-related abdominal or pelvic muscle hematoma according to the management strategy. To evaluate efficacy and safety of conservative management and arterial embolization.

Methods: Between 2015 and 2020, 219 patients were included and 62 were treated with arterial embolization. Demographic data, clinical and biological status at admission, CT findings and treatment elements were recorded. Clinical success was defined as bleeding cessation, hemodynamic stability and hemoglobin level stabilization. Univariate and multivariate analysis were performed to evaluate mortality.

Results: Patients with severe initial presentation were mainly treated with embolization. Patients managed conservatively were receiving more vitamin K antagonists. Patients treated with embolization had larger hematomas, more contrast extravasation and muscular fascia ruptures on CT. Technical success rate of embolization was 93.5%. Clinical success rate was 75%. All cause 30-day mortality rate after embolization was 22.6%. Muscular fascia rupture and large hematoma volume were associated with an increased mortality.

Conclusion: Embolization should be carried out immediately in hemodynamically unstable patients with a large hematoma, active bleeding, and/or muscular fascia rupture on CT. An overdose of anticoagulation therapy should always be corrected first. In patients with a less severe presentation, many factors should be taken into account for an optimal management.

Introduction

Spontaneous muscle hematomas related to anticoagulation therapy in the absence of traumatism or recent surgery is a frequent condition (1–6).

Predominantly reported in elderly patients, spontaneous muscle hematomas often affect ilio-psoas group or the rectus sheath causing nonspecific clinical symptoms. Delayed diagnosis may lead to hospital admission and increased mortality (2,7,8).

Computed tomography (CT) is the key for an early diagnosis and to define the optimal management strategy (9).

Even though conservative treatment including anticoagulation treatment withdrawal or reversal, volume expansion and transfusion, appears to be efficient in most cases, muscle hematomas may become life-threatening and require a more invasive strategy (10–13).

Transcatheter arterial embolization is favored over surgery because bleeding vessels may be difficult to identify and the anatomical tamponade may be lifted in open surgery (10,14).

Embolization is effective to treat muscle hematomas after failure of conservative management, with high clinical success rate and a low risk of complications (15–20).

Recent studies have identified factors associated with failure of conservative management notably volume of hematoma, ruptured muscular fascia, contrast-material extravasation on CT, hemoglobin decrease and need for transfusions (21,22).

There is no consensus opinion on the optimal management strategy that often depends on local experience and availability of interventional radiology.

The primary objective of this study was to compare the clinical presentation, biological and imaging findings at admission of patients admitted for anticoagulant-related abdominal or pelvic muscle hematoma according to the management strategy. Secondary objectives included evaluation of efficacy and safety of conservative management and arterial embolization. Survival of patients 30 days after admission was also evaluated in both groups.

Materials and methods

The medical and imaging records of patients who were under anticoagulant and/or antiplatelet therapy and admitted at our hospital because of spontaneous non-traumatic abdominal and/or pelvic hematoma between January 1st 2015 and December 31st 2020 were reviewed retrospectively.

This study was conducted in a university hospital and was approved by the Research Ethics Committee (CLERS identification number 2315).

Patient selection

A total of 2,958 imaging reports were assessed to select patients with abdominal or pelvic muscle hematomas. Then, 458 patients with imaging-proven hematoma were eligible. A total of 239 patients were excluded: patients who underwent a surgical intervention within a month before admission (n=90), patients with history of recent trauma (n=78), patients who underwent a recent invasive procedure potentially accounting for hematoma (arterial puncture n=22, venous puncture n=6, biopsy n=9), patients with constitutional hemostasis disorder (n=12), patients who had had recent fibrinolytic therapy (n=2) and patients not treated with anticoagulant therapy (n=20). The final number of patients meeting the study criteria was 219 (Fig 1).

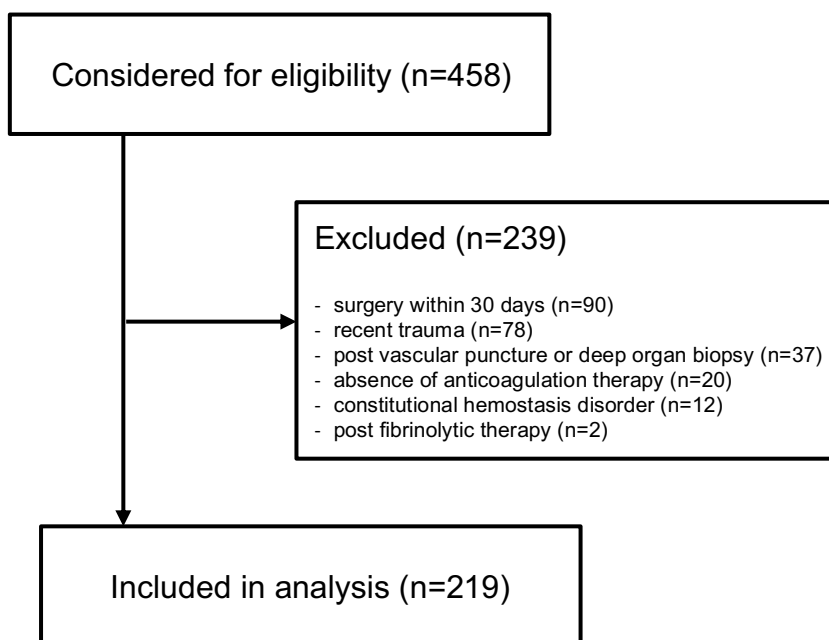


Figure 1 : study flowchart

Collected variables

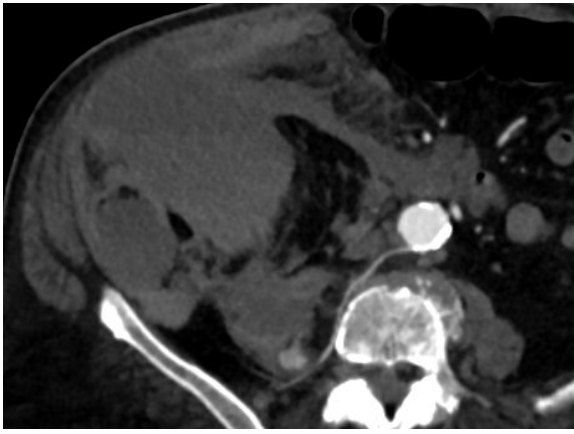
The demographic data, comorbidities of patients, history of anticoagulant/antiplatelet drug usage, presenting signs and symptoms, clinical follow-up, treatment methods and end-results were evaluated. International normalized ratio (INR) results and length of hospital stay were also documented and we also tried to identify predisposing factors for spontaneous abdominal wall hematoma.

For each included patient, demographic data were collected including sex, age, height, weight, body mass index, comorbidities, anticoagulant therapy type and indication, antiplatelet therapy. Clinical and biological data were collected at day 0 (defined as the initial CT evaluation

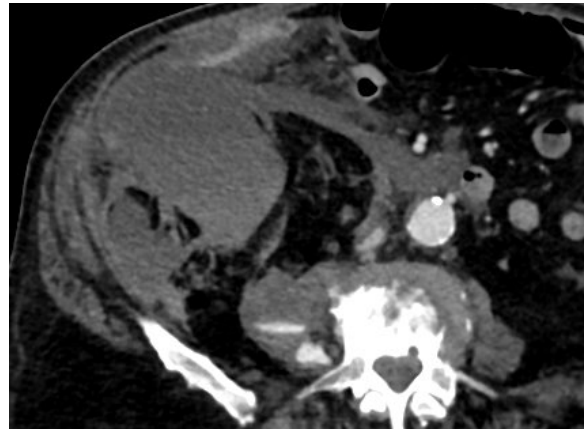
date), and day 1 (defined as the day after day 0 for patients managed conservatively, and the day after procedure for patients treated with arterial embolization): blood pressure, heart rate, hemodynamic shock, hemoglobin level, platelet count, estimated glomerular filtration rate (eGFR), prothrombin time (PT), activated partial thromboplastin time (APTT), international normalized ratio (INR) and anti-Xa activity.

Imaging findings

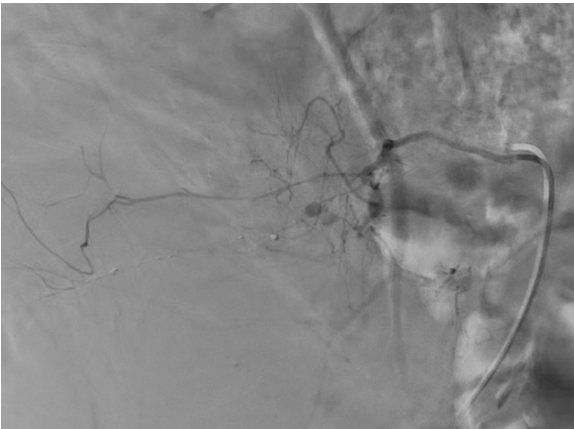
All patients underwent CT examination, mostly triphasic contrast-enhanced CT consisting in unenhanced acquisition followed by arterial and portal phases. Delayed phase was added at the radiologist discretion. The location of the hematoma (abdominal wall, retroperitoneal, thigh and gluteal) was noted. Rupture of the muscular fascia associated with intra-peritoneal, retro-peritoneal or sub-peritoneal extension was tabulated. The longest diameter, surface and volume of the hematoma were calculated using a commercially available semi-automatic segmentation software (Aquarius iNtuition, TeraRecon, USA). When contrast-enhanced CT was available, the presence of active bleeding (arterial phase or venous phase) was recorded and measured (longest diameter and volume). The corresponding visible bleeding vessels were noted. Active bleeding was defined as the presence of contrast material extravasation that appeared on arterial phase CT images and grew on venous and delayed-phase images (Figs 2, 3). The number of extravasation sites (single or multiple) was noted.



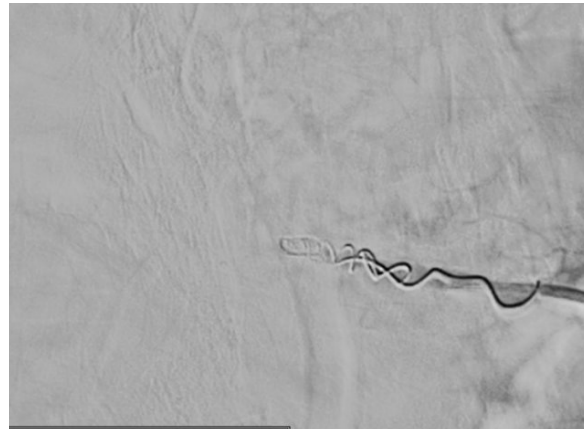
a.



b.



c.



d.

Figure 2: 86-year-old male under vitamin K antagonists and low-molecular weight heparin (bridging anticoagulation). Abdominal pain associated with hemodynamic instability. **(a)** Iodinated contrast-enhanced CT image obtained at the arterial phase, axial plane 4 mm maximum intensity projection reconstructions, demonstrates a psoas muscle hematoma with active bleeding from a lumbar artery with retroperitoneal and intraperitoneal infiltration. **(b)** Contrast-enhanced CT image obtained at the portal phase shows an increase of the extravasation size in the psoas muscle. **(c)** Selective angiogram of the third right lumbar artery shows a proximal contrast extravasation. **(d)** Selective control angiogram shows a satisfactory occlusion with coils and particles and no residual extravasation.

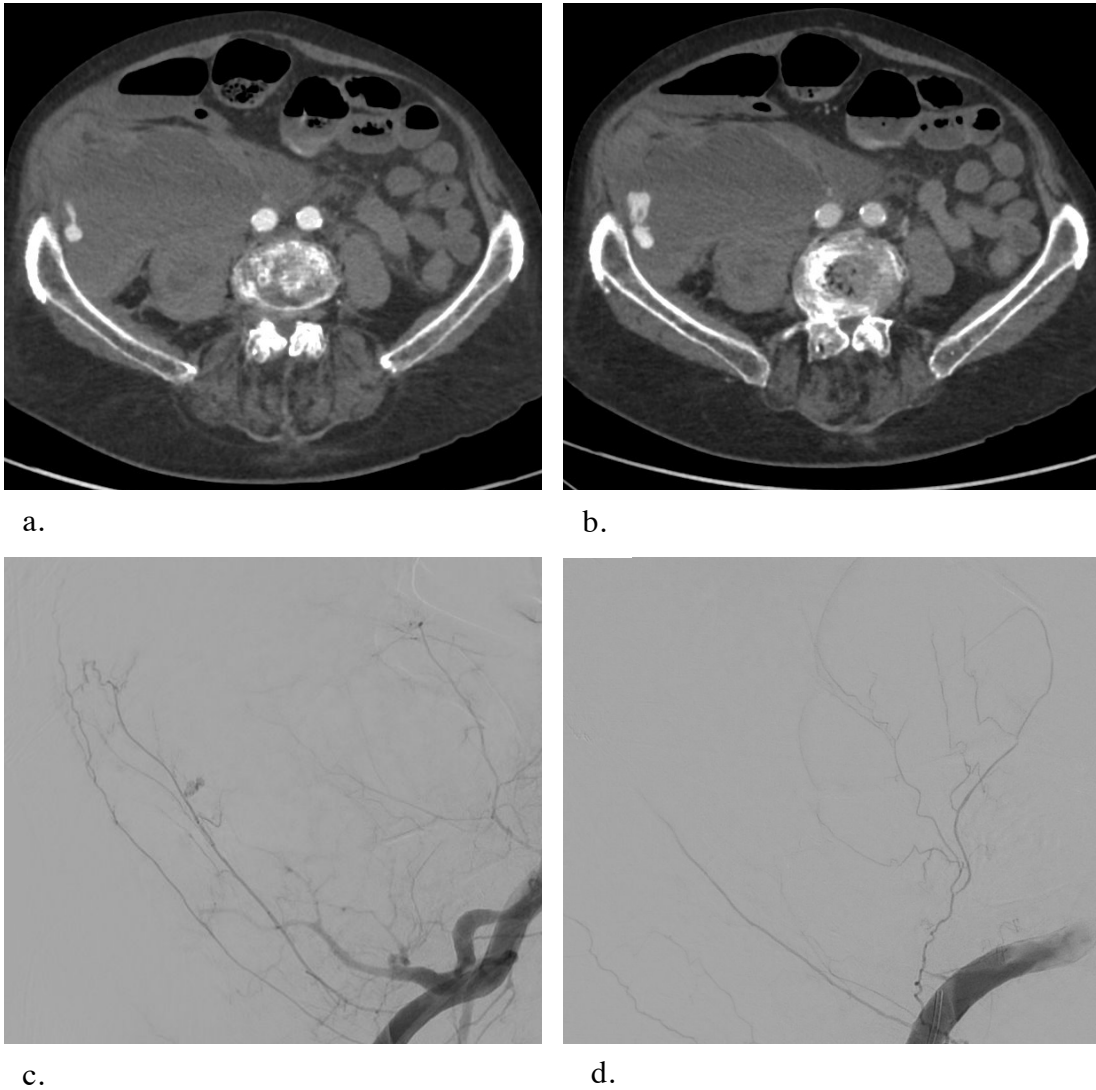


Figure 3: 85-year-old female under vitamin K antagonists and low-molecular weight heparin (bridging anticoagulation). Hemorrhagic shock. **(a)** Iodinated contrast-enhanced CT image obtained at the arterial phase in axial plane shows an iliopsoas muscle hematoma with contrast extravasation, and retroperitoneal and intraperitoneal infiltration. **(b)** Contrast-enhanced CT image obtained at the portal phase in axial plane shows an increase in the area of extravasation. **(c)** Angiogram shows a distal contrast extravasation from the right deep circumflex iliac artery. **(d)** Selective control angiogram shows a reduced proximal flow and distal occlusion with acrylic glue with no residual extravasation.

Management strategy

Management data were collected for every included patient. Patients with vitamin K antagonists or heparin overdose were treated with treatment discontinuation and/or reversal of anticoagulation using protamine sulphate, vitamin K or prothrombin complex according to the prothrombin time, and transfusion. The number of red blood cells units and platelets concentrates administered were noted before and after embolization if performed. If required, the use of catecholamine support was recorded, with the time for weaning after the intervention.

Embolization procedure

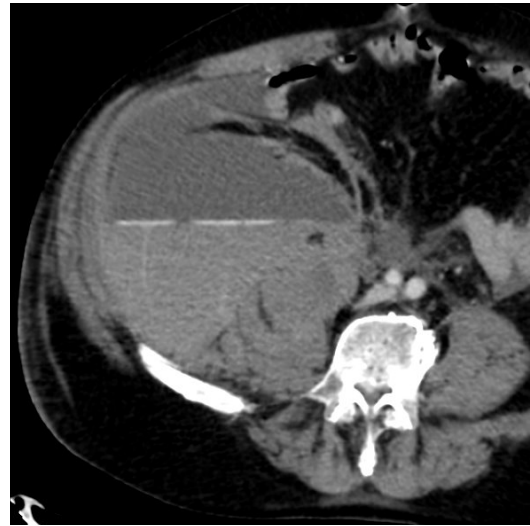
Arterial embolization was performed by a senior interventional radiologist with 5 or more years of experience in arterial embolization under fluoroscopic guidance. The culprit artery was selectively catheterized using different shapes of 5-F catheter (Cobra, Simmons, and JR) and a 2.7-2.8-F microcatheter was advanced co-axially. The embolic material was selected by the operator.

The following procedural data were recorded: delay between initial CT and embolization, contrast extravasation and involved arteries, embolized arteries (including empiric embolization if performed), embolic material used (gelatin sponge, tris-acryl microspheres, metallic coils, acrylic glue or a combination of agents). Technical success was defined as successful catheterization of target arteries with absence of active bleeding on final control angiogram (Figs 4, 5). In patients with no visible active bleeding, angiographic success was defined as a complete occlusion of the arteries corresponding to the hematoma territory (i.e. lumbar arteries in case of retroperitoneal hematoma). Complications were classified according to the Society of Interventional Radiology grading system (23).

The need and indication for additional treatment including repeat embolization or surgery were recorded.



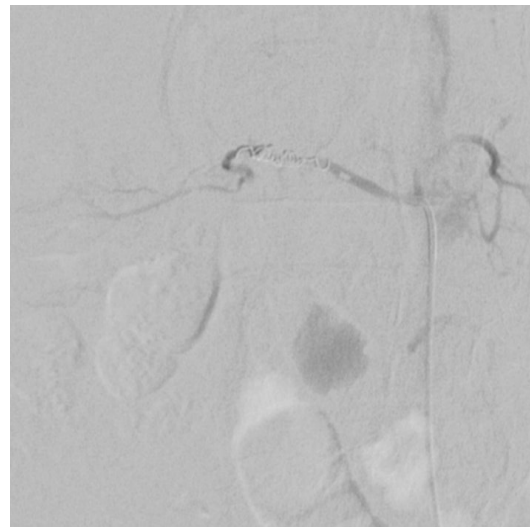
a.



b.



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d.

Figure 4: 60-year-old male under low-molecular weight heparin for pulmonary embolism. Hemorrhagic shock. **(a)** Iodinated-contrast-enhanced CT image obtained at the arterial phase in axial plane shows an iliopsoas muscle hematoma with a fluid-fluid level and contrast material extravasation, and an intraperitoneal infiltration. **(b)** Contrast-enhanced CT image obtained at the portal phase in axial plane shows multiple areas of extravasations. **(c)** Selective angiogram confirms the presence of multiple contrast extravasations from the third right lumbar artery. **(d)** Selective control angiogram shows a markedly reduced flow after embolization with coils and no residual extravasation.

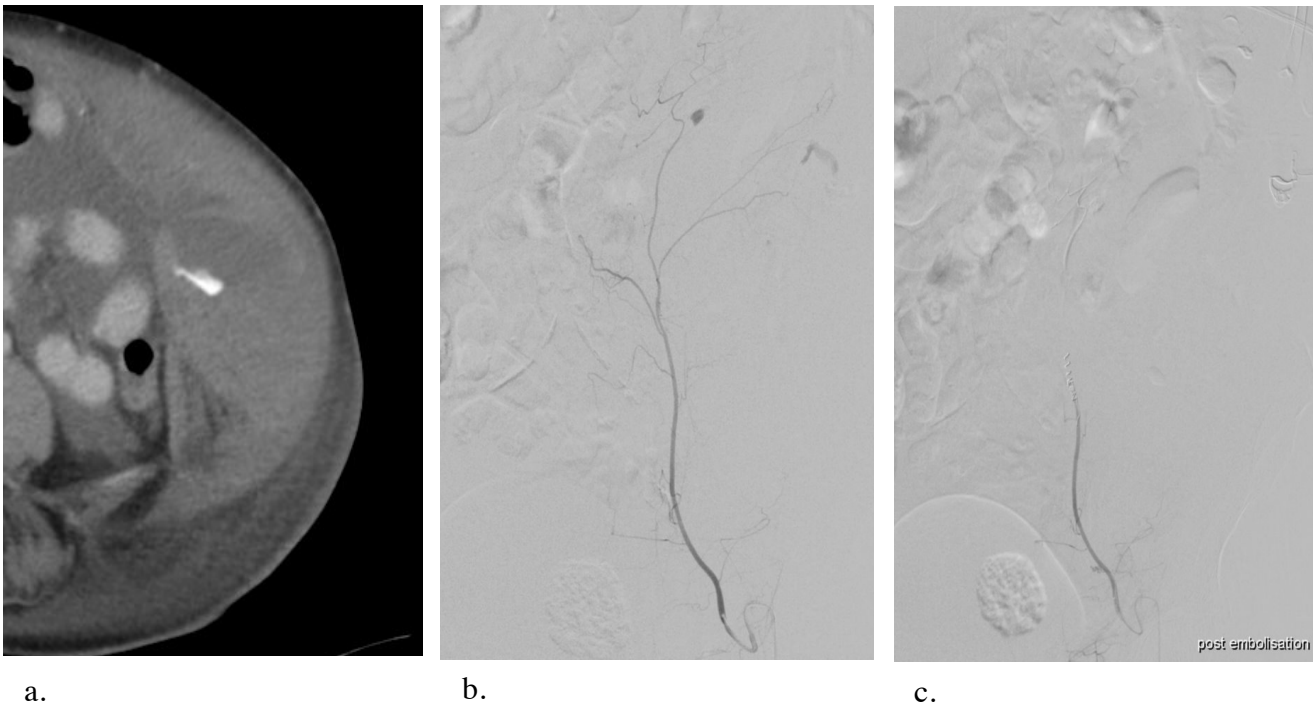


Figure 5: 85-year-old female under vitamin K antagonists for cardiac arrhythmia. Diffuse abdominal pain, abdominal anterior wall mass and anemia. **(a)** Iodinated contrast-enhanced CT image obtained at the portal phase in axial plane shows an anterior abdominal wall muscle hematoma with a fluid-fluid level and contrast extravasation. **(b)** Selective angiogram of left inferior epigastric artery shows two distal areas of contrast extravasation. **(c)** Selective angiogram shows an occlusion after embolization with coils and particles with no residual extravasation.

Outcome

Primary clinical success of embolization was defined as bleeding cessation with hemodynamic stability (normal blood pressure, decreased heart rate, weaning of catecholamine support) and serum hemoglobin level stabilization with decreased requirement for red blood cells units transfusion.

In case of persistent or recurrent bleeding (defined as clinical failure after embolization and confirmed increasing volume of the hematoma and/or persistent active bleeding on CT), a second embolization was performed. The delay and the artery embolized were noted. Secondary clinical success was defined as hemodynamic and biological stabilization after a second session embolization.

The 30-day mortality was recorded.

Statistical analysis

Continuous variables were described by their mean and standard deviation. Qualitative variables were expressed as absolute and relative frequencies. Continuous and categorical variables were compared by using the Student t test and χ^2 test, respectively. Association with main outcome was studied by an univariate Cox model for both continuous and categorical variables. Survival was assessed by the Kaplan-Meier estimator. The log-rank test was used to compare survival curves. Overall survival was calculated from the date of embolization until death from any cause within the 30 days following the embolization. Follow-up information was available for all patients until death or censored at 30 days. Univariable analyses were conducted by using a Cox proportional hazards model to identify potential prognostic factors of survival and to estimate adjusted hazard ratios (HRs) and their 95% CI. Covariates and confounders were taken into account in the multivariable Cox proportional hazards model. A p value less than 0.05 was considered indicative of statistical significance.

Results

Demographic data

A total of 219 patients meeting the inclusion criteria were included. Patient characteristics are listed in Table 1.

There were 113 (52%) men and 106 (48%) women. Mean age 78.2 ± 10.7 years. Eighty percent of patients had previous history of hypertension and 91.2% had a heart disease, mostly rhythmic cardiopathy encountered in 67.9% of patients.

All patients were receiving anticoagulant therapy with 45.6% treated with vitamin K antagonist, 36.9% receiving heparin, 6.5% receiving a direct oral anticoagulant, and 11.0% treated with a combination of two treatments (anticoagulant bridging, acute myocardial infarction).

At the admission, 41 of 219 (18.9%) patients were hemodynamically unstable.

A total of 75.3 % of patients were anemic with a mean hemoglobin level of 8.6 ± 2.2 g/dL.

Table 1: patient characteristics

Sex	
female	106 (48)
male	113 (52)
Age	78.2 ± 10.7 (80.6; 24.3-97.3)
Body mass index	26.6 ± 5.9 (26.2; 12.5-47.1)
Hemodynamic instability n=217	41 (18.9)
systolic arterial pressure	118.8 ± 28.4 (120.0; 60.0-193.0)
diastolic arterial pressure	62.6 ± 15.5 (63.0; 30.0-140.0)
heart rate	88.0 ± 20.8 (87.0; 30.0-150.0)
Previous history	
hypertension n=210	168 (80.0)
cardiopathy n=193	176 (91.2)
cardiac arrhythmia	131 (67.9)
other cardiopathy	66 (34.2)
diabetes mellitus	47 (24.3)
cancer - hemopathy	20 (10.4)
Anticoagulation n=217	
heparin	80 (36.9)
vitamin K antagonist	99 (45.6)
direct oral anticoagulant	14 (6.5)
combination	24 (11.0)
Indication for anticoagulation n=210	
cardiac arrhythmia	123 (58.6)
cardiac valve	17 (8.1)
venous thrombosis	43 (20.5)
associated indications	6 (2.8)
other	21 (10.0)
Anticoagulant overdose n=186	82 (44.1)
Antiplatelet therapy n=215	66 (30.7)
Biological evaluation n=211	
hemoglobin level (g/dL)	8.6 ± 2.2 (8.3; 3.5-14.6)
anemia	159 (75.3)
platelet count (per mm ³)	273.5 ± 131.8 (250.0; 17.0-795.0)
prothrombin time (%)	46.0 ± 29.7 (45.0; 10.0-100.0)
international normalized ratio	3.3 ± 2.5 (2.2; 1.0-11.0)
renal clearance (ml/mn)	50.4 ± 27.7 (48.0; 4.0-146.0)

Quantitative data are expressed in mean ± standard deviation (median;range).
Qualitative data are expressed in proportion (percentage).

All patients had CT examination, among which 198 of 219 (90.4%) underwent contrast-enhanced administration. The remaining patients underwent non-contrast material-enhanced CT only because of renal function impairment (estimated glomerular filtration rate <30 mL/mn). Hematomas occurred most commonly in the retroperitoneum in 100 of 219 (45.7%) patients and rectus sheath muscles in 73 of 219 (33.3%) patients (Table 2). The mean hematoma volume was 637.9 ± 534.9 cm³ and active bleeding was found in 100 of 198 (50.5%) patients. When active bleeding was noted, the contrast extravasation was identified at the arterial phase in 65 of 95 (64.3%) patients, and at the venous phase in 98 of 99 (98%) patients.

Table 2: hematoma characteristics on CT

Hematoma location (%) n=219	
anterior	73 (33.3)
retroperitoneal	100 (45.7)
gluteal	13 (5.9)
thigh	33 (15.1)
Hematoma characteristics n=218	
unique	205 (94)
multiple	13 (6)
long diameter (mm)	180.9 ± 58.6 (178; 58-363)
surface (cm ²)	135.8 ± 72.7 (121.0; 14.0-515.0)
volume (cm ³)	637.9 ± 534.9 (487.5; 28.0-4,316.0)
active bleeding at CT n=198	100 (50.5)
extravasation at arterial phase n=95	65 (64.3)
extravasation at venous phase n=99	98 (99)
single extravasation n=99	50 (50.5)
multiple extravasations n=99	49 (49.5)
ruptured hematoma n=219	48 (21.9)

Quantitative data are expressed in mean \pm standard deviation (median;range). Qualitative data are expressed in proportion (percentage).

Group comparison

A total of 157 patients received conservative management and 62 were treated with arterial embolization. There was no significant difference concerning sex, age and previous history of patients (Table 3).

Anticoagulation therapy was different between the two groups: more than half of patients managed conservatively were receiving vitamin K antagonist (79 of 157, 50.4%) versus a third of embolized patients (20 of 60, 33.0%) ($p=0.024$). They had higher INR values 3.7 ± 2.7 versus 2.2 ± 1.7 ($p<0.001$).

There were more patients receiving a combination of two anticoagulation treatments in the group treated with embolization 12 out of 60 (20.0%) versus 12 out of 157 (7.6%) in the conservative management group (p=0.009).

Clinical-biological presentation was also different between the two groups: there were significantly more patients with hemodynamic instability in the embolization group (25 out of 60, 41.7%) than in the conservative management group (16 of 157, 10.2%) (p<0.001). Hemoglobin level was lower in the embolized group (7.9 ± 1.9 g/dL) than in the conservative management group (8.8 ± 2.2 g/dL) (p = 0.009). There was no significant difference in platelet count and eGFR at admission between the two groups (Table 3).

Table 3: patient characteristics

	Embolization group n=62	Non embolized group n=157	p value	OR [IC95]
Sex				
female	33 (53)	73 (46.5)	0.45	
male	29 (47)	84 (53.5)		
Age	76.3 ± 9.2 (78.5; 47.8-87.9)	79.0 ± 11.2 (81.7; 24.3-97.3)	0.087	
Body mass index	26.6 ± 5.6 (25.1, 16.8-46.0) n=50	26.6 ± 6.1 (26.4; 12.5-47.1) n=123	0.948	
Hemodynamic parameters				
instability	25/60 (41.7)	16/157 (10.2)	< 0.001	
systolic arterial pressure	105.2 ± 29.6 (101.5; 60.0-193.0)	123.5 ± 26.4 (126.0; 67.0-192.0)	< 0.001	
diastolic arterial pressure	60.2 ± 15.7 (63.0; 31.0-108.0)	63.4 ± 15.3 (63.0; 30.0- 140.0)	0.237	
heart rate	99.6 ± 21.4 (101.0; 60.0-150.0)	84.0 ± 19.1 (82.5; 30.0-139.0)	< 0.001	
Previous history				
hypertension	47/57 (82.5)	121/153 (79.1)	0.587	
cardiopathy	46/50 (92.0)	130/143 (90.9)	1	1,1492 [0,3326;5,0832]
cardiac arrhythmia	33/50 (66.0)	98/143 (68.5)	0.741	
other cardiopathy	29/50 (58.0)	82/143 (57.3)	0.935	
diabetes mellitus	12/50 (24.0)	35/143 (24.5)	0.946	
cancer - hemopathy	8/50 (16.0)	12/143 (8.4)	0.128	
Anticoagulation				
heparin	26/60 (43.0)	54/157 (34.4)	0.222	
vitamin K antagonist	20/60 (33.0)	79/157 (50.4)	0.024	
direct oral anticoagulant	2/60 (4.0)	12/157 (7.6)	0.359	0.418 [0.0441;1.9668]
combination	12/60 (20.0)	12/157 (7.6)	0.009	
Indication for anticoagulation				
cardiac arrhythmia	26/57 (45.6)	97/153 (63.4)	0.019	
cardiac valve	9/57 (15.8)	8/153 (5.2)	0.020	3.374 [1.088;10.676]
venous thrombosis	15/57 (26.3)	28/153 (18.3)	0.200	
associated indications	3/57 (5.3)	3/153 (2.0)	0.347	2.762 [0.359;21.2539]
other	4/57 (7.0)	17/153 (11.1)	0.379	
Anticoagulant overdose	13/51 (25.5)	69/135 (51.1)	0.00169	
Antiplatelet therapy	17/58 (29.3)	49/157 (31.2)	0.788	
Biological evaluation				
hemoglobin level (g/dL)	7.9 ± 1.9 (7.6; 4.1-12.8)	8.8 ± 2.2 (8.6; 3.5-14.6)	0.009	
anemia	48/55 (87.3)	111/156 (71.2)	0.017	
platelet count (per mm3)	267.8 ± 134.7 (234.0; 55.0-786.0)	275.3 ± 131.3 (261.5; 17-795.0)	0.733	
prothrombin time (%)	58.2 ± 24.6 (65.0; 10.0-97.0)	41.5 ± 30.2 (29.5; 10.0-100.0)	< 0.001	
activated partial thromboplastin time ratio	1.7 ± 1.2 (1.2; 0.8-7.0)	1.7 ± 1.1 (1.4; 0.6-8.0)	0.954	
international normalized ratio	2.2 ± 1.7 (1.5; 1.0-10.0)	3.7 ± 2.7 (3.0; 1.0-11.0)	< 0.001	
renal clearance (mL/mn)	48.7 ± 26.8 (39.0; 8.0-98.0)	50.9 ± 28.0 (48.0; 4.0-146.0)	0.634	

Quantitative data are expressed in mean ± standard deviation (median;range). Qualitative data are expressed in proportion (percentage).

Imaging findings were significantly different in the two groups (Table 4). Patients treated with embolization had larger hematomas with a longest diameter of 214.8 mm \pm 51.2 mm versus 167.7 \pm 56.0 mm ($p < 0.001$), a wider surface of 181.5 \pm 75.8 cm² versus 118.1 \pm 63.4 cm² and a larger volume of 1,023.7 \pm 653.4 cm³ versus 488.0 \pm 391.1 cm³ ($p < 0.001$).

There were more active bleeding on CT in the embolization group (51 out of 59, 86.4%) than in the conservative management group (49 out of 138, 35.5%) ($p < 0.001$) and a higher rate of extravasation at the arterial phase (41 of 48, 85.4% versus 24 of 47, 51.0%) ($p < 0.001$).

Table 4: imaging findings

	Embolization group n=62	Non embolized group n=157	p value
Hematoma location			
anterior	26/62 (41.9)	47/157 (29.9)	0.089
retroperitoneal	35/62 (56.5)	65/157 (41.4)	0.043
gluteal	0 /62 (0.0)	13/157 (8.3)	0.021
thigh	1/62 (1.6)	32/157 (20.4)	<0.001
Hematoma characteristics			
unique	59/61 (96.7)	146/157 (93.0)	0.523
multiple	2/61 (3.3)	11/157 (7.0)	
long diameter (mm)	214.8 \pm 51.2 (212.0; 113.0-363.0)	167.7 \pm 56.0 (164.0; 58.0-331.0)	<0.001
surface (cm ²)	181.5 \pm 75.8 (182.0; 44.0-515.0)	118.1 \pm 63.4 (106.0; 14.0-405.0)	<0.001
volume (cm ³)	1,023.7 \pm 653.4 (1,017.0; 79.0-4,316.0)	488.0 \pm 391.1 (373.0; 28.0-2,530.0)	<0.001
active bleeding at CT	51/59 (86.4)	49/138 (35.5)	< 0.001
extravasation at arterial phase	41/48 (85.4)	24/47 (51.0)	< 0.001
extravasation at portal phase	49/50 (98.0)	49/49 (100.0)	1
single extravasation	12/50 (24.0)	32/49 (65.3)	< 0.001
multiple extravasations	32/50 (64.0)	17/49 (34.7)	0.003
ruptured hematoma	28/62 (45.2)	20/157 (12.7)	< 0.001

Quantitative data are expressed in mean \pm standard deviation (median;range).

Qualitative data are expressed in proportion (percentage).

Embolization procedure

The embolization procedure data are summarized in Table 5. Contrast extravasation was detected on the initial angiogram in 37 out of 61 (60.7%) patients. The target artery was embolized in 37 out of 37 (100%) cases, and when no contrast extravasation was found, an empiric embolization was performed in 20 of 21 (95.2%) patients.

Technical success rate was 93.5% (58 out of 62 patients). The reasons for technical failures were catheterization failure (n=2), spastic artery (n=1), and patient agitation (n=1).

Primary clinical success rate was 75% (42 out of 56 patients) with a recurrence of bleeding in 12 out of 56 (21.4%) patients. Secondary clinical success rate after a repeat embolization was 92.5% (49 out of 53 patients).

Two major complications grade E according to the SIR classification occurred. Two patients presented transient medullar ischemia confirmed by MRI with no sequelae in the long-term (Fig 6).

Nine patients among which 8 had an embolization, required surgery for several reasons: persistent bleeding after embolization with hemodynamic instability in 2 cases, abdominal compartment syndrome in 3 cases, neurological deficit in 2 cases and mesenteric ischemia induced by low cardiac output in 2 cases.

Table 5: angiographic data

Angiography	62/219 (28.3%)
Contrast extravasation on angiogram	37/61 (60.7%)
Embolization of target artery	37/37 (100%)
Empiric embolization	20/21 (95.2%)
Main embolized artery	
inferior epigastric	22/57 (38.6%)
lumbar	28/57 (49.1%)
deep circumflex iliac	6/57 (10.5%)
ilio-lumbar	1/57 (1.8%)
Embolization agent	
particular	34/57 (59.7%)
liquid	6/57 (10.5%)
coils	4/57 (7%)
combination	13/57 (22.8%)
Technical success	58/62 (93.5%)
Primary clinical success	42/56 (75%)
Recurrent bleeding	12/56 (21.4%)
Secondary clinical success	49/53 (92.5%)
Major complication (SIR classification)	2/62 (3.2%)

Qualitative data are expressed in proportion (percentage).

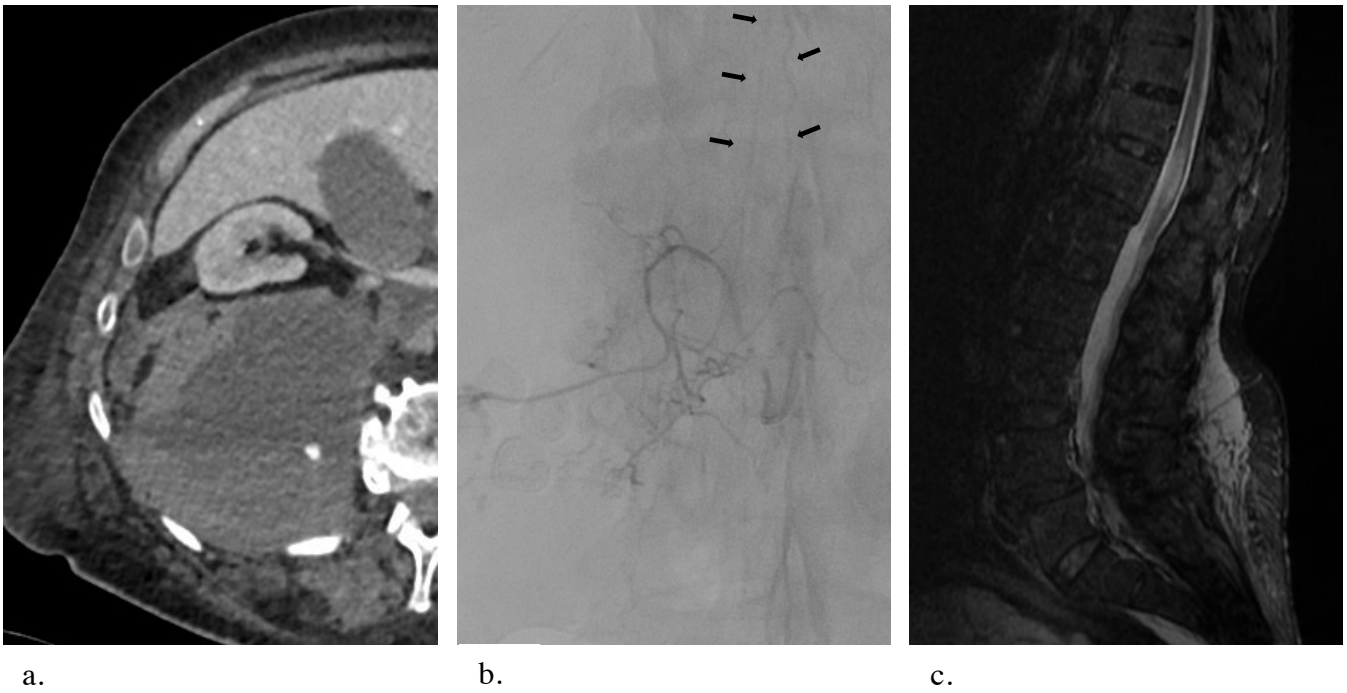


Figure 6: 86-year-old female. Hemorrhagic shock under dual antiplatelet therapy and anticoagulant treatment for a myocardial infarction. **(a)** Iodinated contrast-enhanced CT image obtained at the portal phase in axial plane shows a retroperitoneal hematoma with contrast extravasation. **(b)** Selective angiogram of second right lumbar artery demonstrates the anterior radiculo-medullary artery, which was not easily identified during the procedure due to the patient movements. Embolization of L1 to L4 lumbar arteries was performed using large 500-700 μm microspheres. **(c)** Lumbar MRI image in sagittal plane, performed for occurrence of a paraplegia shows a spinal cord infarction of the conus medullaris. The patient made a full recovery.

Outcome

For the whole population, variables significantly associated with mortality in univariate analysis (listed in Table 6) were renal clearance ($p=0.00059$) and ruptured hematoma (HR 1.83, CI 1.04-3.24, $p=0.0369$).

Univariate analysis on mortality in the first thirty days following initial CT (Table 7) revealed that hematoma surface ($p=0.00999$) and volume ($p=0.0375$) were significantly associated with early mortality.

Multivariate analysis on overall mortality (Table 8) has not demonstrated any significant difference.

In patients treated with embolization, variables significantly associated with mortality in univariate analysis (listed in Table 9) included renal clearance ($p=0.0338$), hematoma surface ($p=0.00838$), and hematoma volume ($p=0.0416$).

Embolization was not associated with increased or decreased mortality in univariate and multivariate analysis.

Table 6: univariate analysis on mortality

	Alive (n=145)	Dead (n=57)	beta	HR (95% CI)	p-value
Sex					
female	72 (49.7)	25 (43.9)			
male	73 (50.3)	32 (56.1)	0.2346	1.2644 (0.75-2.14)	0.382
Body mass index	26.40 (5.68)	26.69 (6.34)	0.003759	1.003766 (0.95-1.05)	0.883
Hypertension					
no	33 (22.9)	7 (12.7)			
yes	111 (77.1)	48 (87.3)	0.4947	1.6400 (0.74-3.63)	0.222
Previous history					
cardiac arrythmia	73 (57.0)	30 (55.6)	-0.1242	0.8832 (0.34-2.28)	0.798
other cardiopathy	43 (33.6)	19 (35.2)	-0.1423	0.8674 (0.32-2.33)	0.778
Hemoglobin level (g/dL)	8.59 (2.27)	8.43 (2.14)	-0.0286	0.9718 (0.86-1.10)	0.631
Platelet count (G/L)	286.26 (128.64)	247.65 (121.52)	-0.002237	0.997766 (0.99-1)	0.072
Renal clearance (mL/mn)	54.60 (28.01)	38.31 (20.23)	-0.018916	0.981262 (0.97-0.99)	0.00059
Indication for anticoagulant					
cardiac arrythmia	80 (57.1)	36 (64.3)			
venous thrombosis	31 (22.1)	8 (14.3)	-0.408643	0.664552 (0.3-1.43)	0.296
cardiac valve	10 (7.1)	5 (8.9)	0.038603	1.039358 (0.40-2.68)	0.936
Prothrombin time	45.11 (30.69)	45.98 (28.29)	0.002435	1.002438 (0.99-1.01)	0.599
International normalized ratio	3.49 (2.67)	2.94 (2.21)	-0.08794	0.91581 (0.81-1.04)	0.169
Anticoagulant overdose					
no	66 (52.8)	29 (56.9)			
yes	59 (47.2)	22 (43.1)	-0.2233	0.7999 (0.46-1.40)	0.432
Hematoma location					
anterior	43 (29.7)	19 (33.3)			
gluteal or thigh	34 (23.4)	11 (19.3)	-0.44071	0.64358 (0.30-1.36)	0.248
retroperitoneal	68 (46.9)	27 (47.4)	-0.03159	0.96890 (0.54-1.74)	0.916
Hematoma long diameter (mm)	179.15 (57.57)	178.04 (57.80)	0.0004461	1.0004462 (0.99-1.00)	0.842
Hematoma surface (cm2)	128.58 (61.65)	139.55 (88.74)	0.002203	1.002206 (0.99-1.00)	0.202
Hematoma volume (cm3)	579.37 (430.31)	698.30 (706.88)	0.0004145	1.0004146 (1-1.001)	0.0635
Extravasation at arterial phase					
no	17 (29.3)	9 (33.3)			
yes	41 (70.7)	18 (66.7)	0.1475	1.1590 (0.5-2.59)	0.719
Extravasation at portal phase					
no	1 (1.7)	0 (0.0)			
yes	58 (98.3)	27 (100.0)			
Ruptured hematoma					
no	117 (80.7)	40 (70.2)			
yes	28 (19.3)	17 (29.8)	0.6061	1.8333 (1.04-3.24)	0.0369
Embolization					
no	112 (77.2)	40 (70.2)			
yes	33 (22.8)	17 (29.8)	0.3584	1.4310 (0.81-2.52)	0.216

Quantitative data are expressed in mean \pm standard deviation (median;range). Qualitative data are expressed in proportion (percentage).

Table 7: univariate analysis on early mortality

	> 30 days (n=36)	< 30 days (n=14)	beta	HR (95% CI)	p-value
Sex					
female	16 (44.4)	10 (71.4)			
male	20 (55.6)	4 (28.6)	-0.8812	0.4143 (0.1-1.35)	0.143
Body mass index	25.90 (4.46)	27.85 (6.79)	0.06482	1.06696 (0.96-1.19)	0.242
Hypertension					
no	7 (19.4)	1 (7.7)			
yes	29 (80.6)	12 (92.3)	1.929e+01	2.394e+08 (–)	0.998
Previous history					
cardiac arrythmia	14 (46.7)	6 (46.2)	0.01719	1.01734 (0.12-8.71)	0.987
other cardiopathy	13 (43.3)	6 (46.2)	0.22426	1.25140 (0.15-10.40)	0.836
Hemoglobin (g/dL)	7.95 (2.05)	7.58 (2.02)	-0.1565	0.8552 (0.62-1.17)	0.332
Platelet count (G/L)	269.23 (110.19)	258.75 (117.93)	-0.0008663	0.9991341 (0.99-1.005)	0.751
Renal clearance (mL/mn)	50.93 (27.96)	39.00 (24.92)	-0.02220	0.97805 (0.95-1.00)	0.115
Indication for anticoagulant					
cardiac arrythmia	15 (42.9)	6 (46.2)			
venous thrombosis	10 (28.6)	2 (15.4)	-5.419e-01	5.816e-01 (0.12-2.88)	0.507
cardiac valve	4 (11.4)	4 (30.8)	5.772e-01	1.781e+00 (0.50-6.33)	0.373
Prothrombin time (%)	55.62 (27.29)	59.50 (21.19)	0.00592	1.00594 (0.98-1.03)	0.607
International normalized ratio	2.49 (2.02)	1.85 (0.79)	-0.3715	0.6897 (0.38- 1.27)	0.23
Anticoagulant overdose					
no	20 (64.5)	11 (84.6)			
yes	11 (35.5)	2 (15.4)	-1.6241	0.1971 (0.025-1.53)	0.12
Hematoma location					
anterior	12 (33.3)	6 (42.9)			
gluteal or thigh	1 (2.8)	0 (0.0)	-1.709e+01	3.797e-08 (0-Inf)	0.998
posterior	23 (63.9)	8 (57.1)	-7.673e-02	9.261e-01 (0.30-2.83)	0.893
Hematoma long diameter (mm)	205.03 (48.43)	228.86 (55.04)	0.009043	1.009084 (0.99-1.02)	0.0752
Hematoma surface (cm2)	158.31 (55.19)	216.93 (106.48)	0.006199	1.006218 (1.001-1.011)	0.00999
Hematoma volume (cm3)	900.74 (506.45)	1296.29 (966.50)	0.0005555	1.0005556 (1-1.001)	0.0375
Extravasation at arterial phase					
no	5 (16.7)	0 (0.0)			
yes	25 (83.3)	10 (100.0)			
Extravasation at portal phase					
no	1 (3.4)	0 (0.0)			
yes	28 (96.6)	10 (100.0)			
Ruptured hematoma					
no	18 (50.0)	6 (42.9)			
yes	18 (50.0)	8 (57.1)	0.1126	1.1192 (0.37-3.33)	0.84

Quantitative data are expressed in mean \pm standard deviation.
Qualitative data are expressed in proportion (percentage).

Table 8: multivariate analysis on mortality

	beta	HR	HR CI lower .95	HR CI upper .95	p-value
Sex					
female					
male	7.105e-01	2.035	0.74453	5.563	0.1661
Body mass index					
	-1.276e-02	0.9873	0.90751	1.074	0.7667
Indication for anticoagulant					
cardiac arrythmia					
venous thrombosis	-9.783e-02	0.9068	0.24113	3.410	0.8849
cardiac valve	-1.228e+00	0.2927	0.03009	2.848	0.2899
Hematoma long diameter (mm)					
	-7.001e-03	0.9.930	0.97997	1.006	0.2996
Hematoma surface (cm2)					
	7.600e-03	1.008	0.99320	1.022	0.3018
Hematoma volume (cm3)					
	7.616e-05	1.000	0.99842	1.002	0.9282
Recurrent bleeding after first embolization					
no					
yes	1.393e+00	4.025	0.93119	17.402	0.0622
Hematoma location					
anterior					
gluteal or thigh	-2.231e-01	0.8001	0.13934	4.594	0.8025
posterior	6.914e-01	1.996	0.56781	7.020	0.2812
Extravasation at arterial phase					
no					
yes	5.918e-02	1.061	0.34356	3.276	0.9181
Embolization					
no					
yes	-1.143e+00	0.3189	0.07279	1.397	0.1294
Ruptured hematoma					
no					
yes	3.108e-01	1.365	0.38605	4.823	0.6295

Table 9: univariate analysis on mortality in patients treated by embolization

	Alive (n=33)	Dead (n=17)	beta	HR (95% CI)	p-value
Sex					
female	17 (51.5)	9 (52.9)			
male	16 (48.5)	8 (47.1)	0.1864	0.8300 (0.322-1.6)	0.702
Body mass index	25.66 (4.35)	27.95 (6.39)	0.07637	1.07937 (0.98-1.19)	0.126
Hypertension					
no	7 (21.2)	1 (6.2)			
yes	26 (78.8)	15 (93.8)	1.085	2.961 (0.40-22.46)	0.294
Previous history					
cardiac arrhythmia	13 (48.1)	7 (43.8)	0.3013	1.3516 (0.17-11.01)	0.778
other cardiopathy	11 (40.7)	8 (50.0)	0.4201	1.5221 (0.189-12.21)	0.692
Hemoglobin level (g/dL)	8.12 (2.21)	7.31 (1.52)	-0.0286	0.9718 (0.86-1.09)	0.631
Platelet count (G/L)	277.07 (109.09)	246.73 (115.80)	-0.002135	0.997867 (0.99-1.00)	0.379
Renal clearance (mL/mn)	55.22 (29.48)	33.67 (16.15)	-0.02534	0.97498 (0.95-0.99)	0.0338
Indication for anticoagulant					
cardiac arrhythmia	13 (40.6)	8 (50.0)			
venous thrombosis	10 (31.2)	2 (12.5)	-0.8139	0.4431 (0.09-2.09)	0.303
cardiac valve	4 (12.5)	4 (25.0)	0.3044	1.3558 (0.41-4.52)	0.620
Prothrombin time (%)	58.28 (27.26)	54.38 (23.00)	-0.00112	0.99888 (0.98-1.02)	0.906
International normalized ratio	2.45 (2.06)	2.03 (1.08)	-0.1515	0.8594 (0.61-1.22)	0.395
Anticoagulant overdose					
no	18 (64.3)	13 (81.2)			
yes	10 (35.7)	3 (18.8)	-0.6972	0.4980 (0.14-1.75)	0.277
Hematoma location					
anterior	12 (36.4)	6 (35.3)			
gluteal or thigh	0 (0.0)	1 (5.9)	1.050466	2.858982 (0.34-23.97)	0.333
posterior	21 (63.6)	10 (58.8)	-0.005118	0.994895 (0.36-2.74)	0.992
Hematoma long diameter (mm)	205.12 (48.36)	224.47 (54.86)	0.006863	1.006886 (0.99- 1.02)	0.13
Hematoma surface (cm2)	158.16 (53.12)	206.88 (103.53)	0.006185	1.006204 (1.002-1.011)	0.00838
Hematoma volume (cm3)	892.12 (508.31)	1242.71 (903.28)	0.0005304	1.0005306 (1-1.001)	0.0416
Extravasation at arterial phase					
no	5 (17.9)	0 (0.0)			
yes	23 (82.1)	12 (100.0)			
Extravasation at portal phase					
no	1 (3.7)	0 (0.0)			
yes	26 (96.3)	12 (100.0)			
Ruptured hematoma					
no	15 (45.5)	9 (52.9)			
yes	18 (54.5)	8 (47.1)	-0.1473	0.8630 (0.33-2.24)	0.762
Extravasation on initial angiogram					
no	12 (36.4)	6 (37.5)			
yes	21 (63.6)	10 (62.5)	-0.007501	0.992527 (0.36-2.73)	0.988
Recurrent bleeding after first embolization					
no	27 (81.8)	12 (70.6)			
yes	6 (18.2)	5 (29.4)	0.4401	1.5528 (0.54- 4.43)	0.41

Quantitative data are expressed in mean \pm standard deviation.
Qualitative data are expressed in proportion (percentage).

Discussion

Abdominopelvic muscle hematomas are among the most common sites of anticoagulant-related hematomas (5,6). In recent years, their incidence have increased because of the broader use of anticoagulants therapies (1,3,7,16,21). They can be associated with high rates of morbidity and mortality particularly when diagnosis and treatment are delayed or insufficient (2,7,8,18,22).

The clinical diagnosis is difficult with unspecific symptoms. Iodinated contrast-enhanced CT should be performed to confirm the suspicion of abdominopelvic hematoma in elderly patients presenting with abdominal pain, abdominal wall mass, hypotension and anemia (7,8). In our study all cases were diagnosed using CT.

There are two main strategies to manage patients with spontaneous abdominopelvic hematomas related to anticoagulation therapies (1,7,10,12,21). Conservative management consists in fluid resuscitation, blood transfusion, discontinuation or reversal of anticoagulation treatment, and careful monitoring. A more invasive strategy consists in the use of percutaneous trans-arterial embolization. Open surgery can be a third option in specific cases. Few treatment algorithms have been reported for managing patients but there is no consensus opinion on the optimal strategy.

Previous studies have evaluated conservative management and reported its efficacy in a majority of patients (14,16–18,22). Factors associated with failure include requirement for blood transfusion, rate of hemoglobin decrease, volume of hematoma and presence of contrast extravasation on CT (13,21,22). In addition, a muscular fascia rupture has been suggested as an indicator for emergent intervention (18,24,25).

Arterial embolization is usually favored in hemodynamically unstable patients or after failure of conservative management (15,19,26). Embolization is an effective and safe endovascular procedure to control bleeding, with a high technical success rate ranging from 75 to 100% and an acceptable clinical success rate ranging from 57 to 96% (8,14,15,17,22,27).

No validated guidelines on the optimal management of spontaneous abdominopelvic hematomas are available. The respective efficacy of arterial embolization and conservative management have not been evaluated in patients with various initial clinical presentations.

In our study, we compared the clinical, biological and imaging findings of patients either treated conservatively or with arterial embolization. Patients with severe initial clinical presentation notably hemodynamic instability, lower systolic blood pressure, tachycardia were mainly referred for arterial embolization. The type of anticoagulation therapy was significantly different between the two groups with a higher frequency of patients receiving vitamin K antagonists and a higher rate of overdose in the conservative management group. Correction of blood

parameters before considering arterial embolization is key in this subgroup of patients. CT demonstrated that patients treated with embolization had larger hematomas, more contrast extravasation seen at the arterial phase of CT, and more often a muscular fascia rupture with extended hemorrhage. These findings are obvious indications for embolization because of the imaging severity and the identification of target territories and arteries. These results are also consistent with previous studies (13,21).

The technical success rate of embolization was 93.5% and the primary clinical success rate was 75%, consistent with the literature (8,14,15,17,22,27). In our center, the target artery is embolized super-selectively in case of contrast extravasation on the initial angiogram, and the anastomotic arteries potentially accounting for revascularization of the bleeding site (e.g., embolization of adjacent lumbar arteries in retroperitoneal hematomas). If no contrast extravasation was noticed, an empiric embolization was almost always performed based on the abnormalities identified on CT.

Using univariate analysis, renal clearance and ruptured hematoma were significantly associated with an increased mortality. Hematoma surface and volume were associated with early mortality in the thirty days after the initial CT. These findings are consistent with previous studies that identified the volume of hematomas as a prognosis factor for poor outcome (17,18,22,27).

Conversely, we have not identified any variable associated with an increased mortality using multivariate analysis, in particular, the use of embolization was not associated with a poorer outcome. It may be hypothesized that patients properly selected for arterial embolization have a rather favorable outcome. The all cause 30-day mortality rate in patients treated with embolization was 22.6%, similar to the rates reported in the literature (13,14,16,17).

Our study has some limitations. The study was designed as retrospective and the two groups of patients were not randomized. Indication for arterial embolization was taken on a case-by-case basis considering the initial clinical, biological and imaging presentation. Nevertheless, we succeeded in enrolling 219 patients with 62 treated with embolization.

In conclusion, multiphasic iodinated contrast-enhanced CT should be obtained in patients under anticoagulation therapy suspected to have a spontaneous muscular hematoma.

In hemodynamically unstable patients, when CT exhibits a large hematoma with contrast extravasation at the arterial phase and/or muscular fascia rupture, embolization should be carried out immediately. In stable patients, many factors should be taken into consideration for an optimal management. An overdose of anticoagulation therapy should always be corrected as it seems to be an important part of a successful conservative management.

CT images should be carefully evaluated to identify abnormalities i.e., large volume, extravasation at the arterial phase, ruptured hematoma which may lead to arterial embolization even in patients with a less severe initial presentation.

Although difficult to conduct in patients requiring emergency management, randomized controlled-trials are necessary to identify factors associated with poor outcomes in patients treated conservatively or embolized.

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Conclusion

Les hématomes musculaires abdomino-pelviens de survenue spontanée sous traitement anticoagulant peuvent se présenter sous diverses formes et menacer le pronostic vital des patients.

Un scanner multiphasique avec injection de produit de contraste iodé devrait être réalisé chez tous les patients sous traitement anticoagulant chez lesquels il existe une suspicion clinique d'hématome musculaire.

Chez les patients instables pour lesquels le scanner objective un hématome de volume important avec extravasation de produit de contraste à la phase artérielle et/ou une rupture du fascia musculaire, une embolisation devrait être effectuée immédiatement.

Un surdosage en traitement anticoagulant devrait systématiquement être corrigé, cette correction semble constituer un facteur important de succès du traitement médical seul.

Chez des patients ayant une présentation clinique moins sévère, certaines anomalies scanographiques telles qu'un hématome de volume important, une extravasation de produit de contraste à la phase artérielle ou une rupture du fascia musculaire, peuvent faire poser l'indication d'embolisation.

Des essais cliniques randomisés, difficiles à conduire chez des patients nécessitant une prise en charge urgente, seraient nécessaires pour identifier des facteurs prédictifs de mortalité chez les patients traités médicalement ou par embolisation artérielle.

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Le Doyen

ANNEE DE SOUTENANCE : 2021

NOM ET PRENOM DE L'AUTEUR : MOYNE Thibault

TITRE DE LA THESE : Embolisation artérielle versus traitement médical dans la prise en charge des hématomes musculaires abdomino-pelviens spontanés sous traitement anticoagulant.

RESUME DE LA THESE EN FRANÇAIS :

Objectifs : Évaluer les caractéristiques cliniques, biologiques et de l'imagerie chez les patients admis pour hématome musculaire abdomino-pelvien spontané sous anticoagulant en fonction de la stratégie de traitement. Évaluer efficacité et sécurité du traitement médical et de l'embolisation artérielle.

Méthodes : Entre 2015 et 2020, 219 patients ont été inclus et 62 ont été traités par embolisation. Les données démographiques, cliniques et biologiques à l'admission, caractéristiques de l'hématome au scanner et éléments de la prise en charge ont été relevés. Le succès clinique était défini par un arrêt du saignement avec stabilisation hémodynamique et du taux d'hémoglobine. Des analyses univariées et multivariées ont été effectuées pour évaluer la mortalité.

Résultats : Les patients avec présentation sévère étaient plus traités par embolisation. Les patients traités médicalement avaient plus d'anti-vitamine K. Les patients embolisés avaient des hématomes plus volumineux, plus de saignement actif et de rupture de fascia musculaire au scanner. Les taux de succès technique et clinique étaient de 93.5% et 75%. La mortalité à 30 jours après embolisation était de 22.6%. Une rupture du fascia musculaire et un hématome de volume important étaient associés à une mortalité augmentée.

Conclusions : Une embolisation devrait être réalisée immédiatement chez les patients instables avec hématome volumineux au scanner, saignement actif et/ou rupture du fascia musculaire. Un surdosage en anticoagulant devrait être corrigé rapidement. Chez les patients avec une présentation moins sévère de nombreux facteurs devraient être considérés pour un traitement optimal.

MOTS CLES : hématome-hématome musculaire-paroi abdominale-anticoagulant-radiologie interventionnelle-embolisation artérielle

TITRE DE LA THESE EN ANGLAIS : Arterial Embolization vs Conservative Treatment in the Management of Spontaneous Abdominal and Pelvic Muscle Hematomas due to Anticoagulation Therapy

RESUME DE LA THESE EN ANGLAIS :

Objectives: To compare the clinical presentation, biological and imaging findings of patients admitted for anticoagulant-related abdominal or pelvic muscle hematoma according to the management strategy. To evaluate efficacy and safety of conservative management and arterial embolization.

Methods: Between 2015 and 2020, 219 patients were included and 62 were treated with arterial embolization. Demographic data, clinical and biological status at admission, CT findings and treatment elements were recorded. Clinical success was defined as bleeding cessation, hemodynamic stability and hemoglobin level stabilization. Univariate and multivariate analysis were performed to evaluate mortality.

Results: Patients with severe initial presentation were mainly treated with embolization. Patients managed conservatively were receiving more vitamin K antagonists. Patients treated with embolization had larger hematomas, more contrast extravasation and muscular fascia ruptures on CT. Technical success rate of embolization was 93.5%. Clinical success rate was 75%. All cause 30-day mortality rate after embolization was 22.6%. Muscular fascia rupture and large hematoma volume were associated with an increased mortality.

Conclusions: Embolization should be carried out immediately in hemodynamically unstable patients with a large hematoma, active bleeding, and/or muscular fascia rupture on CT. An overdose of anticoagulation therapy should always be corrected first. In patients with a less severe presentation, many factors should be taken into account for an optimal management.

KEY WORDS : hematoma-muscular hematoma-abdominal wall-anticoagulant-interventional radiology-arterial embolization