Navicular disease...remembrance of things past

Over the past few decades, the development of magnetic resonance imaging (MRI) for the diagnosis of equine lameness has revolutionized our understanding of what used to be termed ‘navicular disease’. Prior to MRI, navicular disease was diagnosed primarily by a positive response to palmar digital nerve blocks (PDNB) and identification of characteristic radiological findings; particularly changes involving the distal border synovial invaginations, originally referred to as nutrient foramina, or canals (1, 2). Scintigraphy provided highly sensitive but non-specific ancillary indications of navicular bone pathology, but soft tissue structures within the hoof capsule was largely inaccessible, apart from autopsy examinations (3). Given the limited diagnostic options that were then available, response to therapy was far from predictable (4).

With the advent of MRI and, more recently, contrast-enhanced computed tomography (CECT), the complexity of navicular disease pathology became apparent (5–7). Consequently, navicular disease was transiently elevated to the status of a syndrome, before being re-branded as the somewhat pedestrian but more encompassing entity, ‘palmar foot pain’ (PFP). Although PDNB localize the source of pain to a relatively small anatomical landscape, the horse’s digit contains many vital structures that can contribute to lameness issues. Comparative MRI and histological analyses of PFP cases have defined the range of primary navicular bone pathologies within the medullary cavity, flexor surface, dorsal articular surface, and the proximal and distal borders (8, 9). However, the clinical significance and ubiquity of lesions within the deep digital flexor tendon (DDFT) are now well recognized, and pathology in the distal sesamoidean impar ligament (DSIL), the collateral sesamoidean ligaments, and navicular bursa are also present in many cases (10–13). Horses diagnosed with PFP can be expected to have pathological changes within multiple structures within the navicular-DDFT complex (13).

Clearly, MRI has provided equine practitioners with an embarrassment of riches in terms of potential pathological causes for foot lameness, but our ability to identify the specific source(s) of pain in any given case is sadly lacking. In the current edition of VCOT, Olive and Videau used CECT to clearly demonstrate that the synovial invaginations penetrating the distal border of the navicular bone represent extensions of the distal interphalangeal joint (DIPJ) cavity (corroborating previous contrast radiology and diffusion studies) but do not communicate directly with the navicular bursa (14–16). This demonstration has significant implications for the underlying pathogenesis of distal border osteolysis, and also impacts our interpretation of responses to distal limb diagnostic analgesia.

Distal border synovial invaginations were originally considered to be nutrient foramina for the distal sesamoidean vasculature (2, 15, 17). Their number, distribution and morphological changes were linked to altered arterial patterns within the medullary cavity, intraosseous ischemia and lameness in navicular disease cases (1). More recent assessments have linked changes in the distal border synovial invaginations to adjacent DSIL pathology and distal navicular border fragmentation, perhaps reflective of a more general enthesopathic response along the distal navicular border (10, 18). Given that DSIL lesions are present in approximately 40% of lameness cases localized to the foot, it is hardly surprising that expansion and remodelling of the distal border synovial invaginations is a common indicator of navicular pathology (10). In light of their results, Oliver and Videau propose that distal synovial invagination remodelling should be considered to be an indication of DIPJ synovitis/arthropathy, as opposed to primary navicular pathology. Accepting the technical ac-
Accuracy of their suggestion, more generalized arthritic change in the DIPJ is not a consistent finding in horses with PFP, despite the fact that the navicular bone provides approximately 25% of the distal articular surface of the DIPJ (10, 11). Given the intimate connection between the DIPJ synovium and the DSIL and distal navicular bone border, it is likely that focal synovial inflammation develops in the palmar aspect of the DIPJ in response to primary damage to the DSIL origin (2, 11).

Oliver and Videau’s findings provide some clarity for the somewhat contradictory responses to regional anaesthesia observed in ‘navicular disease’ cases. In Wright’s 1993 analysis of 118 navicular disease cases, only 47.5% of cases were sound after palmar digital nerve blocks, while DIPJ analgesia was effective in 49 of 54 cases (91%), matching the responses to direct navicular bursa anaesthesia (20). Dyson and colleagues had similar outcomes in 46 PFP cases with distal DDFT lesions; only 24% were responsive to PDNB while DIPJ anaesthesia was effective in 68% of cases with primary DDFT lesions and 92% of cases with complex pathologies (21). Experimentally, Gough and colleagues demonstrated diffusion of mepivacaine between the DIPJ and navicular bursa in both directions, with approximately half the test cases registering mepivacaine concentrations at levels sufficient for desensitization (22). Whether by direct desensitization of contributing structures, trans-synovial diffusion from the palmar recess, or trans-osseous diffusion via the synovial invaginations, DIPJ anaesthesia impacts painful stimuli from structures palmar to the joint space and clinical responses to DIP desensitization need to be interpreted with these findings in mind. These results also provide a basis for using the DIPJ as a conduit for therapies targeting the navicular bone medullary cavity, although the therapeutic options available for PFP are currently far behind our diagnostic imaging capacity.

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References