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Article type : Case Report

Internal aorto-iliac thrombosis in a Thoroughbred: unsuccessful surgical thrombectomy, a proposed aetiopathogenesis and spontaneous partial regression

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Keywords: horse; thrombosis; internal iliac artery; arteriotomy

Summary

A 4-year-old Thoroughbred gelding with severe left hind limb lameness and colic-like signs lasting 30-60 minutes' post exercise was diagnosed with unilateral internal aorto-iliac thrombosis. The thrombus was identified using transrectal ultrasonography in the terminal aorta and was initially thought to extend into the left external iliac artery. However, attempted thrombectomy via a left femoral arteriotomy performed under general anaesthesia using This is the author manuscript accepted for publication and has undergone full peer review but has not been through the copyediting, typesetting, pagination and proofreading process, which may lead to differences between this version and the Version of Record. Please cite this article as doi: 10.1111/eve.12823

thrombectomy and angioplasty catheters was unsuccessful, as the thrombus was located in the left internal iliac artery. Transrectal ultrasound evaluation was repeated monthly for 12 months post operatively during which time the gelding remained asymptomatic at rest. As removal of thrombi in the external but not internal iliac artery may be possible, correct diagnosis of the location of the thrombus is critical in preventing unnecessary attempts at surgical correction. The aetiology remains unknown, but altered haemodynamics within the abdominal aorta with mechanical stresses during intense exercise are proposed as a cause of thrombus formation. The sequential monitoring of this case also conflicts the assumption that thrombi in this location propagate over time as partial regression was observed 6 months after referral and 9 months after onset of clinical signs.

Introduction

Aorto-iliac thrombosis is an uncommonly diagnosed, reportedly progressive vascular condition of the horse of unknown aetiology (Rijkenhuizen *et al.* 2009). Clinical signs include an exercise induced hind limb lameness which generally resolves promptly with the cessation of exercise (Azzie 1969; Maxie 1985), decreased digital pulses and saphenous fill and a palpably cooler affected limb (Rijkenhuizen *et al.* 2009). A diagnosis is most commonly made via transrectal ultrasonography with or without Doppler visualising the thrombus in situ (Reef *et al.* 1987), nuclear scintigraphy (Boswell 1999; Duggan 2004) or at necropsy (Oyamada *et al.* 2007). The prognosis is generally considered poor (Tillotsen 1966; Branscomb 1968; Brama 1996), but recent advances in surgical thrombectomy techniques may alter prior practitioner conceptions (Hilton 2008; Rijkenhuizen 2009).

Medical therapy has previously been attempted with varying success (Branscomb 1968; Duggan 2004). More recently, attempts at surgical removal with the use of balloon angioplasty (Hilton *et al.* 2008) and graft thrombectomy catheters (Brama 1996; Rijkenhuizen 2009) via a transverse femoral arteriotomy have been successful, with up to 65% of patients regaining athletic activity (Rijkenhuizen *et al.* 2009). Despite this, the prognosis is still generally considered poor, particularly in cases with bilateral involvement (Maxie 1985; Boswell 1999).

This is not the first reported case of aorto-iliac thrombosis involving the internal iliac artery in the horse, although cases more commonly involve the external iliac artery (Warmerdam 1997; Oyamada 2007). The purpose of this report is to highlight the importance of accurate diagnosis of thrombus location before surgical treatment is attempted, discuss

potential aetiologies which have not previously been explored in the literature and question the assumption that this disease is progressive.

Case Details

History and diagnosis

A 4-year-old Thoroughbred gelding was referred to the University of Melbourne's Equine Centre with a history of poor performance, left hind limb lameness and clinical signs of colic following a race start. Moderate left hind limb lameness at the walk was observed following swimming exercise 3 months prior to presentation, with signs resolving after a 30-60 minute period of rest. A complete blood count and serum biochemistry were unremarkable. Swimming exercise was ceased and the horse continued to be exercised under saddle for 3 weeks prior to racing, where he then performed below expectations. Following this race he was severely left hind limb lame, appeared distressed with colic-like signs and became recumbent which resolved following the administration of flunixin meglumine ([Flumav¹] 1.1 mg/kg bwt i.v) and detomidine hydrochloride ([Equisedan® Vet²] 0.01mg/kg bwt i.v). No abnormalities were detected on lameness examination the following day, however a left hind limb lameness could be induced with further swimming exercise. The referring veterinarian performed a transrectal ultrasound examination and a thrombus in the abdominal aorta and left iliac artery identified.

On presentation digital pulses in the left hind limb were palpable and within normal limits. Saphenous fill was present and adequate, and the limb was warm to touch. Lameness was not evident at the walk or trot in-hand, nor at a trot or canter on a lunge rein. Cardiac auscultation revealed no abnormalities. An exercising electrocardiogram (ECG) was performed at the canter on the lunge rein for approximately 30 minutes. No ECG abnormalities were noted pre, during or post exercise and lameness could not be induced. Digital pulses, saphenous fill and palpable heat were unchanged. A resting echocardiogram revealed no abnormalities. A faecal egg count indicated 60 strongyle eggs per gram.

On transrectal ultrasonography with a 7.5 MHz linear transducer, an irregularly shaped, smooth margined, crescent shaped mass was identified within the left external (presumed) iliac artery extending from the quadrifurcation of the abdominal aorta. The mass was adherent to the dorsolateral margin of the vessel and occluded approximately 60% of the arterial diameter at its most occlusal point, confirmed via colour flow Doppler (Fig 1)

extending from the aortic branch for approximately 10 cm. It was homogenous in echogenicity and echotexture, hypoechoic relative to the surrounding retroperitoneal fat and hyperechoic relative to the echogenic arterial blood. Blood flow was visualised moving unimpeded down the ventromedial aspect of the artery. A diagnosis was subsequently made of unilateral aorto-iliac thrombosis involving the terminal aorta and left external iliac artery. Given the occlusal diameter, presumed location and impact on performance, plans for surgical thrombectomy were made. Medical therapy was not attempted given the chronicity and size of the thrombus.

Attempted surgical correction

The gelding was administered procaine penicillin ([Propercillin³] 22 mg/kg bwt i.m), gentamicin sulphate ([Gentam 100³] 6.6 mg/kg bwt i.v), and phenylbutazone ([Salbute⁴] 4.4 mg/kg bwt i.v) before induction of general anaesthesia with a premedicant of xylazine hydrochloride ([Xylazil-100³] 1.1 mg/kg bwt i.v), followed by diazepam ([Pamlin⁴] 0.1 mg/kg bwt i.v) and ketamine hydrochloride ([Ketamine⁴] 2.5 mg/kg bwt i.v). Anaesthesia was maintained with isoflurane and oxygen, and the gelding positioned in left lateral recumbency with the hindquarters tilted to a dorsolateral position. The right hind limb was secured in flexion and abduction to expose the medial aspect of the left thigh. A buffered and balanced polyionic crystalloid solution (Hartmanns⁵) was administered intravenously at a rate of approximately 10 litres per hour throughout the procedure.

Following aseptic preparation of the surgery site, the left femoral artery was approached via a 10 cm incision through the skin and subcutaneous tissue, centred over the site where the saphenous vein diverges from the superficial to deep tissues approximately 15 cm proximal to the femoropatellar joint. Soft tissue dissection was performed down to the femoral triangle exposing the femoral artery, which was then mobilised by careful dissection. Vascular ties were placed proximal and distal to the proposed arteriotomy site and the gelding administered 100 IU/kg bwt of low molecular weight heparin (Fragmin⁶) intravenously before a 0.5 cm transverse incision was made, with haemorrhage cleared from the surgical field using suction.

A 50 cm 6F Fogarty® graft thrombectomy catheter⁷ with an extended spiral diameter of 6.0 mm and retracted spiral diameter of 18 mm was passed proximally through the transverse arteriotomy site in an attempt to engage the thrombus. Multiple passes failed to retrieve any thrombotic material. An over-the-wire 7F Fogarty® thru-lumen embolectomy

(balloon angioplasty) catheter⁷, with a maximum inflated balloon diameter of 14 mm, with corresponding size 8F introducer and 0.038 inch diameter guide wire was then passed proximally in attempts to engage the thrombus. When this was also unsuccessful, a transrectal ultrasound examination was repeated which clearly identified the catheter in the external iliac artery, with the thrombus visible in the adjacent internal iliac artery (Fig 2). Further attempts at thrombus removal were aborted due to inaccessibility of the internal iliac artery. The arteriotomy site was closed with a double, continuous inverting layer with 4-0 polydioxanone suture and overlying fascia, subcutaneous tissue and skin closed routinely.

Post-operative management

Recovery from general anaesthesia was assisted with ropes and was uneventful. Administration of procaine penicillin and gentamicin sulphate continued for 48 hours, and phenylbutazone for 7 days (4.4 mg/kg bwt i.v q24h for 3 days, 2.2 mg/kg bwt per os q24h for 4 days). Light hand walking was commenced immediately post operatively, but adjunctive treatment with acetylsalicylic acid and low molecular weight heparin was not deemed necessary given the thrombus was not removed and hence the risk for further thrombogenic foci not applicable. The only complication observed was the formation of a moderately sized seroma at the incision site diagnosed via transcutaneous ultrasonography, which subsided over 7 days.

Sequential ultrasonographic evaluation

The gelding was subsequently retired from racing. The thrombus was assessed via transrectal ultrasonography and colour flow Doppler monthly with the size, shape, margins, opacity and configuration compared. Over 5 months, the thrombus remained at a maximal occlusal diameter of approximately 60% (vessel area measured on still images at a later date 353 mm², thrombus 207 mm²). The thrombus was not observed to propagate further distally in the internal iliac artery or reform in the abdominal aorta, and the right sided vessels remained unaffected. However, 6 months after initial presentation the thrombus had regressed to a maximal occlusal diameter of 35% (vessel area 295 mm², thrombus 104 mm²). The gluteal vasculature was examined via transcutaneous ultrasound and no migration of the thrombus observed. No other abnormalities in the left hind limb vasculature were noted ultrasonographically.

Discussion

Aorto-iliac thrombosis in the horse has been reported to have a prevalence of up to 1.5% in the flat-racing Thoroughbred population of South Africa (Azzie 1969), with no more comprehensive or new data available. The condition is repeatedly described in veterinary texts (Reef 1996; Young 2004; Fischer 2012), although little has been added to the understanding of the aetiopathogenesis of the disease in the last twenty years. Thoroughbred racehorses are over represented, presumably due to high intensity exercise exacerbating clinical signs coupled with the close observation of these animals (Maxie and Physick-Sheard 1985). Although reported in foals and geriatric horses, horses of racing age are over represented again likely reflecting the in-work racehorse demographic (Moore 1998; Duggan 2004).

No definitive pathogenesis has been determined, with multiple aetiologies proposed including *Strongylus vulgaris* larval migration in the abdominal aorta (Oyamada *et al.* 2007) and systemic infection or sepsis (Mayhew 1975; D'Angelo 2006). The role of *S. vulgaris* in disease pathology remains contentious. Some authors strongly advocate parasitism as the cause of circulatory disturbances in the abdominal aorta, associating the decline in prevalence of parasitism with increased anthelmintic use with the subjectively decreased incidence of aorto-iliac thrombosis (Oyamada *et al.* 2007). Conversely, authors critical of the role of larval migration in the disease argue the lack of histological findings of verminous arteritis (larvae, eosinophils, plaque formation) at the aortic quadrifurcation and that *S. vulgaris* larvae have a predilection site for the cranial mesenteric artery, state this aetiology as unlikely (Merrillat 1944; Azzie 1972; Jubb 1993a). Whilst the complete anthelmintic history was not available for this gelding, he had a significant economic value and had been housed at reputable farms that have well established anthelmintic programs. The limitations of faecal egg counts are acknowledged as not detecting larval stages of parasites, but as the gelding in this case report had a low worm burden with no other clinical signs of parasitism, it is considered an unlikely contributing factor (Nielsen *et al.* 2013).

The incidence of clinical disease in males has been consistently higher in previous reports (Azzie 1969, 1972; Maxie 1985), the reason for which is unknown. Hormonal influences have been proposed in similar human conditions with low oestrogen levels associated with reduced fibrinolysin production and an increased incidence of thrombosis, suggesting that high androgen levels may be important (Azzie 1969). Authors have also queried whether collateral supply to the hind limbs via the ovarian and uterine arteries in mares require more severe vascular obstruction prior to clinical signs observed (Maxie and

Physick-Sheard 1985). It may also be that poor performance in mares is less likely to be investigated with these horses preferentially retired for breeding. Of note, one author has proposed a hereditary predisposition, with one stallion producing six affected progeny and another mare producing four (Azzie 1969).

Aortic thromboembolism is also observed in cats secondary to hypertrophic cardiomyopathy when emboli from the left atrium or ventricle dislodge and enter peripheral circulation (Schoeman 1999). A cardiac examination conducted in this gelding did not reveal any abnormalities, however an ECG was performed at a canter rather than during strenuous exercise due to the risk of inducing severe lameness and distress. Cardiovascular disturbances altering blood flow and contributing to arterial and cardiac thrombosis through endothelial injury (Kiyomura *et al.* 2006) should be ruled out horses with aorto-iliac thrombosis.

We propose that the most likely cause of thrombus formation in athletic horses is due to mechanical stresses and secondary alterations in haemodynamics. The dorsal wall of the abdominal aorta is on the outer circumference of the spinal arc during flexion, with the internal iliac arteries and branches closely adhered to the deep sacral fascia preventing movement in relation to adjacent structures. Subsequent endothelial (*tunica interna*) damage can theoretically occur secondary to stretching with spinal flexion, or increased intra-abdominal pressure at the gallop (Azzie 1969, Lorenzo-Figueras and Merritt 2002, Maxie 1985). When the normal laminar blood flow becomes turbulent, platelets are brought into contact with the endothelium and the retarded inflow of clotting factor inhibitors may permit an environment for thrombus formation (Kiyomura *et al.* 2006). In addition, the catecholamine release at the onset of exercise in horses is also associated with splenic contraction and a significant increase in haematocrit (Young 2004), which could theoretically predispose to thrombus formation as described in pregnant women with haemoconcentration (Kiyomura *et al.* 2006).

A recent review of the human literature assessing sports-related vascular insufficiency in cyclists supports the theory of alterations in haemodynamics, postulating that the main aetiological factor is mechanical strain secondary to posture and performance intensity (di Alencar *et al.* 2013). Lesions were commonly identified in the external and internal iliac arteries, with 81.5% (119/146) of cases being male and 18.5% (27/146) being female. Tortuosity caused by flexion of the coxofemoral joint and arterial compression by the psoas muscle during pedalling are the proposed factors responsible for blood flow reductions, with

increased arterial tension resulting in endothelial damage, stenosis and claudication. It is also suggested that a combination of the repetitive movements and vascular compression cause the endothelium to convert from an anti-thrombogenic to a pro-thrombotic state, resulting in the production of fibronectin and von Willebrand factor (di Alencar *et al.* 2013). Unlike the horse, the primary lesion is endofibrotic stricture of vessels, which can cause secondary arterial thrombosis if severe. Although different manifestations of occlusion, similar factors may contribute to aorto-iliac thrombosis in the horse.

A diagnosis of aorto-iliac thrombosis is most commonly made via transrectal ultrasonography. The thrombus location in this case was incorrectly identified before surgery, resulting in unnecessary patient morbidity and potential cost to the client. Although the difficulties of imaging the iliac arteries are described (Edwards 1988; Whitcomb 2016), little information is available to the practitioner on how to ascertain the difference transrectally between the internal and external iliac arteries. Anatomically, the aortic quadrifurcation occurs on dorsal midline at the level of the fifth or sixth lumbar vertebrae. The internal iliac arteries pass caudally under the wings of the sacrum, ventrally along the pelvic surface of the ilial body and along the ventral border of the iliac head of the *obturatorius internus*, before dividing ventral to the lumbosacral articulation into the caudal gluteal and internal pudendal arteries which supply the gluteal musculature (Sisson *et al.* 1975). The external iliac artery branches from the abdominal aorta at the level of the fifth lumbar vertebrae generally cranial to the origin of the internal, but individual variation may result in some branching at the level of the most caudal lumbar vertebrae. The external iliac artery travels down the side of the pelvic inlet adjacent to the tendon of the psoas minor before reaching the level of the cranial border of the pubis and continuing down the distal limb as the femoral artery (Sisson *et al.* 1975). The anatomical landmarks should be identified ultrasonographically to ensure the correct vessel is identified to aid in treatment decision making.

Aorto-iliac thrombosis is repeatedly referred to as a continually progressive vascular disease (Maxie 1985; Edwards 1987; Rijkenhuizen 2009) and has historically carried a poor prognosis (Tillotsen 1966; Branscomb 1968; Brama 1996). The sequential monitoring of this case showed no lesion propagation and an eventual reduction in thrombus size and degree of arterial occlusion, consistent with fibrotic re-organisation and contracture (Reef 1988). The reduction could also be due to thrombus recanalisation following the proteolytic activity of recruited inflammatory cells coupled with mechanical retraction and proangiogenic enhancement as postulated in the human literature (Chabasse *et al.* 2015). Although the

gelding has yet to be trialled under strenuous exercise, given the reduction in size and minimal impedance to blood flow, the prognosis for returning to high level performance should be considered fair.

Although the aetiopathogenesis of aorto-iliac thrombosis is unknown, the over representation of the disease in racehorses suggests mechanical alterations in haemodynamics as a major contributing factor. Aorto-iliac thrombosis is a differential diagnosis of exercise induced hind limb lameness and efforts should be made to ensure correct lesion localisation to prevent unnecessary efforts to remove the thrombus surgically. With recent thrombectomy applications from the human field in addition to adjunctive medical therapy, there is no reason to encompass this disease as carrying a poor prognosis in all cases. Furthermore, the spontaneous reduction in thrombus size contradicts prior assumptions that the disease is progressive.

Authors' declaration of interests

Authors declare no conflicts of interest.

Ethical animal research

Owner consent was obtained for all aspects of case management; institutional animal ethics approval was not required.

Source of funding

No funding was received.

Antimicrobial stewardship policy

Authors declare no off label antimicrobial use.

Authorship

All authors contributed to case diagnosis, treatment and management. The report was drafted by K. A. Lloyd and revised by listed authors. The final document was approved by all authors prior to submission.

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⁴Ceva Animal Health Pty. Ltd, Glenorie, New South Wales, Australia.

⁵Baxter Healthcare, Toongabbie, New South Wales, Australia.

⁶Pfizer, Auckland, New Zealand.

⁷Edwards Lifesciences, Irvine, California, USA.

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Figure legends

Fig 1: Transrectal Doppler ultrasound image of the thrombus (T) in the iliac artery at time of diagnosis.

Fig 2: Intra-operative transrectal ultrasound images: Left: the hyperechoic catheter and gas (arrow) within the left external iliac artery and the adjacent abdominal aorta (A) Right: the thrombus (T) in the left internal iliac artery.

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Title:

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Date:

2019-07-01

Citation:

Lloyd, K. A., Vallance, S. A., Denton, M. J. & Steel, C. M. (2019). Internal aorto-iliac thrombosis in a Thoroughbred: Unsuccessful surgical thrombectomy, a proposed aetiopathogenesis and spontaneous partial regression. EQUINE VETERINARY EDUCATION, 31 (7), pp.365-369. <https://doi.org/10.1111/eve.12823>.

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