Complications following tarsal arthrodesis using bone plate fixation in dogs.

S.P. Roch, R.A.S. Mitchell, C. Downes, T.J. Gemmill, D.N. Clements, C. Macias, W.M. McKee

Tarsal arthrodeses are salvage procedures performed for the treatment of hock conditions including intractable tarsal pain, severe tarsal fractures, shearing injuries and tarsal joint instability (McKee 1994). Pantarsal arthrodesis is the fusion of the talocruural, intertarsal and tarsometatarsal joints whereas partial tarsal arthrodesis is the fusion of the intertarsal and/or tarsometatarsal joints. Adequate joint exposure, meticulous articular cartilage debridement, rigid fixation and the application of cancellous bone graft are prerequisites for successful arthrodesis.

Previously reported complications following tarsal arthrodesis procedures include calcaneal fracture, distal limb swelling, implant loosening or breakage, sepsis, angular and rotational deformity of the distal limb, persistent lameness, wound dehiscence and pressure sores. Complication rates ranging from 25% to 80% have been reported with tarsal arthrodesis procedures (Allen 1993, Dyce and others 1998, Muir and Norris 1999, Fettig and others 2002, McKee and others 2004).

A retrospective study was performed to determine the incidence of major and minor complications following either pantarsal (PanTA) or partial tarsal arthrodesis (ParTA) with bone plate application. A previously unreported complication, plantar necrosis (PN) is also described. Major complications were defined as those requiring additional surgery or where the complications or necessary treatment had serious implications for the patient, for example sepsis, amputation or euthanasia. Plantar necrosis (PN) was defined as the postoperative loss of soft tissue from the plantar surface of the tarsus and metatarsus including the metatarsal pad. All other complications were considered minor.

Intraoperative complications were reported in 12/40 (30%) cases and included difficult wound closure (n=6), rotational deformity (n=3), suboptimal calcaneotibial or talar screw placement (n=3) displaced metatarsal bones (n=2) and plate malalignment (n=1). Postoperative complications were reported in 30/40 (75%) and included major complications in 13/40 (32.5%) and minor complications in 17/40 (42.5%). Major complications included PN (n=6), implant deformation or loosening (n=3), sepsis (n=2), severe pressure sores (n=1), and wound dehiscence (n=1). Bodyweight, medial plate application and PanTA appeared statistically significant risk factors for the development of major complications.
Minor complications included pressure sores (n=13), limb swelling (n=2), screw bending that did not necessitate implant removal (n=2) and wound breakdown (n=1). Minor complications were common and as with previous studies, were often secondary to prolonged external coaptation (Dyce and others 1998, Muir and Norris 1999, Fettig and others 2002, McKee and others 2004).

All minor complications resolved following dressing removal or symptomatic treatment.

**Plantar necrosis**

The most common major complication recognised was PN (6 / 40 cases, 15%). The skin of the plantar metatarsus and the deep tissues of the metatarsal pad became devitalised in all affected cases with the skin covering digits II and V variably affected. The median time to presentation for the assessment of PN following surgery was 16 days (range 6-18 days). PN was only recognised where debridement of the tarsometatarsal joint had been performed although tarsometatarsal joint debridement was not significantly associated with the development of PN. Plantar necrosis was noted in a significantly higher number of cases plated medially (5/15 cases, 33%) than cases plated laterally (1/ 25 cases, 4%). Surgeon experience was not significantly different from cases which did not develop PN. Difficulty achieving tension free closure was also reported in two cases. Age, weight, type of arthrodesis, tarsometatarsal arthrodesis and surgery by a resident were not associated with the development of PN and plantar necrosis was not observed in any cases treated by intertarsal joint debridement.

Plantar necrosis was initially managed by surgical debridement and wound dressing in all dogs. Two cases healed following prolonged wound management although one remained persistently lame. Amputation of digit II was necessary in three cases of which one dog was additionally treated with a free skin graft. Limb amputation was performed in one case. One dog was euthanased as a result of PN.

In view of the consistent distribution and time of onset of the condition, we hypothesise that PN may occur following vascular injury sustained during articular cartilage debridement or during the application of plates and screws. Other factors such as excessive wound tension, inappropriate dressings and postoperative swelling of the distal limb may also be important. We have not recognised PN following other methods of tarsal arthrodesis with concomitant external coaptation.

The principle arterial supply to the hind paw distal to the tarsus originates from the cranial tibial artery. This vessel becomes the dorsal pedal artery at the level of the talocrural joint.
The dorsal pedal artery runs dorsal to the tarsometatarsal joint and gives rise to the perforating metatarsal artery which passes in a dorsoplantar direction, between the proximal second and third metatarsal bones to supply the plantar metatarsal arteries II, III and IV via the plantar arch.

The proximity of the dorsal pedal artery to the tarsometatarsal joint and the interosseous location of the perforating metatarsal artery may confer vulnerability to injury or occlusion during tarsal arthrodesis surgery. The medial location of the perforating metatarsal artery may predispose to vessel injury during medial plate application and explain the significant difference between medial and lateral plating. Vascular injury could occur by laceration during cartilage debridement of the tarsometatarsal joint or by direct drill / screw impingement. Vessel compression between the second and third metatarsal bones would appear less likely. Intraoperative haemorrhage was not recorded in any case affected by PN. The level of collateral circulation development via the medial and lateral plantar after injury to the perforating metatarsal artery is undetermined.

Excessive wound tension, postoperative swelling and inappropriate dressing or splint application may adversely affect perfusion of the distal limb. Due to the characteristic distribution of PN, the link with application of the bone plate on the medial side of the hock and the absence of PN following other tarsal surgeries and distal hind limb coaptation, it seems unlikely that these factors would individually cause PN. Collectively however, they may be important in preventing adequate collateral circulation.

Complication rates, both major and minor are common after tarsal arthrodesis procedures. Strict attention to surgical technique and proper postoperative coaptation is critical to reduce the potential for complications with tarsal arthrodesis. Plantar necrosis is a serious complication that occurred in 15% of cases with medial plate application and tarsometatarsal joint debridement significant risk factors. We suspect PN is associated with injury to the perforating metatarsal and/or dorsal pedal arteries, although postoperative wound swelling, excessive soft tissue tension and poorly applied postoperative dressings are also important.

References


Fettig, A. A., McCarthy, R. J. & Kowaleski, M. P. (2002) Intertarsal and tarsometatarsal arthrodesis using 2.0/2.7-mm or 2.7/3.5-mm hybrid dynamic compression plates. Journal of the American Animal Hospital Association 38, 364-369


