Symptomatic vertebral hemangiomas during pregnancy

Report of 2 cases

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Symptomatic vertebral hemangiomas during pregnancy are rare, as only 27 cases have been reported in the literature since 1948. However, symptomatic vertebral hemangiomas can be responsible for spinal cord compression, in which case they constitute a medical emergency, which raises management difficulties in the context of pregnancy. Pregnancy is a known factor responsible for deterioration of these vascular tumors. In this paper, the authors report 2 clinical cases of symptomatic vertebral hemangiomas during pregnancy, including 1 case of spontaneous fracture that has never been previously reported in the literature. The authors then present a brief review of the literature to discuss emergency management of this condition. The first case was a 28-year-old woman at 35 weeks of gestation, who presented with paraparesis. Spinal cord MRI demonstrated a vertebral hemangioma invading the body and posterior arch of T-3 with posterior epidural extension. Laminctomy and vertebroplasty were performed after cesarean section, allowing neurological recovery. The second case involved a 35-year-old woman who presented with spontaneous fracture of T-7 at 36 weeks of gestation, revealing a vertebral hemangioma with no neurological deficit, but it was responsible for pain and local instability. Treatment consisted of postpartum posterior interbody fusion. With a clinical and radiological follow-up of 2 years, no complications and no modification of the hemangiomas were observed. A review of the literature reveals discordant management of these rare cases, which is why the treatment course must be decided by a multidisciplinary team as a function of fetal gestational age and maternal neurological features.

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Key Words • pregnancy • spinal cord compression • vascular tumor • vertebral fracture • vertebral hemangioma

Vertebral hemangiomas are common benign vascular tumors of the spine, belonging to the group of hamartomas. According to postmortem and radiological data, the prevalence of vertebral hemangiomas would be 10%–12% in the general population.12,18 They are generally asymptomatic, as only 1% of hemangiomas are symptomatic in the general population.16 They can be responsible for simple back pain or may present as a diagnostic and therapeutic emergency with spinal cord compression, usually related to bone expansion within the vertebral canal.12,18

Pregnancy is a recognized factor in deterioration of hemangiomas.12,18 When vertebral hemangiomas become symptomatic during pregnancy, they very often present with signs of spinal cord compression, which nevertheless remains a rare complication. Since the first case report was published in 1948,13 only 27 cases have been reported in the literature. Vertebral hemangiomas are usually diagnosed during the third trimester and are most commonly located in the thoracic spine, raising problems of therapeutic management requiring a multidisciplinary approach to take into account the maternal neurological risk and the risk of fetal immaturity.

In this article, we report 2 cases of symptomatic vertebral hemangioma during pregnancy, which was responsible for spontaneous vertebral fracture in 1 case, a previously unreported complication. The main aspects of the treatment decision will then be discussed in light of a brief review of the literature.

Case Reports

Case 1

History and Presentation. The first case involved a 28-year-old woman with no notable medical history, presenting spontaneously at 35 weeks of gestation with back pain, paresthesia of the lower limbs, and progressive onset of gait disorders. A physical examination demonstrated an incomplete T-3 spinal cord compression syndrome (American Spinal Injury Association Grade D). This spinal cord compression syndrome was composed of spinal syndrome, with back pain scored 8/10 by the patient on a visual analog scale (VAS) of pain; a syndrome at the
level of the lesion, consisting of bilateral T-3 nerve root pain scored 7/10 on the VAS; and finally, a syndrome below the level of the lesion, consisting of posterior column syndrome and pyramidal syndrome with T-3 paraparesis (all lower limb nerve roots scored 4/5 according to the American Spinal Injury Association motor score), bilateral Babinski sign, and brisk Achilles and patellar deep tendon reflexes.

Emergency MRI revealed spinal cord compression due to invasion of the T-3 vertebra. The T-3 vertebra presented signal anomalies, with a high-intensity signal on T2-weighted imaging, low-intensity signal on T1-weighted imaging, and homogeneous enhancement of the vertebral body, pedicles, laminae, and spinous process associated with posterior epidural extension responsible for spinal cord compression. The same signal anomalies were observed in the anterior part of C-7, the posteroinferior corner of T-7, and the right pedicle of T-10 (Fig. 1A and B). After multidisciplinary case review, taking into account the gestational age of the baby and the dynamics of neurological deterioration, we decided to perform fetal extraction by emergency cesarean section to allow complete assessment and propose surgical treatment of this spinal cord compression.

Postpartum physical examination results remained unchanged. Chest, abdominal, and pelvic CT scans were performed, looking for other lesions to exclude the hypothesis of a metastatic lesion of T-3. No tumor was detected. In contrast, a modification of trabecular patterns was demonstrated in T-3, T-7, and T-10, corresponding to the MRI findings (Fig. 1C and D).

**Operation and Postoperative Course.** Emergency posterior decompression by a T2–4 laminectomy was performed. A very hemorrhagic spinous process of T-3 and a hypervascular posterior epidural lesion responsible for frank compression of the dural sheath at this level were observed at operation. Resection of this lesion was macroscopically complete and histological examination demonstrated a cavernous hemangioma with no signs of malignancy. The postoperative course was uneventful with complete neurological recovery.

Follow-up MRI performed at 3 months (Fig. 2A and B) demonstrated effective spinal cord decompression at T-3 with no residual compression at this level. However, this MRI revealed signs of postoperative myelopathy at T-3, but with no signs of progression of the other lesions described on the initial MRI. Ten months after the operation, due to persistence of back pain localized over T-3, a complementary vertebroplasty was performed on this vertebra to treat the hemangioma and stabilize the vertebra. No complications (in particular no cement leakage) were observed and the patient’s back pain was relieved. With a follow-up of 18 months, the patient was asymptomatic and imaging revealed a stable appearance of the lesions (Fig. 2C and D).

**Case 2**

**History and Presentation.** A 35-year-old woman attended the obstetrics emergency department at 36 weeks of gestation of her third pregnancy, with back pain radiating to the chest and left lower limb pain. This patient had a history of fibroadenoma of the left breast and thyroidectomy for thyroid adenocarcinoma. She reported progressive onset of pain during a car trip. A pulmonary embolism was initially suspected in this setting. Venous Doppler ultrasonography did not reveal any signs of thrombosis. Computed tomography angiography was therefore performed, but also failed to demonstrate any signs of thrombosis, yet it did reveal a pathological collapse fracture with compression of the anterior and middle columns of T-7 (Fig. 3).

A physical examination revealed signs of spinal syndrome (interscapular back pain scored 6/10 on the VAS), and bilateral nerve root syndrome (moderate T-7 intercostal nerve root pain scored 2/10 on the VAS), with no symptoms or signs below the lesion. A secondary tumor was initially suspected. In view of the absence of neuro-
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Logical deficit and any marked instability of the fracture, it was decided to continue the pregnancy with strict bed rest until a cesarean section scheduled at term decided by the obstetric team (39 weeks of gestation), but the cesarean section was eventually performed at 38 weeks of gestation due to onset of uterine contractions.

Postpartum assessment was performed to identify the primary tumor. Complete-spine MRI revealed multiple vertebral lesions at T-2, T-7, and T-8, with a low-intensity signal on T1-weighted sequences and Gd enhancement. Complete invasion of the T-7 vertebra was observed with anterior epidural extension, narrowing the vertebral canal (Fig. 4). The most likely hypothesis continued to remain a secondary tumor. Chest, abdominal, and pelvic CT, breast ultrasonography, and PET-CT scans were performed, revealing only hepatic angiomias.

Operation and Postoperative Course. After a multidisciplinary case review, we decided to perform posterior interbody fusion (Fig. 5) together with transpedicular corpectomy biopsy of T-7 due to instability of the fracture site, displacement of the vertebral posterior wall, the uncertain diagnosis, and finally the patient’s desire to breastfeed, making treatment by rigid immobilization (thermoformed resin corset) extremely difficult. Because the intraoperative biopsy failed to provide a histological diagnosis, a second CT-guided percutaneous biopsy of T-2 was performed and demonstrated a capillary hemangioma. Twenty-one months after the operation, the patient was independent and asymptomatic, and MRI demonstrated a stable appearance of the angiomatous lesions.

Discussion

Vertebral hemangiomas are benign cavernous or capillary vascular tumors. They have a prevalence of 10% in the general population, but are symptomatic in only 1% of cases. Based on a retrospective review of symptomatic patients, Fox and Onofrio reported that 54% of patients complained of back pain and 45% presented with neurological signs. Several mechanisms of spinal cord compression have been identified. The most common mechanism is related to bone expansion in the vertebral canal. The other mechanisms are related to compression by the vascular tumor, the presence of an epidural hematoma or, more rarely, a pathological fracture or ischemia due to vascular shunting.

Pregnancy is a factor that predisposes to progression of hemangiomas. The first case of symptomatic vertebral hemangioma during pregnancy was reported in the literature in 1948 by Guthkelch. Since that first publication, 27 new cases have been described (Table 1). The diagnosis was usually made during the third trimester (22 of 27 cases). The mean maternal age at the time of diagnosis was 30 years. Hemangiomas were located in the thoracic spine in 89% of cases and in the upper thoracic spine in 63% of cases. All patients presented with a neurological deficit, related to bone expansion or epidural vascular compression. The 2 cases reported in this paper confirm these demographic data, although the second case constitutes the first reported case of spontaneous vertebral fracture during pregnancy. Based on a series of hemangiomas in the general population, Fox and Onofrio reported that pain related to the hemangioma often preceded onset of the neurological deficit, as observed in our first case. Two factors have been proposed in the literature to explain this propensity for decompensation of these be-
malignant lesions during pregnancy. The main predisposing factor is hemodynamic, related to the increased blood volume (by 30%–50% at 32 weeks of gestation) and the increased venous pressure beyond the seventh month of pregnancy. This venous hypertension is related to the increased uterine volume, inducing inferior vena cava compression.\textsuperscript{19,28} These hemodynamic modifications obviously result in expansion and dilation of the hemangioma. This mechanism is supported by case reports of spontaneous improvement of back pain or neurological deficits after delivery.\textsuperscript{6,11,13,22} The second factor described in the literature that predisposes patients to decompensation of hemangiomas during pregnancy is hormonal, but this mechanism is much more controversial.\textsuperscript{23} This hypothesis is related to the angiogenic actions of estrogen and progesterone (present at considerably higher levels during pregnancy), which would participate in growth of the hemangioma. However, this hypothesis was not supported by an immunohistological study that failed to demonstrate estrogen or progesterone receptors on hemangioma endothelial cells.\textsuperscript{23} The diagnosis of vertebral hemangioma should therefore be considered in pregnant women who present with features of spinal cord compression during the last trimester of pregnancy.

Very few articles in the existing literature address acute spinal cord compression during pregnancy. Most cases concern the management of patients with preexisting lesions. Apart from vertebral hemangiomas, no cause of spinal cord compression appears overrepresented during pregnancy.

Magnetic resonance imaging is the first-line complementary investigation in this setting, both to avoid radiation during pregnancy and because of its high sensitivity. However, MRI findings remain nonspecific. Hemangiomas present a high-intensity signal on T1- and T2-weighted sequences, reflecting the presence of adipocytes and blood vessels in the interstitium,\textsuperscript{17} and are Gd enhancing (reflecting tumor hypervascularization). They may also present with a low-intensity signal on T1-weighted sequences and a high-intensity signal on T2-weighted sequences, indicating the active nature of the hemangioma with a risk of deterioration and therefore spinal cord compression.\textsuperscript{28} These MRI findings were reported in 2 previously published cases\textsuperscript{5,8} and in both of the cases presented here. In particular, in the first case, 2 other hemangiomas presented with a high-intensity signal on T1- and T2-weighted sequences, while the hemangioma responsible for spinal cord compression presented with a low-intensity signal on the T1-weighted sequences. In this setting, the differential diagnoses, especially secondary tumors, must be excluded by histopathological analysis. A CT scan performed after delivery provided additional signs guiding the diagnosis and allowing analysis of the quality, and therefore the stability, of vertebral bone. The trabecular patterns of the vertebra presented with a typical hatched, or beehive, appearance.\textsuperscript{16}

After establishing the diagnosis, therapeutic management remains difficult and controversial, as it must take into account the maternal neurological risk, the fetal risk in utero, and the risk of prematurity. The first decision concerns continuation or termination of the pregnancy. Vaginal delivery is usually contraindicated to avoid straining (inducing increased venous hypertension) that could exacerbate spinal cord compression. Fetal extraction by cesarean section is therefore performed, when compatible with fetal gestational age. By decreasing inferior vena cava pressure, this procedure alone can be sufficient to improve or even allow complete recovery of the neurological deficit in rare cases.\textsuperscript{6,13} In our first case, the neurological deficit was first observed at an advanced gestational age (35 weeks of gestation). The risks of complications for the fetus were considered to be acceptable with respect to the maternal risk, and a multidisciplinary reviewed decided on cesarean section. The second case did not present any objective signs of neurological deficit and the vertebral fracture did not present any major
criteria of instability. After multidisciplinary discussion, it was decided to continue the pregnancy in the hospital with strict bed rest and neurological surveillance to allow fetal maturation.

Of the 27 cases reported in the literature (Table 1), 12 were described at gestational ages corresponding to severe prematurity (less than 32 weeks of gestation). Of these 12 cases, only half were treated antepartum. Chi et al. have proposed a treatment algorithm.

Despite the absence of sufficient data or even a consensus, Chi et al. have proposed a treatment algorithm.

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### TABLE 1: Cases of symptomatic vertebral hemangiomas during pregnancy reported in the literature

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Maternal Age (yrs)</th>
<th>Wks of amenorrhea</th>
<th>Level</th>
<th>Clinical Features</th>
<th>AP/PP</th>
<th>Treatment</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Guthkelch, 1948</td>
<td>34</td>
<td>35</td>
<td>T-6</td>
<td>paraplegia</td>
<td>AP</td>
<td>laminectomy</td>
<td>maternal death</td>
</tr>
<tr>
<td>Acquaviva &amp; Thevenot, 1957</td>
<td>40</td>
<td>34</td>
<td>T-3</td>
<td>known</td>
<td>PP</td>
<td>laminectomy</td>
<td>complete</td>
</tr>
<tr>
<td>Askenas &amp; Behmaram, 1957</td>
<td>20</td>
<td>39</td>
<td>T-11</td>
<td>paraplegia</td>
<td>PP</td>
<td>laminectomy, radiotherapy</td>
<td>complete</td>
</tr>
<tr>
<td>Fields &amp; Jones, 1957</td>
<td>30</td>
<td>28</td>
<td>T-6</td>
<td>paraplegia</td>
<td>PP</td>
<td>no treatment</td>
<td>complete</td>
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<tr>
<td>Newman, 1958</td>
<td>34</td>
<td>39</td>
<td>T-4-5</td>
<td>paraplegia</td>
<td>AP</td>
<td>laminectomy</td>
<td>maternal death</td>
</tr>
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<td>Nelson, 1964</td>
<td>24</td>
<td>39</td>
<td>T-2</td>
<td>paraparesis</td>
<td>PP</td>
<td>laminectomy</td>
<td>complete</td>
</tr>
<tr>
<td>Esparza et al., 1978</td>
<td>22</td>
<td>28</td>
<td>T5-6</td>
<td>paraparesis</td>
<td>PP</td>
<td>laminectomy, embolization, radiotherapy</td>
<td>complete</td>
</tr>
<tr>
<td>Bouchez et al., 1984</td>
<td>24</td>
<td>31</td>
<td>T-2</td>
<td>paraplegia</td>
<td>PP</td>
<td>embolization</td>
<td>complete</td>
</tr>
<tr>
<td>Faria et al., 1985</td>
<td>40</td>
<td>34</td>
<td>T-3</td>
<td>paraplegia</td>
<td>PP</td>
<td>laminectomy, radiotherapy</td>
<td>complete</td>
</tr>
<tr>
<td>Lavi et al., 1986</td>
<td>25</td>
<td>31</td>
<td>T-4</td>
<td>paraplegia</td>
<td>AP</td>
<td>laminectomy</td>
<td>complete</td>
</tr>
<tr>
<td>Liu &amp; Yang, 1988</td>
<td>25</td>
<td>26</td>
<td>T-4</td>
<td>paraparesis</td>
<td>AP</td>
<td>laminectomy</td>
<td>complete</td>
</tr>
<tr>
<td>Schwartz et al., 1989</td>
<td>30</td>
<td>31</td>
<td>T-5</td>
<td>paraplegia</td>
<td>AP</td>
<td>laminectomy; PP 2nd laminectomy</td>
<td>complete</td>
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<tr>
<td>Redekop &amp; Del Maestro, 1992</td>
<td>20</td>
<td>34</td>
<td>T-12</td>
<td>paraparesis</td>
<td>PP</td>
<td>laminectomy, radiotherapy</td>
<td>complete</td>
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<tr>
<td>Tekkök et al., 1993</td>
<td>25</td>
<td>39</td>
<td>T-5</td>
<td>paraplegia</td>
<td>PP</td>
<td>laminectomy</td>
<td>complete</td>
</tr>
<tr>
<td>Abi-Fadel et al., 1997</td>
<td>28</td>
<td>34</td>
<td>T-9</td>
<td>paraplegia</td>
<td>PP</td>
<td>laminectomy, radiotherapy</td>
<td>complete</td>
</tr>
<tr>
<td>Castel et al., 1999</td>
<td>27</td>
<td>31</td>
<td>T-8</td>
<td>paraplegia</td>
<td>PP</td>
<td>laminectomy</td>
<td>complete</td>
</tr>
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<td>Schwartz et al., 2000</td>
<td>29</td>
<td>41</td>
<td>T-11</td>
<td>paraplegia</td>
<td>PP</td>
<td>corpectomy &amp; fusion, radiotherapy</td>
<td>complete</td>
</tr>
<tr>
<td>Chi et al., 2005</td>
<td>26</td>
<td>29</td>
<td>C-7</td>
<td>paraplegia</td>
<td>AP</td>
<td>corpectomy &amp; fusion</td>
<td>complete</td>
</tr>
<tr>
<td>Inamasu et al., 2006</td>
<td>20</td>
<td>34</td>
<td>L-2</td>
<td>cauda equina syndrome</td>
<td>PP</td>
<td>laminectomy, vertebroplasty, posterior fusion</td>
<td>complete</td>
</tr>
<tr>
<td>Yuksel et al., 2007</td>
<td>21</td>
<td>31</td>
<td>T-9</td>
<td>paraparesis</td>
<td>PP</td>
<td>laminectomy</td>
<td>complete</td>
</tr>
<tr>
<td>Vijay et al., 2008</td>
<td>22</td>
<td>29</td>
<td>T-11</td>
<td>paraplegia</td>
<td>AP</td>
<td>laminectomy, posterior fusion; PP embolization, corpectomy, &amp; fusion</td>
<td>complete</td>
</tr>
<tr>
<td>Kiroglu et al., 2009</td>
<td>22</td>
<td>39</td>
<td>T-4</td>
<td>paraplegia</td>
<td>PP</td>
<td>embolization, vertebroplasty, posterior fusion</td>
<td>complete</td>
</tr>
<tr>
<td>Shinozaki et al., 2010</td>
<td>27</td>
<td>31</td>
<td>T-2</td>
<td>paraplegia</td>
<td>PP</td>
<td>laminectomy (partial recovery), second-line embolization, posterior fusion, corpectomy &amp; fusion</td>
<td>complete</td>
</tr>
<tr>
<td>Blecher et al., 2010</td>
<td>35</td>
<td>39</td>
<td>L-4</td>
<td>L-4 sciatic pain w/ deficit</td>
<td>PP</td>
<td>embolization, laminectomy, vertebroplasty</td>
<td>complete</td>
</tr>
<tr>
<td>Jankowski et al., 2012</td>
<td>32</td>
<td>35</td>
<td>T-6</td>
<td>paraparesis</td>
<td>PP</td>
<td>corpectomy &amp; fusion</td>
<td>partial</td>
</tr>
</tbody>
</table>

* AP = antepartum; PP = postpartum.
gestation, they recommend first-line fetal extraction by cesarean section.

Limited treatment options are available antepartum due to the ongoing pregnancy. The proposed treatments must therefore be associated with a low risk of termination of the pregnancy. Optimal treatment such as a corpectomy, allowing decompression and treatment of the hemangioma, cannot therefore be envisaged in the thoracic or lumbar spine. Only the cervical spine is accessible to such treatment, as performed by Chi et al., resulting in complete maternal neurological recovery and a normal-term pregnancy with no fetal complications. For the other cases involving the thoracic and lumbar spine, posterior decompression by laminectomy was always performed as a first-line procedure (Table 1). With the exception of 2 cases of maternal and fetal death,20,22 (before 1960), this procedure always allowed neurological recovery.5,19-21,24 However, it does not preclude a possible relapse, especially as the pregnancy-related factors of deterioration of the hemangioma are still present. For example, after antepartum laminectomy and partial neurological recovery in a patient, Schwartz et al.24 described a new episode of neurological deterioration during pregnancy, after which a cesarean section was performed together with another posterior decompression procedure at the same spinal level, allowing complete neurological recovery. Surveillance or treatment of the hemangioma is therefore essential, especially as relapse can also occur after delivery.28 In this setting, Vijay et al.28 treated a hemangioma by embolization followed by corpectomy.

The postpartum surgical management performed by most authors is laminectomy (12 of 19 cases),1,3-5,7,9,10,14,22,26,27,29 without a complementary procedure in half of the cases. Neurological recovery was observed in all but 1 case in this group;26 recovery was only partial after laminectomy and the patient was subsequently treated by embolization and corpectomy. This patient presented with severe spinal cord compression with anterolateral predominance, which was poorly accessible to posterior decompression. In our first case, spinal cord compression was predominantly posterior, constituting a good indication for laminectomy, which achieved good neurological recovery. This patient experienced persistent back pain related to the hemangioma. After multidisciplinary discussion, we decided to perform vertebroplasty to treat the hemangioma and to stabilize the vertebral body. Other authors have also performed first-line complementary procedures in combination with laminectomy,1,4,5,9,10,14,16,21 comprising radiotherapy, vertebroplasty, or embolization with good results (Table 1). It is surprising that in this type of surgery associated with a high bleeding risk, embolization was performed prior to laminectomy in only 1 case,2 although this procedure has been shown to reduce intraoperative bleeding.2 No surgical decompression procedure was performed in 2 published cases, who were treated by embolization alone. In the first case,4 complete neurological recovery was obtained without relapse. In the second case,16 symptoms recurred 2 years after the initial episode, despite initial complete recovery. This relapse was treated by vertebroplasty and posterior interbody fusion. In the second case reported here (spontaneous vertebral fracture), only posterior interbody fusion was performed in the absence of neurological deficit. Vertebroplasty was not performed in this case due to the initial risk of cement leakage into the vertebral canal due to effraction of the posterior wall. At subsequent follow-up, the patient refused a second operation and did not want to stop breastfeeding. She also did not desire another pregnancy. This patient was therefore submitted to close clinical and radiological surveillance.

Maximum surgical treatment by corpectomy and fusion allows 1-stage spinal cord decompression and treatment of the hemangioma. Only 4 authors have reported this type of treatment, which allowed complete recovery in 2 cases8,24 and partial recovery in another 2 cases,5,21 in which no poor prognostic factors were identified.

In both the antepartum and postpartum settings, laminectomy therefore appears to be a simple and rapid procedure allowing effective spinal cord decompression. However, this procedure may not be sufficient in patients with predominantly anterior spinal cord compression and it cannot prevent antepartum or postpartum relapse. A complementary procedure (such as vertebroplasty, embolization, or external beam radiotherapy) may therefore be necessary to treat the hemangioma and prevent recurrence, especially when the woman desires a subsequent pregnancy. Finally, a more radical procedure by corpectomy allows 1-stage spinal cord decompression and treatment of the hemangioma, but can only be performed postpartum, except in the rare cases occurring in the cervical spine, and is associated with a risk of significant intraoperative bleeding unless it is preceded by preoperative embolization.2

In this paper we report the first case of spontaneous vertebral fracture caused by vertebral hemangioma during pregnancy, not associated with any neurological deficit and treated postpartum by spinal fusion, and a second case of spinal cord compression treated by postpartum laminectomy and complementary vertebroplasty. Although rare, symptomatic vertebral hemangioma during pregnancy is responsible for spinal cord compression, predominantly in the thoracic spine and during the last trimester of pregnancy, and constitutes a diagnostic and therapeutic emergency. The decision to continue or terminate pregnancy must be based on multidisciplinary case review as a function of fetal gestational age and maternal neurological features. Surgical decompression, either alone or in combination with complementary procedures, ensures good maternal neurological recovery, resulting in a good prognosis.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Conception and design: Buffenoir, Bord. Acquisition of data: Moles, Perret. Analysis and interpretation of data: Buffenoir, Hamel. Drafting the article: Buffenoir, Moles, Bord, Robert. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Buffenoir.
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