Synostosis of the first and second ribs in six horses

Tibor Rovel DVM

Virginie Coudry DVM

Jean-Marie Denoix DVM, PhD

Fabrice Audigie DVM, PhD

From the Centre d'imagerie et de recherche sur les troubles locomoteurs équins, Ecole Vétérinaire d'Alfort, Université Paris-Est, 14430 Goustranville, France. Dr. Rovel's present address is the Department of Medical Imaging of Domestic Animals, Faculty of Veterinary Medicine, Ghent University, Salisburylaan 133, 9820 Merelbeke, Belgium.

Address correspondence to Dr. Rovel (rovel.tibor@gmail.com).

CASE DESCRIPTION

Over a 2-year period, 6 horses (4 Selle Français, 1 Hanoverian, and 1 Thoroughbred) were referred for evaluation of forelimb lameness. All horses had radiographic evidence of synostosis of the first and second ribs (SFSR).

CLINICAL FINDINGS

For 1 horse, the SFSR was considered the probable cause of the lameness (grade 3/5), with a shortening of the cranial phase of the stride in the affected limb. For 3 horses, it was considered a possible cause of the lameness (grade 1/5) for the same reason. For 2 horses, SFSR was considered an incidental finding unassociated with any clinical signs. The 4 horses with lameness suspected as attributable to SFSR had a moderate to severe amount of irregularly marginated new bone formation at the site of the SFSR, with a cranial displacement of the first rib, compared with findings for the 2 horses in which the SFSR was considered incidental. A likely congenital abnormality of the first rib was first suspected on nuclear scintigraphy in the 1 horse for which it was performed or on radiography of the caudal cervical portion of the vertebral column (3 horses) or shoulder joint (2 horses).

TREATMENT AND OUTCOME

The horse in which SFSR was considered the probable cause of the lameness was retired to the field and remained chronically lame. Two of the 3 horses in which SFSR was considered a possible cause of lameness received an IV infusion of tiludronate disodium and mesotherapy over the caudal cervical and cranial thoracic regions; both returned to competition but with poor results. One of the 2 horses with subclinical SFSR never developed lameness on the affected side. No follow-up information was available for the other 2 horses.

CLINICAL RELEVANCE

SFSR can be an incidental finding in horses, with or without clinical manifestations. This abnormality should be considered as a differential diagnosis for horses with forelimb lameness and associated shortening of the cranial phase of the stride that fails to improve with diagnostic analgesic techniques. (*J Am Vet Med Assoc* 2018;253:611–616)

A 4-year-old Selle Français gelding (horse 1) was referred because of left forelimb lameness of 8 months' duration observed when the horse was in the field. The horse had never been trained because of the lameness and had no history of trauma. Despite 2 weeks of NSAID treatment, the lameness failed to improve.

Physical examination revealed muscular atrophy of the left shoulder region and proximal portion of the left forelimb. The left forelimb was held statically in retraction, and mild internal rotation of the limb was evident. A lameness examination was performed, revealing grade 3/5 lameness as well as a shortening of the cranial phase of the stride in the left forelimb. This lameness appeared exacerbated after warm-up exercise and when circling to the right. Flexion testing of the distal portion of the left forelimb yielded mildly positive results (ie, a mild increase in the observed lameness following flexion). Performance of distal digital, distal metacarpal, ulnar, and median nerve blocks resulted in no improvement of the lameness. Diagnostic anesthesia of the proximal attachment of the suspensory ligament in situ and intra-articular anesthesia of the scapulohumeral joint also had no effect on the lameness.

Nuclear scintigraphy of the cervical portion of the vertebral column and forelimbs was performed. On the left side, rib abnormalities were first suspected because of moderately, focally increased radiopharmaceutical uptake over the middle third of R1 and R2 and the laterally concave appearance of the left R1 on the cranial view of the pectoral region **(Figure 1)**. No other abnormality was detected by nuclear scintigraphy.

Complete radiographic and ultrasonographic examinations of the cranial ribs were performed, and comparative images of the left and right ribs were obtained. Radiographic examination revealed an SFSR on the left side, which was clearly identified on a craniomedial-caudolateral oblique view of R1 and R2 with

ABBREVIATIONS

R1	First rib
R2	Second rib
SFSR	Synostosis of the first and second ribs

the limb protracted on the side of the examined rib.[1] The R1 and R2 inserted distally on a broad common shaft that was wider than the combined width of the 2 ribs. A cranial deformation with an oblique radiolucent line (caudoproximal-craniodistal orientation) was identified on R1 at the site of SFSR, corresponding to the most prominent part of the rib. This radiolucent line was delineated by regular and smooth margins **(Figure 2)**. Ultrasonographic examination revealed a severe amount of irregular new bone formation on the cranial aspect of R1 at the site of the synostosis. Compared with the other side, cranial displacement of R1 at the site of the SFSR was suspected and the scalene muscles appeared thinner between the site of SFSR and the pectoralis muscles **(Figure 3)**.

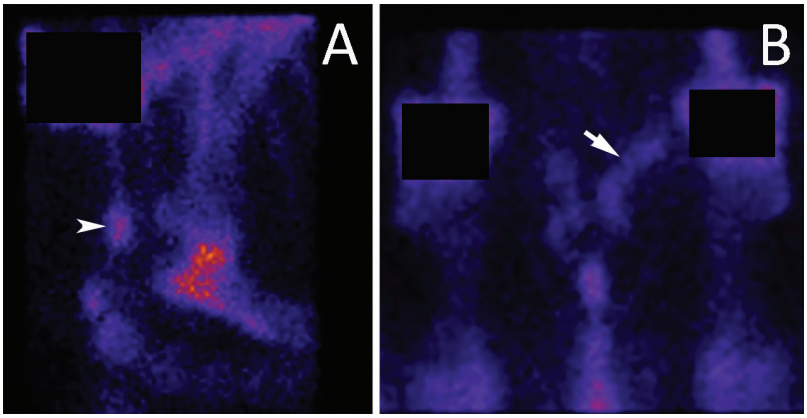

Figure 1—Left lateral non–weight-bearing (A) and cranial (B) scintigraphic views of the cranial thoracic region of a 4-year-old Selle Français gelding (horse 1) referred because of left forelimb lameness of 8 months' duration. A—The left forelimb was retracted caudally. A moderate increase in radiopharmaceutical uptake is visible over the middle third of R1 (arrowhead). B—The left R1 appears misshaped, with a marked lateral concavity (arrow).

Comprehensive radiographic and ultrasonographic assessments of the cervical portion of the vertebral column and proximal portion of the lame limb were also performed, revealing no remarkable findings. Therefore, the SFSR was considered the probable cause of the lameness and the horse was retired to the field and remained chronically lame. Anti-inflammatory drugs were prescribed (phenylbutazone, 4.4 to 8.8 mg/kg [2 to 4 mg/kg], PO, q 24 h), but no information was available regarding the horse's response to this treatment.

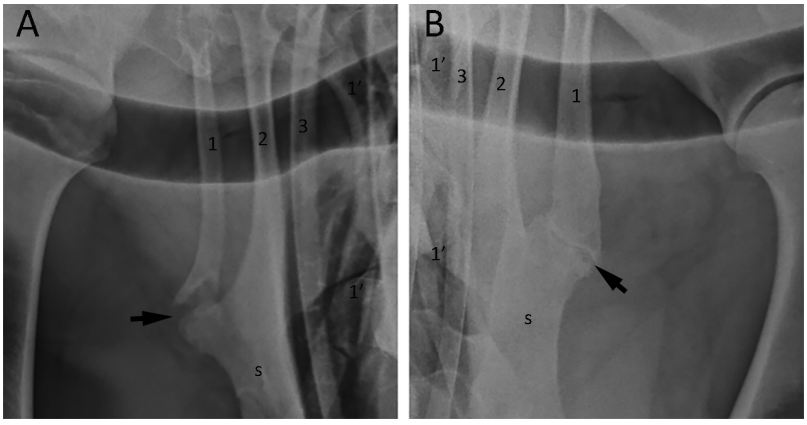

Figure 2—Craniomedial-caudolateral oblique radiographic views of horse 1 (A) and a 6-year-old Thoroughbred gelding referred because of right hind limb lameness of 2 weeks' duration (horse 6; B). Notice how R1 is more prominent and the site of SFSR (arrow) is wider in horse 1 than in horse 6. 1 = R1. 2 = R2. 3 = Third rib. 1′ = Contralateral R1. s = Common shaft.

A 5-year-old Hanoverian gelding (horse 2) used for dressage was referred because of right forelimb lameness of 12 months' duration. The horse had no history of trauma. Physical examination revealed right forefoot atrophy (higher heels than in the left forefoot). Lameness examination revealed a grade 1/5 lameness as well as a briefer cranial (vs caudal) phase of the stride in the right forelimb. Flexion testing of the distal portion of the affected limb yielded mildly positive results. Performance of distal digital and distal metacarpal nerve blocks resulted in no improvement.

Diagnostic nuclear scintigraphy was offered to the owner but refused. Radiography of the caudal cervical portion of the vertebral column was performed, and a laterolateral view revealed an abnormal cranial orientation of the right R1, raising the suspicion of rib abnormalities. Complete radiographic and ultrasonographic examinations of the cranial ribs were performed, and comparative images of the left and right ribs were obtained. Unilateral SFSR was clearly identified on a craniomedial-caudolateral oblique view of R1 and R2 on the right side. Compared with horse 1, R1 and R2 were inserted distally on a thinner piece of bone and a second radiolucent line was visible on the distal portion of R2 just proximal to the site of SFSR. These radiolucent lines were delineated by irregular and ill-defined margins. Ultrasonographic findings for the SFSR were identical to those described for horse 1, but the amount of irregular new bone formation was more moderate in horse 2.

Comprehensive radiographic and ultrasonographic assessment of the cervical portion of the vertebral column and the lame forelimb was performed, revealing no remarkable findings. Therefore, the SFSR was considered a possible cause of the lameness and shortening of the cranial phase of the stride in the affected limb. Because we suspected bone changes and because new bone formation was identified at the site of the SFSR, horse 2 received an IV infusion of tiludronate disodium (1 mg/kg, IV)[2] and mesotherapy[3] over the

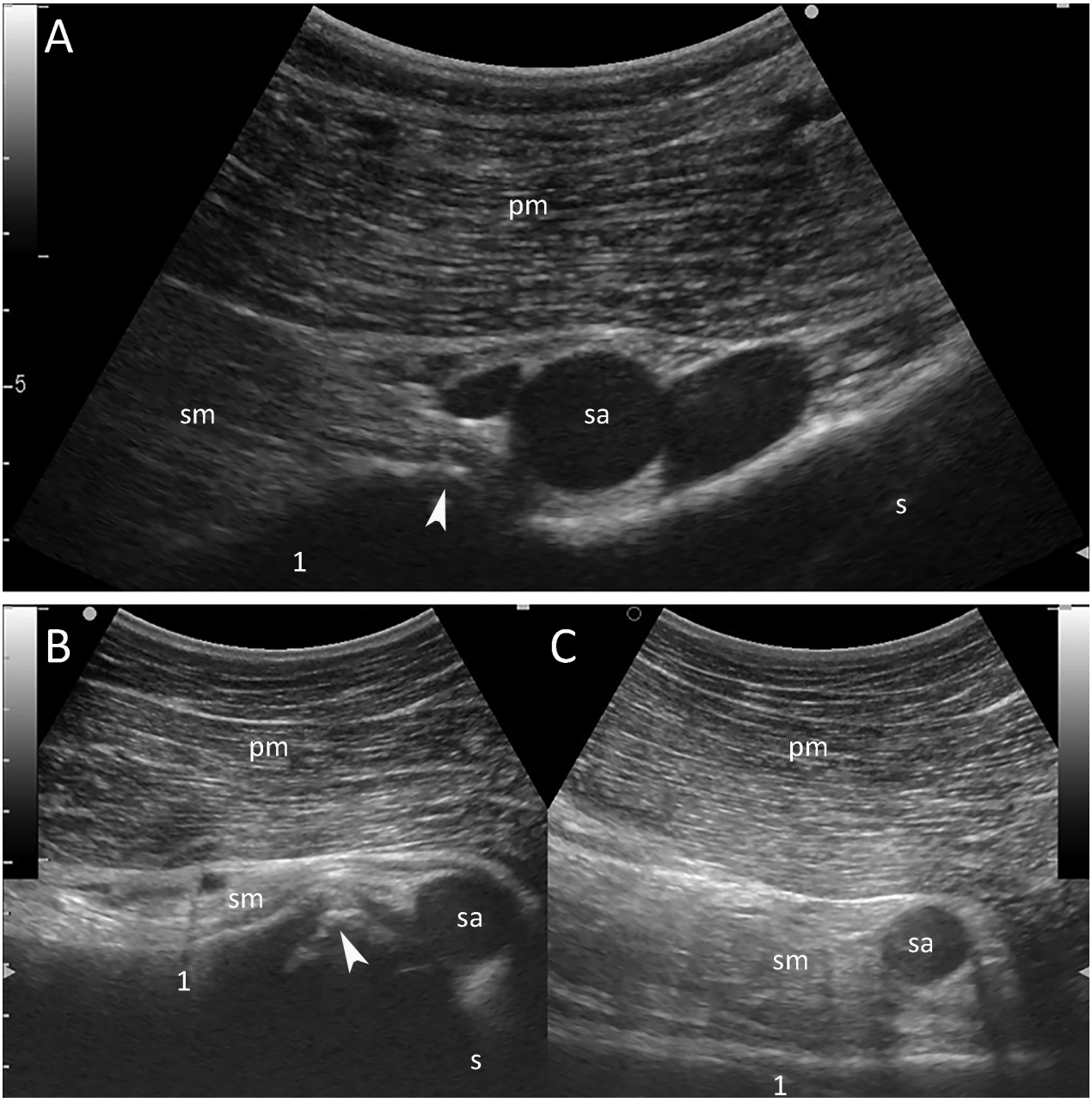

Figure 3—Longitudinal ultrasonographic views of R1 obtained at the site of SFSR (arrowhead) in an 8-year-old Selle Français mare referred because of left forelimb lameness of 3 weeks' duration (horse 5; A) and in horse 1 (B), and reference image of the contralateral R1 at the same level and at the same magnification as for horse 1 (C). The scale used for all images is in centimeters, and dorsal is to the left. The amount of new bone formation at the site of SFSR was considered mild (grade 1) for horse 5 and severe and more irregularly marginated (grade 3) for horse 1. Notice how R1 is more prominent at the site of SFSR than in the contralateral reference image for horse 1. The depth ratio (ie, depth of the SFSR at its most prominent aspect [4.8 cm] divided by the depth of the contralateral R1 [8.2 cm] at the same level) was 0.59. The scalene muscles appear thinner in horse 1 than in horse 5 or the contralateral reference image. 1 = R1. pm = Pectoralis muscles. s = Common shaft. sa = Subclavian artery. sm = Scalene muscles.

caudal cervical and cranial thoracic areas. This horse returned to show jumping competition but with poor results.

A 5-year-old Selle Français mare (horse 3) used for show jumping was referred because of right forelimb lameness of 5 months' duration. The horse had no history of trauma. Physical examination revealed mild atrophy of the right forefoot. Lameness examination revealed a grade 1/5 lameness as well as a briefer cranial (vs caudal) phase of the stride in the right forelimb. Results of flexion testing of the distal portion of the affected limb were negative. No diagnostic anesthesia was performed the day of the examination because the lameness was not consistent enough.

Diagnostic nuclear scintigraphy was offered to the owner but refused. Radiographic and ultrasonographic assessment was performed as for horses 1 and 2. A laterolateral radiographic view of the caudal cervical portion of the vertebral column revealed an abnormal cranial orientation of the right R1, raising the suspicion of rib abnormalities. An SFSR was clearly identified on the right side on a craniomedial-caudolateral oblique radiographic view of R1 and R2. The SFSR was also conspicuous on a mediolateral radiographic view of the right scapulohumeral joint that included the proximal portion of R1 **(Figure 4)**. Ultrasonographic findings for the SFSR were identical to those described for horse 1. Findings of comprehensive radiographic and ultrasonographic assessment of the cervical portion of the vertebral column and the lame limb were unremarkable. Therefore, the SFSR was considered a possible cause of the abnormal findings of lameness examination. The horse received an IV infusion of tiludronate and mesotherapy as described for horse 2. This horse returned to show jumping competition but with poor results.

A 5-year-old Selle Français mare (horse 4) used for show jumping was referred because of right forelimb lameness of 3 weeks' duration. The horse had no history of trauma. Physical examination revealed mild muscle atrophy of the caudal neck region on both sides. Findings of lameness examination were similar to those of horses 2 and 3, and flexion test results were unremarkable. No diagnostic anesthesia was performed the day of the examination because the lameness was not consistent enough, nor was nuclear scintigraphy performed.

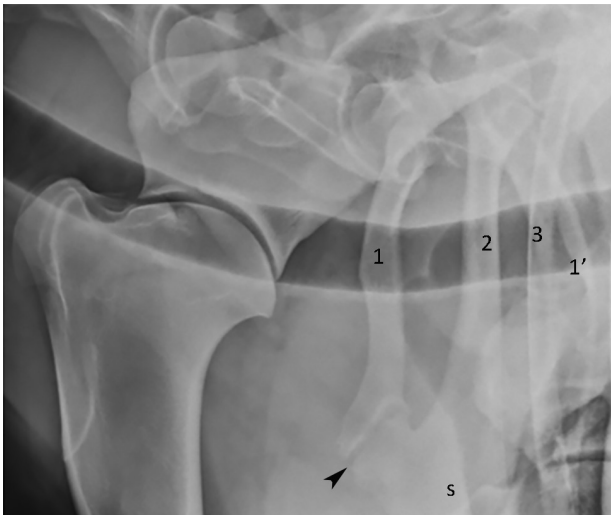

Figure 4—Mediolateral view of the right scapulohumeral joint and cranial thoracic region of a 5-year-old Selle Français mare referred because of right forelimb lameness of 5 months' duration (horse 3). This view was deliberately obtained more caudally and distally than usual to allow better visibility of the SFSR. The right R1 appears misshaped with a cranial convexity at the dorsal aspect and at the site of SFSR (arrowhead). Both R1 and R2 appear to insert on a common broad shaft. ***See*** Figure 2 for remainder of key.

As for other horses, a laterolateral radiographic view of the caudal cervical portion of the vertebral column revealed an abnormal cranial orientation of the right R1, raising the suspicion of rib abnormalities. Further diagnostic imaging was performed as for horses 2 and 3, and a right-sided SFSR was clearly identified. Radiographically, compared with the findings for horse 1, the radiolucent line was narrower and delineated by more irregular and ill-defined margins. Moreover, although the SFSR was unilateral, the R1 contralateral to the SFSR was inserted more proximally than usual and on the common broad shaft formed by the SFSR on the other side. Ultrasonographic findings were similar to those described for horse 1. No abnormalities were identified on comprehensive radiographic and ultrasonographic assessment of the cervical portion of the vertebral column and the lame limb. The SFSR was consequently considered a possible cause of abnormal findings of lameness examination. No follow-up information was available.

An 8-year-old Selle Français mare (horse 5) used for show jumping was referred because of left forelimb lameness of 3 weeks' duration. The horse had no history of trauma. Physical examination revealed mild muscle atrophy of the left point of the shoulder as well as a long toe and underrun heels[4] on the left forefoot. A grade 2/5 left forelimb lameness was identified, and the horse had a positive response to a distal digital nerve block.

Mediolateral radiographic views of the left and right shoulder joints and proximal portion of R1 revealed that the left R1 had an abnormal cranial orientation. A left-sided SFSR was clearly identified on a craniomedial-caudolateral oblique radiographic view of R1 and R2. Compared with the findings for horse 1, the identified radiolucent line was narrower and delineated by more irregular and ill-defined margins and the site of SFSR was less prominent. Ultrasonographic findings included a mild amount of smooth new bone formation on the cranial aspect of R1 at the site of the synostosis. Compared with the right side, mild cranial displacement of R1 at the site of the SFSR was suspected and the scalene muscles appeared mildly thinner between the site of SFSR and the pectoralis muscles (Figure 3). Comprehensive radiographic and ultrasonographic examinations of the left forefoot were performed. Podotrochlear syndrome (navicular disease) was diagnosed, and a positive response to a distal digital nerve block was observed. Consequently, horse 5 had clinical manifestations unrelated to the SFSR, and the SFSR was considered an incidental finding. No follow-up information was available.

A 6-year-old Thoroughbred gelding (horse 6) used for steeplechase events was referred initially because of right hind limb lameness of 2 weeks' duration. The horse had no history of trauma. Physical examination revealed muscle atrophy of the neck and back. Results of lameness examination were similar to those of horses 2, 3, and 4. A mediolateral radiographic view of the

left and right shoulder joints and proximal portion of R1 was requested by the referring veterinarian, revealing an abnormal cranial orientation of the left R1. A left-sided SFSR was clearly identified on a craniomedial-caudolateral radiographic view of R1 and R2 (Figure 2). Radiographic and ultrasonographic findings for the SFSR were identical to those described for horse 5. Because this horse had lameness contralateral to the SFSR, its clinical manifestations were considered unrelated to the SFSR, which was considered an incidental finding. According to the owner and the referring veterinarian, the horse never developed lameness on the side of the SFSR.

All 6 horses were evaluated over a 2-year period. Overall, the results of ultrasonographic examination indicated various degrees of new bone formation on the cranial aspect of R1 at the site of SFSR. To compare horses with (ie, horses 1, 2, 3, and 4) and without (ie, horses 5 and 6) a suspicion of clinical signs associated with SFSR, the amount of new bone formation and the degree of irregularity of its margins were graded from 0 to 3 (0 = normal, 1 = mild, 2 = moderate, and 3 = severe). Mean grade of new bone formation for horses with clinical signs (2.2/3) was higher than that of horses with no clinical signs (0.5/3). Because we suspected cranial displacement of R1 at the site of the SFSR, the depth of the most prominent and modeled part of R1 (ie, the site of SFSR) was measured on ultrasonographic images and compared with the depth of the contralateral R1, and ratios of these values were calculated (Figure 3). Mean depth ratio at the level of the SFSR was lower for horses with clinical signs (0.55) than for horses with no clinical signs (0.83). Consequently, we considered the presence of the SFSR to be associated with greater cranial displacement of R1 for horses with versus without a suspicion of clinical signs associated with the SFSR.

Moreover, the scalene muscles appeared thinner between the site of SFSR and the pectoralis muscles for all 6 horses, and this feature was more conspicuous for horses 1 and 3. We attempted to use the subclavian artery as a landmark in ultrasonographic images and assess the position of this artery relative to the site of SFSR. The subclavian artery crossed R1 distal to the site of SFSR for all horses except horses 2 and 4, in which R1 crossed the artery proximal to the site of SFSR.

Discussion

To the authors' knowledge, the horses of the present report represented the first reported cases of SFSR in mature horses. Abnormalities of the cranial ribs are a reported cause of recurrent forelimb lameness, and lesions in the adjacent brachial plexus (plexus brachialis) have been suspected in affected horses.[5,6] Unilateral SFSR in horses has been mentioned only in a clinical commentary.[7] In humans, ipsilateral SFSR is not uncommon and was first described by anatomists.[8] The condition is usually recognized as an incidental finding identified on postmortem examination or thoracic radiography.[9] It is also regarded as a predisposing factor for so-called thoracic outlet syndrome, which causes musculoskeletal pain or compression of the neurovascular structures associated with the brachial plexus cranial to R1.[9–11]

The brachial plexus in horses is formed by the convergence of the ventral ramus of the nerves from C6 through T2. These roots converge toward the cranial aspect of R1, where they fuse. At this level, the brachial plexus passes between the scalene medius muscle dorsally and the scalene ventralis muscle ventrally and is parted from the subclavian vessels by the scalene ventralis muscle. The nerves arising from the brachial plexus join the axillary vessels more distally and laterally.[12]

Synostosis of R1 and R2 is believed to affect 0.3% to 2% of the human population.[11,13] Over the 2-year period during which the horses of the present report were evaluated, the prevalence of SFSR in our referral hospital population was 0.3%, although this statistic is unlikely to represent the general horse population. In humans, SFSR is generally considered a congenital malformation that develops unilaterally in 95% of affected individuals. The SFSR was unilateral in all horses of the present report.

In humans, fusion abnormalities are classified as bicipital rib type with fused ventral ends and shafts but separate dorsal ends, bridged rib type with fused shafts but separate dorsal and ventral ends, or forked rib type with fused dorsal ends but a separate shaft as well as separate ventral ends. The most common type in humans is the bicipital rib type,[14] and this was the only type identified in the 6 horses of the present report. Human case reports[9,11,13,14] of SFSR describe a hypoplastic, misshaped R1 positioned more cranially than usual, inserting on the cranial margin of R2 with bony exostosis at the site of fusion and a broadened common shaft. All of these features were identified in the horses and are known to be a cause of thoracic outlet syndrome in humans by compression of the adjacent structures.[9–11,13]

The radiolucent line at the site of SFSR could be mistaken for a fracture, particularly on underexposed radiographs where the SFSR is not clearly visible. Indeed, a moderate increase in radiopharmaceutical uptake was identified over R1 in horse 1 of the present report, which is a finding commonly reported for horses with R1 fracture.[6,15] A correctly exposed radiograph with good visibility of the site of SFSR and the common shaft allows ruling out of possible rib fractures. Several humans with thoracic outlet syndrome and SFSR have reported a traumatic injury before the onset of clinical signs,[10] although none of the horses of the present report had a history of traumatic injury. Nevertheless, luxation of R1 or some degree of instability at the site of SFSR could not be excluded and was suspected in horse 1 given the radiographic and scintigraphic findings. We considered radiography the gold standard technique for the diagnosis of SFSR, but ultrasonography allowed more accurate evaluation of R1 cranial displacement and the amount of irregularly marginated new bone formation

at the site of SFSR. Ultrasonography also allowed evaluation of the anatomic relationships between the bone lesions and adjacent soft tissues. Nevertheless, it may be difficult to differentiate SFSR and R1 fracture by means of ultrasonography alone. Furthermore, because SFSR is due to abnormal segmentation of bony tissue during development, associated anatomic variations may exist in the disposition of vessels and nerves.[16] Consequently, the crossing of the subclavian artery over R1 should not be used as a landmark to determine the proximodistal location of the SFSR.

Three types of thoracic outlet syndrome have been identified in humans: a neurologic type with brachial plexus or related nerve compression, a vascular type with subclavian vessel compression, and a musculoskeletal type with pain secondary to muscular compression. The neurologic type is the most common, affecting 97% of human patients.[17,18] Numbness, paresis, and paresthesia have been reported[10,11] for humans with neurologic thoracic outlet syndrome. All horses of the present report with clinical signs suspected as associated with the SFSR (ie, horses 1 through 4) had lameness and a shortening of the cranial phase of the stride in the affected limb as well as a higher mean grade of new bone formation and cranial displacement at the site of fusion, compared with horses in which the SFSR was an incidental finding (horses 5 and 6). Some degree of paresis secondary to nerve compression was suspected for horses 1 through 4, although this remained unproven because of the lack of postmortem examination. It would have been interesting to perform electromyography to assess and document possible neuropathy.[19,20] Musculoskeletal pain or a vascular injury secondary to the deformation of R1 could also have explained the observed gait abnormalities. Nevertheless, in horse 1, the lameness remained unchanged following 2 weeks of NSAID treatment. Moreover, vascular compression of the subclavian vessels in the interscalene space is unlikely in horses because the subclavian vessels cross R1 distal to the scalene ventralis muscle, contrary to the situation in humans.[12] We propose that this would suggest that gait abnormalities in the horses with SFSR were most likely related to a neurologic or mechanical cause. In the future, gabapentin administration could be considered for horses with SFSR in which neuropathy is suspected or confirmed without improvement of the lameness following NSAID treatment.

On the basis of the findings reported here, SFSR should be considered as a differential diagnosis for horses with forelimb lameness and shortening of the cranial phase of the stride, without improvement following distal limb diagnostic anesthesia. Diagnosis of this condition in mature horses is difficult because of the limited options available for obtaining diagnostically useful images for this anatomic region and the possibility that this congenital abnormality could remain clinically silent. Postmortem examination of affected horses could yield a better understanding of the pathophysiologic mechanisms and clinical incidence of SFSR in horses.

Acknowledgments

Supported by the Conseil Régional de Basse-Normandie, European Parliament (European Regional Development Funds), Pôle de Compétitivité Filière Equine, and Hippolia.

References

1. Butler JA, Colles CM, Dyson SJ, et al. Chapter 6. The shoulder, humerus, elbow and radius. In: *Clinical radiology of the horse*. 3rd ed. Oxford, England: Blackwell Science, 2008;273–300.
2. Coudry V, Thibaud D, Riccio B, et al. Efficacy of tiludronate in the treatment of horses with signs of pain associated with osteoarthritic lesions of the thoracolumbar vertebral column. *Am J Vet Res* 2007;68:329–337.
3. Denoix JM, Dyson SJ. Chapter 52. The thoracolumbar spine. In: Ross MW, Dyson SJ, eds. *Diagnosis and management of lameness in the horse.* 2nd ed. St Louis: Elsevier-Saunders, 2011;592–605.
4. Parks AH. Chapter 27. The foot and shoeing. In: Ross MW, Dyson SJ, eds. *Diagnosis and management of lameness in the horse.* 2nd ed. St Louis: Elsevier-Saunders, 2011;282–309.
5. Beech J, Bernard WV. Chapter 11. Neurological examination and neurological conditions causing gait deficits. In: Ross MW, Dyson SJ, eds. *Diagnosis and management of lameness in the horse*. 2nd ed. St Louis: Elsevier-Saunders, 2011;136–145.
6. Dahlberg JA, Ross MW, Martin BB, et al. Clinical relevance of abnormal scintigraphic findings of adult equine ribs. *Vet Radiol Ultrasound* 2011;52:573–579.
7. Denoix JM. Thoracolumbar malformations or injuries and neurological manifestations. *Equine Vet Educ* 2005;17:191–194.
8. Turner W. Cervical ribs and the so called bicipital ribs in man, in relation to corresponding structures in the Cetacea. *J Anat Physiol* 1883;17:384–400.
9. Anupama K, Prathap Kumar J, Radhika PM. An unusual case of synostosis of first and second rib. *Int J Anat Res* 2013;01:104–106.
10. Sanders RJ, Hammond SL. Management of cervical ribs and anomalous first ribs causing neurogenic thoracic outlet syndrome. *J Vasc Surg* 2002;36:51–56.
11. Reidler JS, Das De S, Schreiber JJ, et al. Thoracic outlet syndrome caused by synostosis of the first and second thoracic ribs: 2 case reports and review of the literature. *J Hand Surg Am* 2014;39:2444–2447.
12. Barone R, Simoens P. Chapitre II. Nerfs spinaux, III-plexus brachial. In: *Anatomie comparée des mammifères domestiques. Tome 7: neurologie II, système nerveux périphérique, glandes endocrines, esthésiologie.* Paris: Vigot, 2010:169–229.
13. Etter LE. Osseous abnormalities in thoracic cage seen in forty thousand consecutive chest photoroentgenograms. *Am J Roentgenol* 1944;51:359–363.
14. Anita R, Archana R, Jyoti C, et al. Synostosis of first and second rib—case report. *J Anat Soc India* 2009;58:189–191.
15. Ross MW, Stacy VS. Chapter 19. Nuclear medicine. In: Ross MW, Dyson SJ, eds. *Diagnosis and management of lameness in the horse*. 2nd ed. St Louis: Saunders, 2011;215–234.
16. Gupta V, Suri R, Rath G, et al. Synostosis of first and second thoracic ribs: anatomical and radiological assessment. *Int J Anat Var* 2009;2:131.
17. Sanders RJ, Hammond SL, Rao NM. Diagnosis of thoracic outlet syndrome. *J Vasc Surg* 2007;46:601–604.
18. Meyer R, Jones KJ. Thoracic outlet compression syndrome. In: Wolfe SW, Hotchkiss RN, Pederson WC, et al, eds. *Kozin Green's operative hand surgery*. 6th ed. Philadelphia: Churchill Livingstone, 2011;1015–1034.
19. van Wessum R, Sloet Van Oldruitenborgh-Oosterbaan MM, Clayton HM. Electromyography in the horse in veterinary medicine and in veterinary research—a review. *Vet Q* 1999;21:3–7.
20. Wijnberg ID, Back W, De Jong M, et al. The role of electromyography in clinical diagnosis of neuromuscular locomotor problems in the horse. *Equine Vet J* 2004;36:718–722.