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Uterine rupture as a cause of postpartum peritonitis in the horse

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Summary

Five recently foaled mares were presented to the Equine Internal Medicine Clinic with signs of peritonitis that, it turned out, had been caused by a uterine tear. In three of the cases, the uterine tear was diagnosed ante mortem, in two cases at post mortem examination. Establishing this diagnosis by per rectum or per vaginam examination proved to be difficult, especially if the tear was small and located near the tip of the previously gravid uterine horn. Because ultrasonographic examination appears not always to be rewarding either, the diagnosis of a uterine tear can be missed in a routine examination. In two mares presented to our clinic, the presence of a uterine tear was established by manual examination in an early stage of the work-up. One of these mares was not treated and in the other mare the tear was sutured transvaginally, but without a satisfactory outcome. In one mare suspected of having a uterine tear, transcervical videohysteroscopy was performed. This rather simple, minimally invasive procedure proved to be a helpful diagnostic tool. Surgical correction of the tear in this case was achieved successfully via a flank laparotomy in a standing position. Although a surgical approach often offers better results eventually, conservative management of uterine tears has been described to be successful in case of small tears as well. The prognosis for a mare with a uterine tear depends upon the time that elapses between origination of the tear and treatment.

Keywords: horse, periparturient complication, foaling injury, postpartum toxemia, transcervical videohysteroscopy

Gebärmutterwandruptur als Ursache für postpartale Peritonitis beim Pferd

Fünf kurz zuvor abgefohlte Stuten wurden mit klinischen Symptomen einer Peritonitis in die Klinik für Pferdemedizin eingewiesen. Ursache für die Peritonitis war in allen 5 Fällen ein Einriss der Gebärmutter. In 3 Fällen wurde die Ruptur ante mortem gestellt, in 2 Fällen geschah dies post mortal. Die Diagnosestellung durch vaginale oder rektale Untersuchung erwies sich als schwierig, besonders in den Fällen wenn der Riss klein war und/oder sich im vormalig graviden, kranialen Gebärmutterhorn befand. Da auch die transrektale Ultraschalluntersuchung wenig zur Diagnosefindung beitragen konnte war der Gebärmuttereinriss bei einer Routineuntersuchung leicht zu übersehen. Bei 2 an der Klinik vorgestellten Stuten wurde der Riss in einer frühen Phase der Untersuchung während einer rektalen Untersuchung festgestellt. Bei einer dieser Stuten wurde darauf verzichtet eine Behandlung vorzunehmen, bei der zweiten Stute wurde der Riss per vaginam übernäht, allerdings mit einem unbefriedigenden Resultat. Bei einer weiteren Stute, bei der eine Gebärmutterruptur vermutet wurde, konnte die Diagnose per transzervikaler Hysteroskopie gestellt werden. Diese relativ einfache, minimal-invasive Untersuchungmethode bewies sich in der Diagnostik als sehr hilfreich. In diesem Falle wurde die Läsion der Gebärmutterwand über eine Flankenlaparotomie am stehenden Pferd korrigiert. Obwohl die chirurgische Intervention oftmals die besseren Resultate liefert, sind auch erfolgreiche, konservative Behandlungen, vor allem bei kleinen Einrissen in der Literatur beschrieben. Die Prognose für eine derart verletzte Stute hängt vom Zeitraum zwischen Ruptur und Beginn einer Behandlung ab.

Schlüsselwörter: Pferd, postpartale Komplikationen, Geburtsverletzungen, Geburtstoxämie, transzervikale Videohysteroskopie

Introduction

Uterine rupture in the mare can occur during parturition as a result of prolonged or excessive manipulation during dystocia, fetal malpositioning or fetotomy, but it has also been reported after apparently normal parturition or after postpartum uterine lavage (*Roberts 1971, Patel and Lofstedt 1986, Vivrette 1997*). Rarely, prepartum uterine rupture has been reported to occur in cases of uterine torsion (*Wheat and Meagher 1972*) and hydramnios (*Honnas et al. 1988*). Large uterine tears often result in visceral herniation or in rapid onset of hemorrhagic shock leading to death. Smaller tears however, produce much less dramatic clinical signs and may not be diagnosed immediately, especially if they are located in the most cranial part of the uterine horn. After a short period of time they may cause peritonitis of variable severity with clinical symptoms including depression, ileus, inappetence, fever, colic and vaginal haemorrhage (*Patel and Lofstedt 1986, Perkins and Frazer 1994*). The purpose of this retrospective study was to describe in greater detail the clinical features, laboratory findings, treatment and outcome in five mares with a small uterine tear admitted to the Equine Internal Medicine of the University of Utrecht. All five mares were presented with signs of peritonitis that developed after foaling, but only one was referred with the diagnosis of a uterine tear.

Case reports

Horse 1

A 16-year-old Thoroughbred mare was referred because of anorexia and fever during the previous two days. During foaling, four days prior to admission, veterinary assistance had been required to correct a breech presentation and, subsequently, the placenta had been retained and eventually removed by manual traction. The veterinarian treated the mare by flushing its uterus and by administering NSAID's and antibiotics systemically, but because the mare did not improve, it was referred to the Equine Internal Medicine Clinic. On arrival, the mare was depressed and uncomfortable, it had a heart rate of 58 beats/min, a rectal temperature of 37.5 ° C and slightly yellowed mucous membranes. Bowel sounds were lower than normal,

and the mare had a reddish brown, non-odorous vaginal discharge. Blood hematology and biochemistry revealed anemia (hematocrit, 0.24 litres/litre; reference range 0.36 to 0.42 litres/litre) and a slight leucopenia (5.4 x 10^9 white blood cells/litre; reference range 7 to 10 x 10^9 /litre). Per rectum examination demonstrated that the uterus was quite large and firm and that its palpation elicited pain. Furthermore, per vaginam examination of the uterus revealed a tear near the tip of the left uterine horn. The owner elected not to proceed with a potentially expensive course of treatment and, therefore, the mare was euthanized. During a subsequent post-mortem examination, a 10 cm tear near the oviductal junction was identified as the cause of a accompanying acute peritonitis.

Horse 2

A veterinarian was called out to examine an eleven-year old Dutch Warmblood mare because of signs of dystocia. He diagnosed a dead foal presenting with a bilateral shoulder flexion and, after correction of the postural abnormality, the foal was delivered. Two hours later the placenta was removed manually and at this time a uterine tear was detected, and the mare was referred to the Equine Obstetrics Clinic. On arrival the mare was depressed and uncomfortable. It had a heart rate of 60 beats/min, reddened mucous membranes and a raised hematocrit of 0.52 litres/litre. A per vaginam examination revealed a tear, approximately 15 cm in length located dorsolaterally in the uterus and just cranial to the cervix. The tear was sutured transvaginally under epidural anesthesia and the mare was treated with ceftiofur-sodium[a] (2 mg/kg bodyweight IV, q 12h), flunixin meglumine[b] (1.1 mg/kg bodyweight IV, q 12h) and oxytocin[c] (10 iu IM, q 3h). Nine litres of 0.9 % NaCl solution was infused over four hours. Despite treatment, the mare deteriorated over the next two days, and it was transferred to the Equine Internal Medicine Clinic. By this time the mare had a rectal temperature of 39.4 °C, and after a per rectum examination, a provisional diagnosis of peritonitis was made. This diagnosis was confirmed by abdominocentesis during which a copious quantity of serosanguineous fluid was recovered. Cytological examination of this fluid demonstrated large quantities of degenerate neutrophils and bacteria. In consultation with the owner, it was decided to euthanize the mare. A post-mortem examination revealed that the peritonitis had caused degeneration of the parenchymatous organs. The uterine tear was indeed closed along its entire length, but when light pressure was applied to this organ, cloudy fluid present in the uterine lumen leaked out of the wound.

Horse 3

A 5-year-old Dutch Warmblood mare that required minimal assistance with foaling, and passed the placenta completely within one hour, was presented to the referring veterinarian 24 hours after foaling because it seemed off-colour. The veterinarian flushed the uterus, which yielded a lot of hemorrhagic, malodorous fluid. A further 24 hours later, the mare was referred to the Equine Internal Medicine Clinic. On arrival, the mare was depressed, had a rectal temperature of 39.4° C, a heart rate of 76 beats per minute and reddened mucous membranes. No bowel sounds were detected and the mare had a tense abdomen. Hematological examination revealed a hematocrit of 0.58 litres/litre, a WBC count of 8.5 x 10^9/litre and a plasma total protein concentration of 56 g/litre (reference range 58 to 77 g/litre). Per vaginam examination of the uterus revealed the presence of copious amounts of foul-smelling fluid, but no laceration was detected. Per rectum examination suggested a peritonitis, which was confirmed by the copious quantities of hemorrhagic fluid and fibrinous material collected during abdominocentesis. Laboratory analysis of this fluid revealed large quantities of erythrocytes and WBC's (16,4 x 10^9/litre), among which 95% of the neutrophils were degenerate. Bacteriological culture demonstrated the presence of both Escherichia coli and a Streptococcus group D. It was thought that the septic peritonitis may have been secondary to ischemic necrosis of the intestinal wall resulting from bruising during parturition. The mare was treated with 30 mg/kg bodyweight sulfadoxine/trimethoprim[d] (IV, q 12h) and 1.1 mg/kg bodyweight flunixin meglumine[b] (IV, q 12h) and in addition 20.000 iu heparin[e] was injected (SC, q 8h). To prevent circulatory collaps, 24 litres of 0.9% NaCl solution were infused as a continuous drip. Two days later, the antibiotic treatment was changed to 2 mg/kg bodyweight ceftiofur-sodium[a] (IV, q 12h) because antibiotic sensitivity testing of the cultured organisms suggested only limited susceptibility to potentiated sulphonamides. Thereafter, the mare seemed to improve, it started to eat and peristaltic intestinal movements restored. However, a further 24 hours later, the mare once again deteriorated and was found to be suffering from severe dehydration and gastric reflux. Considering the poor prognosis, the owner decided the mare should be euthanized. During the subsequent post mortem examination, it became clear that the mare had developed an acute, fulminant peritonitis as a result of a 3 cm rupture in the tip of the right uterine horn.

Horse 4

A 16-year-old Warmblood mare delivered without assistance a foal with an angular deformity of the left hind limb. The day after delivery the mare became febrile and colicky and was treated, by the referring veterinarian, with antibiotics and NSAID's. In addition remnants of the placenta were recovered during a uterine lavage. After four days of treatment, however, the mare's condition had not improved, and it was referred to the Equine Internal Medicine Clinic. At the time of admission, the mare was uncomfortable and sweaty, it had a heart rate of 80 beats/min, a rectal temperature of 39.2 °C, and exhibited bruxism. Furthermore, it had a tense and distended abdomen in which few borborygmia could be heard, and a stomach overload of 21 litres. A hematological examination indicated dehydration (hematocrit was 0.58 litres/litre) and a leucocytosis (19.7 x 10^9 WBC's/litre) while serum biochemical analysis revealed uremia (plasma urea level 28 mmol/l; reference range 2–8 mmol/l) and azotemia (creatinine level 368 μmol/l; reference range 106–168 μmol/l). During a per rectum examination, some loops of gas-distended small intestine were detected, the uterus appeared to be involuting normally. Examination of the uterus per vaginam did not reveal any obvious perforation, the mucoid vaginal discharge observed, was considered normal for a mare that had recently foaled. Abdominocentesis resulted in the collection of copious quantities of turbid redbrown fluid that contained 55.2 x 10^9 WBC's/litre, of which 94% were neutrophils. Bacteriological culture of this peritoneal fluid revealed the presence of a mixture of bacteria resembling that found in feces flora. Despite the poor prognosis,

the owner wanted the treatment to be continued and the mare was, therefore, started on a course of ampicillin-sodium[f] (10 mg/kg bodyweight) and gentamicin[g] (3.3 mg/kg bodyweight) intravenously every 12 hours. In addition, anti-endotoxic doses of flunixin meglumine[b] (0.4 mg/kg bodyweight IV, q 8h) were given. Intravenous fluid support was provided for by a continuous infusion of 0.9% NaCl solution. Nevertheless, the mare's condition continued to deteriorate with urea levels reaching 42 mmol/l, peristaltic movement of the intestines failing to improve and the stomach requiring decompression at frequent intervals. Furthermore, despite the infusion of 36 litres of NaCl in 24 hours, the mare's hydration status could not be maintained and when the mare started to show signs of circulatory shock, it was decided to put it down. The post-mortem examination revealed an 8 cm tear in the tip of the right uterine horn which had caused an acute peritonitis leading to degeneration of the kidneys and liver.

Horse 5

A 3-year-old Dutch Warmblood mare that foaled over night at pasture was, when found the following morning, recumbent and depressed. The placenta had passed and the foal was apparently healthy. The attending veterinarian lavaged the uterus, and treated the mare with NSAID's and antibiotics because of fever. After four days of treatment, temperature still was elevated and the mare spent unusually long periods of time lying down. It was, therefore, referred to the Equine Internal Medicine Clinic. On arrival, the mare was very dull, it had a temperature of 38.4 °C, pale yellow mucous membranes, poor skin turgor and a heart rate of 60 beats per minute. The mare shivered frequently and had a tense abdomen. Hematological examination revealed leucopenia (2.9 x 10^9 WBC's /litre) and anemia (hematocrit 0.25 litres/litre). Serum biochemical analysis demonstrated a high total protein concentration (total serum protein concentration 90 g/litre) due, at least in part, to dehydration. A per rectum examination revealed a rubbery structure cranial to the tip of the right uterine horn and palpation of this structure caused the mare obvious pain. The broad ligaments appeared to be unaffected and no further abnormalities were noted. Abdominocentesis yielded large quantities of a dark serosanguinous fluid containing 12.7 x 10^9 WBC's / litre and 0.12 litres/litre of RBC's. Bacteriological culture of this fluid demonstrated the presence of Escherichia coli bacteria. Although a uterine tear was suspected, none was found during a per vaginam examination of the uterine wall. The uterus was then lavaged with saline to remove the thick brownish fluid it contained. Antimicrobial therapy consisted of ampicillin-sodium[f] (10 mg/kg bodyweight IV, q12h) and gentamicin[g] (3.3 mg/kg bodyweight IV, q 12h). In addition, the mare was treated with flunixin meglumine[b] (0.4 mg/kg IV, q 8h) and with oxytocin[c] (four doses of 10 iu IM at 3 hour intervals) and nine litres of 0.9% NaCl solution was infused intravenously over 3 hours. During the next few days, the mare's condition remained stable, but because it was still febrile and because the bacteriological sensitivity test suggested that the peritoneal infection might be more susceptible to ceftiofur, the antimicrobial therapy was changed to ceftiofur-sodium[a] (2 mg/kg bodyweight IM, q12h). Five days after admission, the mare's hematocrit had decreased to 0.17 litres/litre and it was, therefore, given a blood transfusion. Abdominocentesis was repeated because of the fear of intra-abdominal bleeding and the peritoneal fluid recovered had a chocolate-brown colour, like old blood. Since we were still suspicious of a uterine tear, we decided to perform a hysteroscopy using a 12.8 mm flexible videoendoscope (ETM, München). For this, the mare was restrained in stocks, its tail was bandaged and its perineum cleaned thoroughly before the endoscope was passed manually through the cervical canal. Distension of the uterine lumen with air enabled a thorough inspection of the uterine wall and thereby the identification of a small tear at the tip of the right uterine horn. The owner consented to surgical correction of the tear, which was performed via a standing right flank approach under heavy sedation with 50 mcg/kg bodyweight detomidine[h] and 4 µg/kg bodyweight buprenorphine[i]. For its added analgesic effects, local anesthesia with lidocain[j] was used. During surgery, no adhesions were found within the peritoneal cavity, but there was an enormous amount of serosanguineus fluid present. As much of this fluid as possible was removed by suction and the abdomen was lavaged with 18 L of lactated Ringer's solution. On inspection of the uterus, a full-thickness tear of about 7 cm in length was found near the tip of the right uterine horn, which was closed using an absorbable suture material (2-metric polygalactin[k]). Bacteriological culture of the abdominal fluid yielded the same E. coli as previously and therefore post-operative management was continued with ceftiofur-sodium. The mare was also still treated with flunixin meglumine[b] (0.4 mg/kg IV, q 8h) and 10 litres of 0.9% NaCl infusion, added to the treatment was calciumcarbasalate[l] (6 mg/kg PO, q 24h) to prevent thrombosis. Subsequently, the mare recovered gradually, becoming more attentive, starting to eat and its hematocrit eventually increased to 0.30 litres/litre. The mare was discharged from the clinic after three weeks. Ten months later the mare was doing well and had managed to rear its foal normally. The owner had no further intention of breeding the mare but plans on bringing it into jumping competition.

Discussion

Uterine rupture occurs infrequently in mares and is, therefore, often neglected as a possible cause of problems during the postpartum period. Nevertheless, uterine tears have been described in horses (*Snoeck 1962, Ponting and David 1972, Brooks et al 1985, Cran 1985, Fischer and Phillips 1986, Perkins et al. 1992, Dascanio et al. 1993, Hassel and Ragle 1994*) as they have in other species, such as cattle (*Blenkhorn and Adams 1974*) and man (*Van Wassenhove and Vanderick 1970, Plauche et al. 1984*).

Classically, rupture of the uterus in horses is associated with problems during foaling. In case 1 and 2 of the present report, there were indeed complications associated with the delivery and both these mares required considerable assistance during parturition because of fetal postural defects. However, uterine rupture has also been reported after apparently normal parturition (*Cran 1985, Patel and Lofstedt 1986, Waldow 1996*). In the current report, cases 4 and 5 foaled spontaneously and unobserved and horse 3 needed only minimal assistance. Intrauterine therapy was not initiated in any of these cases until the horse started to show signs of illness and it was, therefore, almost certainly not the causal factor. In four of the five cases in this report, the uterine tear was found at the extremity of the previously gravid uterine horn, as it was in several of the cases reported in literature (*Brooks et al 1985, Dascanio et al. 1993, Hassel and Ragle 1994*). It seems hard to imagine that a tear

near the tip of the uterine horn could be iatrogenic even if delivery was assisted. In this respect *Dascanio et al. 1993* postulated that continued pressure of a foal's stifle against the maternal pelvis or the forces exerted by a foal's foot against a contracting uterine wall probably could be sufficient to tear the uterus. Finally, it might be possible that severe angular limb deformities of the foal, as in case 4, may in some circumstances predispose to uterine damage as a result of the limited mobility of the affected limb.

Complications of a full-thickness uterine tear can be catastrophic in the case of a large tear, and include herniation of the viscera, haemorrhage, shock and death. Smaller tears on the other hand, especially those located cranially in the uterus, may at first go unnoticed. However, as in the cases described in this report, smaller tears will, after a short period of time often cause a peritonitis accompanied by clinical symptoms such as depression, ileus, inappetence, fever, colic and vaginal haemorrhage (*Pascoe and Pascoe 1988, Perkins and Frazer 1994*). In such a case (repeated) abdominocentesis will result in the recovery of large quantities of (sero)sanguineous fluid with an elevated total protein concentration and white blood cell count, and often containing bacteria (*Brooks et al 1985*). Abdominal taps like this in a postpartum mare are likely to be caused by a systemic problem or parturient related intra abdominal lesion, as *Van Hoogmoed et al. (1996) and Frazer et al. (1997)* have found that peritoneal fluid abnormalities in postpartum mares cannot be attributed to the foaling process itself. Partial thickness tears due to severe bruising of the uterine wall can also result in peritoneal contamination and diapedesis of erythrocytes and bring about a clinical picture similar to that described in case of a small tear (*Brooks et al 1985, Hassel and Ragle 1994*).

The differential diagnose of a uterine tear should include a (non-perforated) gastrointestinal tract lesion, haemorrhage into the broad ligament, retained fetal membranes and (endo)metritis (*Dascanio et al. 1993, Brooks et al 1985*).

Frustratingly, it can be difficult to confirm the diagnosis of a uterine tear by per rectum or per vaginam manual or ultrasonographic examination (*Brooks et al 1985, Dascanio et al. 1993, Hassel and Ragle 1994*). This is particularly true when the uterine tear is small and located near the tip of the previously gravid uterine horn, as it was in cases 1, 3, 4 and 5 in this report, because in recently foaled mares the cranial extremities of the uterine horns are often not readily accessible by either route (*Brooks et al 1985, Hassel and Ragle 1994*). In case 5 of the current report, a uterine tear was suspected strongly but could not be detected by manual or ultrasonographic examinations. For this reason it was decided to use hysteroscopy in an attempt to confirm or rule out this provisional diagnosis. Endoscopy of the equine uterus is used quite frequently in the diagnosis and treatment of intraluminal endometrial cysts or adhesions and in the investigation of infertility of unknown origin (*Traub-Dargatz and McKinnon 1988, Bracher and Allen 1992*). However, to the authors' knowledge it has not been reported previously for the diagnosis of a uterine tear. Certainly hysteroscopy has many advantages over laparascopy, described as a means for identifying a uterine tear by *Hassel and Ragle (1994)*. For instance, hysteroscopy is relatively non-invasive and therefore less of a risk to the mare involved, and it allows easier access to the whole uterus.

With regard to the treatment of mares with a uterine tear, the most common approach is surgical closure via ventral midline celiotomy under general anesthesia which has the advantage of allowing thorough inspection and lavage of the abdomen, if necessary (*Brooks et al 1985, Pascoe and Pascoe 1988, Dascanio et al. 1993*). Suturing a defect after prolapsing the uterus (*Snoeck 1962, Fischer and Phillips 1986*) or via a transvaginal approach have also been reported to be successful (*Perkins et al. 1992*). However, in case 2 of this report, transvaginal suturing of a tear did not turn out to be effective. Certainly, poor visibility and the difficulty of working at a distance in a confined space are considerable disadvantages of the transvaginal approach. In case 5, it was decided to operate on the mare in the standing position because its clinical condition was considered too poor for general anesthesia. In this case this approach allowed sufficient visibility and accessibility to achieve a satisfying result.

Conservative management of a uterine tear can be attempted in case of a relatively small tear, using systemic antibiotics, oxytocin, NSAID's, intravenous fluids and if necessary a blood transfusion *as Patel and Lofstedt (1986), Brooks et al. (1985)* and also *Hassel and Ragle (1994*) describe. Unfortunately in case 3, 4 and 5 of this report, a conservative treatment did not turn out to be successful. If peritonitis has already been established, lavage of the peritoneal cavity may improve the condition of the mare remarkably (*Patel and Lofstedt 1986, Hassel and Ragle 1994*). However, non-surgical peritoneal lavage can be a hazardous procedure (*Perkins and Frazer 1994*) and is one that we avoid, whenever possible, because of previous bad experiences.

The interval from occurrence of a uterine tear to diagnosis and the initiation of treatment influences considerably the prognosis for that mare (*Perkins and Frazer 1994*). Flushing the uterus before recognition of the presence of a tear, as was performed in four of the mares in this report can considerably worsen the situation by further contaminating the abdominal cavity (*Fischer and Phillips 1986*).

There are several descriptions of mares that have survived (*Patel and Lofstedt 1986, Dascanio et al. 1993*), and even returned to breeding, especially when the tear was diagnosed and treated early on (*Brooks et al 1985, Fischer and Phillips 1986*).

We conclude that, in the case of mares suffering from suspected toxemia or peritonitis soon after foaling the possibility of a uterine tear should be considered, even if the foaling was uncomplicated. If there are difficulties in establishing a diagnosis, complementary techniques such as videohysteroscopy (case 5 of this report) and laparoscopy (*Hassel and Ragle 1994*) can help considerably in both diagnosis and assessment of the severity of any lesions.

Footnotes

[a] Ceftiofur-sodium (Excenel®; Pharmacia & Upjohn Animal Health, Woerden)
[b] Flunixinemeglumine (Finadyne®; Scheringh-Plough B.V., Maarssen)
[c] Oxytocin (Oxytocine-S®; Intervet International B.V., Boxmeer)
[d] Sulfadoxine/trimethoprim (Borgal®; Hoechst Roussel Vet B.V., Brussels, Belgium)
[e] Heparin (Heparine®; LEO Pharmaceuticals Products B.V., Weesp)
[f] Ampicillin-sodium (Ampicilline®; Kombivet B.V., Etten-Leur)
[g] Gentamicin (Gentamycine 5%®; Eurovet, Bladel)
[h] Detomidine (Domosedan®; Pfizer Animal Health B.V., Capelle a/d IJssel)
[i] Buprenorphine (Temgesic®; Schering-Plough B.V., Maarssen).
[j] Lidocain (Lidocaïne 2 % + adrenaline; Eurovet, Bladel)
[k] 2-metric polygalactin 9 10 (Vicryl®; Johnson & Johnson Medical B.V., Brussels, Belgium)

[1] Calciumcarbasalate (Carbasalatum calcicum Gf ®; Genfarma B.V., Maarssen)

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