Case of Eagle Fern (Pteridium Aquilinum) Poisoning on a Pig Farm

Agnès Waret-Szkuta (a.waret@envt.fr)
IHAP, Université de Toulouse, INRA, ENVT, Toulouse, France https://orcid.org/0000-0002-6175-4418
Laura Jégou
IHAP, Université de Toulouse, INRA, ENVT
Marie Noelle Lucas
IHAP, Université de Toulouse, INRA, ENVT
Nicolas Gaide
ENVT
Hervé Morvan
Labocea
Guy-Pierre Martineau
IHAP, Université de Toulouse, INRA, ENVT

Case report

Keywords: Eagle fern, mortality, outdoor rearing, vitamin B1, intoxication

Posted Date: October 1st, 2020

DOI: https://doi.org/10.21203/rs.3.rs-83288/v1

Version of Record: A version of this preprint was published on January 4th, 2021. See the published version at https://doi.org/10.1186/s40813-020-00185-9.

Abstract

Background: Free-range pig farming represents a minor proportion of pig production in France but attracts an increasing number of farmers because of societal expectations and the opportunity to use fodder consumed in the meadow. However, this type of farming faces several sanitary challenges, including biosecurity, parasitic management, and contact with wild fauna and pathogenic flora.

Case presentation: Two Gascon pigs raised on an outdoor fattening farm in the Hautes-Pyrenees department of France were submitted after sudden death for necropsy at the National Veterinary School of Toulouse. The pigs were of two different breeds but from the same batch of 85 animals that had grazed on a 4-hectare plot of land being used for the first time. The combination of an in-depth interview with the breeder, the epidemiological information available, and the necropsy and histology examinations enabled the hypothesis of great eagle fern intoxication. Despite the small sample of animals available for diagnosis, the success of the administered therapy helped to confirm our diagnosis. In the short term, it was recommended that the animals be prevented access to the eagle fern by changing their pasture or by removing the plants. Vitamin B1 was administered in the feed in the amount of 1 mL per 10 kg body weight for two days (Ultra B®: 4.46 mg thiamine and 2.06 mg pyridoxine (vitamin B6) per kg body weight per day). A remarkable remission was observed, allowing 6 of 12 intoxicated animals with symptoms to survive (therapeutic success rate: 50%), but without compensating for the loss of the initial body condition. In total, out of the 85 animals in the batch after this intoxication, 6 animals died and 6 recovered.

Conclusion: The interest of this report lies in the scarcity of eagle fern intoxication cases reported in the literature, although it may become a significant problem along with the development of outdoor rearing. In this context, it should be included in the differential diagnosis of nervous system symptoms in swine. The case also underlines the importance of anamnesis and discussions with the farmer to guide the diagnosis and reminds us that this step is essential in the clinical process.

Background

Today, free-range pig farming represents 5% of pig production in France [1]. It faces several challenges, including the climate that may impact feed conversion [2], contact with the soil that may favour parasites, potential interaction with wild animals and ingestion of plants that may be toxic [3]. As this type of farming attracts an increasing number of farmers because of societal expectations [4] and the opportunity to use fodder consumed in the meadow [5, 6], it is most likely that plant poisoning following ingestion, such as with eagle fern, will become more important, although it has been rarely reported until now.

The great eagle fern (*Pteridium aquilinum*) is an invasive plant that grows globally in very diverse environments, leading to it being difficult control in pastures [7]. Intoxication following ingestion is well documented in ruminants, including cattle, sheep and wild cervids [8, 9]. In cattle, bracken fern ingestion

causes enzootic haematuria, characterized by lower urinary tracts haemorrhages and neoplasms of epithelial and mesenchymal origins [9]. Additionally, acute haemorrhagic syndrome, secondary to bone marrow aplasia and retinal atrophy have been documented in sheep [8], as has polioencephalomalacia [10].

In monogastric species, bracken fern poisoning is poorly documented. In horses, poisoning has been associated with the ingestion of poor quality hay containing a high proportion of big eagle fern [11], the use fern as bedding [12] or overgrazing [13]. Case reports in pigs are even rarer [14, 15, 16], probably because outdoor rearing is less prevalent, but it may also be due to the high resistance of pigs that can ingest this plant without being exposed to the poison for a period of six weeks [10].

Bracken fern contains several toxins including ptaquiloside, thiaminase, quercetin and bleeding factors [17, 18]. These toxic molecules, ptaquiloside and thiaminase in particular, are found in high concentrations in the young growing parts of the plants and in the rhizomes. Thiaminase is an enzyme that breaks down thiamine (an essential vitamin B1) into pyrimidine and thiazole. Since vitamin B1 is essential for the metabolism and maintenance of peripheral nerve myelin [10], vitamin B1 deficiency leads to peripheral or central neuropathy, with manifestations including impaired health status, weight loss and slight loss of movement coordination [11]. Symptoms suggestive of heart failure and anorexia have also been reported. Cardiopathy related to vitamin B1 deficiency has been studied in pigs to understand the pathogenesis of beriberi, a human disease caused by a nutritional deficiency in vitamin B1. One study showed that thiamine deficiency was associated with nonhypertrophic cardiac dilatation and histopathological lesions, including focal to diffuse necrosis of the myocardium and neutrophilic and mononuclear myocarditis [19].

The purpose of this report is to describe a case of great eagle fern poisoning with sudden deaths in one batch out of eight on an outdoor Gascon pig fattening farm in the Hautes-Pyrenees department of France. The affected batch of 85 pigs had been reared on a new pasture of 4 hectares.

Case Presentation

The case described here occurred in an outdoor fattening farm with 680 Gascon pigs in the Hautes-Pyrenees department of France. Every 2 or 3 months, 85 animals weighing approximately 30-40 kg arrive from a breeding farm and remain on the same plot until they are approximately a year old with an average carcass weight of 140 kg. The plot will then be subjected to a 2-month period with no pigs present on it. Eight batches are present simultaneously. The animals are fed with triticale flour in addition to what they find in the environment: acorns, chestnuts, grass, brambles, heather and elements from the soil excavation. Watering is provided by well water, which does not undergo any chemical treatment. To prevent parasites, the farmer adds Panacur 4%® to the drinking water at a dosage of 12.5 g per 100 kg of body weight every two months. Biosecurity is limited (there is no electric fencing, but wires are present).

In October 2019, the farmer noticed a loss of body condition and a loss of appetite in approximately ten pigs from a single plot. Breathing difficulties with an open mouth and complaints were observed in many

animals, as well as nervous system signs that manifested as a wobbling gait. There was no diarrhoea. Two pigs that died suddenly were brought for necropsy to the Veterinary School of Toulouse. One pig had been found dead three weeks before this. These animals came from the same batch of 85 animals and were grazed on 4 hectare plot of land being used for the first time.

The clinical signs reported by the breeder were partially non-specific, with signs of weight loss and loss of appetite, but this suggests a less acute evolution than the sudden mortality reported concomitantly. Nervous system symptoms suggested central nervous damage that might have been caused by a wide variety of aetiologies, both infectious (oedema disease) and non-infectious (nutritional deficiency or intoxication). The respiratory symptoms may have also had various causes and may or may not have been related to the nervous system symptoms. Parasites can also lead to death and are important to consider in particular in an outdoor setting, such as metastrongias (*Metastrongylus aprii*) or trichurias (*Trichuris suis*), for example. However, since the plot on which the pigs were located was being used for the first time for fattening and since parasite treatment was administered regularly, this hypothesis was not retained at first.

Epidemiologically, the phenomenon appeared that it could be contagious or anazootic. Since it was limited to a single batch at the time of the call, with a 15% morbidity, 3.5% mortality and 23% lethality, a contagious origin was not retained as first. However, given the context of African swine fever in Europe, particular attention was paid not to overlook the possible detection of this disease, especially as biosecurity was not optimal in this outdoor farm and the wild boar population is increasing in the region [20].

Post-mortem investigation

Two pigs that were found dead were submitted for complete necropsy: an 8 month-old, 89 kg sow and an 8 month-old, 59 kg male pig that died the day before. On the sow, external examination presented congestive ocular and oral mucosa. Cavity openings revealed 500 mL and 15 mL yellow translucent effusion consistent with transudate in the peritoneal and pericardial cavities, respectively. The lung presented a moderate cranio-ventral consolidation and marked diffuse interlobular oedema (Figure 1).

The mediastinal lymph nodes were enlarged 2.5 x 1 cm and 3.5 x 1 cm. Extensive fundic congestion (Figure 2), as well as foamy liquid in the tracheal lumen and diffuse petechiae on the diaphragm, was found.

On the male pig, gross lesions were limited to 70 mL of yellow pericardial effusion, diffuse fundic congestion and multifocal foci of congestion in the caecal mucosa without luminal abnormalities. The rest of the examined organs were within normal limits, including the gross external examination of the brain and meninges.

The necropsic examinations did not reveal any specific lesions supporting a definitive diagnosis of an infectious agent except for in the lung, where there were the lesions might have suggested enzootic

bronchopneumonia (*Mycoplasma hyopneumoniae*), and an underlying acute diffuse inflammatory lesion that did not allow the complete ruling out of a parasitic or viral interstitial pneumonia. At the time of necropsy, the brain, liver, and heart of both animals and the lung, kidney, mediastinal and mesenteric lymph nodes of the sow were harvested and fixed in 10% buffered formalin. After fixation, they are routinely processed in paraffin blocks, sectioned in 4 µm slices and stained with haematoxylin and eosin (HE) for microscopic evaluation.

At the same time, the farmer was contacted again to gather more information on the environment in which the animals evolved, in particular, on the possible neighbourhood issues and the plants present on the plot, bearing in mind that it was being used for the first time for pig fattening and considering the presence of nervous system signs, as well as the absence of clinical signs in the other groups present on the farm, all of which were on plots that had already been used for pig farming.

This second in-depth interview with the breeder revealed the significant presence of great eagle fern (*Pteridium aquilinum*) in the stockyard, which was confirmed by pictures sent by the breeder (Figure 3).

At that point, the lack of specificity of the necropsic findings associated with the presence of the fern without any change in the animals' management, particularly regarding the feed distributed, led to a strong suspicion of plant intoxication by this fern, which can lead to thiamine deficiency.

Histology

Samples taken from the male pig showed no abnormalities except in the brain. Lesions consisted of segmental cortical laminar and multifocal lesions in the brainstem, basal nuclei, and cerebellum, associated with acidophilic neuronal necrosis and spongiosis and admixed with gitter cell infiltration (Figure 4). Inflammation was observed in the leptomeninges and perivascular spaces, including endothelial activation, hyperaemia, and mixed leukocytic infiltration including mononuclear cells and eosinophils. Vascular fibrinoid necrosis and haemorrhages were occasionally seen. A diagnosis of polioencephalomalacia was assessed, suggesting a toxic or metabolic aetiology, including a toxic plant or a thiamine deficiency.

In the sow, the brain, lymph nodes, liver and kidneys were within normal limits. The heart showed lesions consistent with severe progressive polyphasic necrotizing myocarditis with mixed leukocytic infiltration, and the lungs showed leukocytoclastic necrotizing vasculitis with diffuse congestive and oedematous pneumonia (Figure 5).

A systemic infectious origin, notably viral, such as porcine circovirus type 2 (PCV2), was suggested without evidence of pathognomonic lesions, such as basophilic inclusions or granulomatous inflammation, and with myocardial lesions being found more often in runts or small piglets. Despite the nonspecificity, intoxication could not be ruled out, since pulmonary vasculitis was previously described in bracken fern intoxication of pigs [16].

Diagnostic and recommendations

Considering anamnesis, epidemiology, and post-mortem investigations and histology, the most likely diagnostic hypothesis retained was intoxication by great eagle fern. This was confirmed by the good response to the therapy that was implemented.

In the short term, it was recommended to prevent access of the animals to the eagle fern by changing their pasture or by removing the plants. Vitamin B1 was administered in the feed in the amount of 1 mL per 10 kg body weight for two days (Ultra B®: 4.46 mg thiamine and 2.06 mg pyridoxine (vitamin B6) per kg body weight per day). A remarkable remission was observed, allowing 6 of the 12 intoxicated animals with symptoms to survive (therapeutic success rate: 50%), but this did not compensate for the loss of initial body condition. In total, out of the 85 animals in the batch following this intoxication, 6 animals died and 6 recovered.

Discussion And Conclusion

Here, we report a case of eagle fern intoxication in the rearing of post-weaning, free-range fattening Gascon pigs. The interest in this case lies in the scarcity of cases reported in the literature and underlines the importance of anamnesis and discussions with the farmer to guide the diagnosis. This reminds us that this step is essential in the clinical process.

Symptoms suggestive of heart failure and anorexia reported by the farmer were consistent with what has been reported in the literature [14, 15, 16]. Focal or diffuse necrosis of the myocardium has also been recorded by some authors, with neutrophils and mononuclear cells present, similar to cardiomyocyte necrosis with neutrophilic infiltration observed in the sow in our case [19].

Drug treatment with thiamine (vitamin B1) should be administered as early as possible, but its effect is often reported to be illusory in all species [13]. In horses, the recommended dose is 500 mg to 1 g/day of thiamine intravenously on the first day and then intramuscularly for several days [11]. However, in our case, the administration of 4.46 mg thiamine and 2.06 mg pyridoxine (vitamin B6) per kg body weight per day in the feed was followed by a remarkable remission, with a therapeutic success rate of 50%, although without compensation for the loss of the initial body condition.

In the medium- and long-term, the aim is to reduce and remove great eagle fern. Its growth can be slowed by regular mowing several times a year, but this is a time-consuming technique [21]. The use of a fern-breaking roller works better, and its light weight allows the emerging broadleaf and grassy plants to grow and compete with the fern. In addition, there is little damage to the wildlife present as a result of using this tool. This remains the best method on the market to eradicate this fern and can be used on all soils. The best time for its use is between June and July, when the maximum rhizome reserves have been mobilized [22]. This method is effective but time-consuming, with the effects of mowing only becoming visible after two or three years of double mowing (end of June and end of July) [13]. A study conducted in 2006 and 2007 tested an alternative treatment based on 12% vinegar, which showed a good response in 2006 but regrowth in 2007 only a few months after treatment [23]. The authors hypothesized that there was a different soil moisture content during application between these two years. In the same study, a

hexazinone treatment was utilized and showed good results. Nevertheless, this product remains a broad-spectrum herbicide and is therefore not possible in our context, since the animals graze in the pasture during the entire year [24]. Therefore, for the time being, the mechanical method is still the most suitable method for control of great eagle fern.

If therapeutic success indicates that the main hypothesis retained was the most likely cause of the clinical signs in this case, analysing the stomach content and increasing the number of animals examined post-mortem could have strengthened our diagnosis before the implementation of a therapy. Indeed, here the diagnosis was based on the necropsy of two animals out of 85, i.e., 2% of the batch, and one can doubt the representativeness of the lesions observed in the individuals examined. Furthermore, considering neuronal necrosis, perivascular and meningeal inflammation and severe myocarditis, the hypothesis of encephalomyocarditis could have been investigated, but the pattern of cerebrocortical necrosis did not favour neutropic viruses, whose pattern is a multifocal asymmetrical distribution of inflammation, and there is currently no clinical case reported in France confirmed by immunohistochemistry on heart sections, for example. Finally, possible nutritional deficiencies could have been discussed with the farmer, as the growth of the pigs could probably be improved.

Although few cases of pig eagle fern intoxication have been reported in the literature, this case demonstrates that this hypothesis should be included in the differential diagnosis of nervous system symptoms and/or sudden death in swine, particularly in the context of outdoor rearing. In view of the development of this type of farming in connection with a strong societal demand, it is highly likely that diseases of parasitic or toxicological types will become increasingly important in this species in the future. To investigate these, a site visit would be ideal to visualize the facilities in place for the animals. Otherwise, as in our case, in-depth discussions with the breeder are crucial to establish a diagnosis.

Abbreviations

%: percent

kg: kilogram

HE: haematoxylin and eosin

PCV2: porcine circovirus type 2

Declarations

Acknowledgements

The authors would like to acknowledge the support of the herd veterinarian and the farmer.

Funding

Not applicable

Availability of data and materials

Data sharing was not applicable to this article, as no datasets were generated or analysed during the current study.

Author's contribution

AWS and LJ supervised the post-mortem examination and drafted the manuscript. AWS and GPM developed the diagnosis and summarized the results. MNL and NG performed the histology staining, and HM helped in interpreting the results. GPM supervised the project. All authors read and approved the manuscript.

Ethics approval and consent to participate

The present case report does not include experimental data. The investigations were performed as routine diagnostics. Therefore, approval of an animal ethics committee was not necessary.

Consent for publication

Consent for publication was received from the herd veterinarian and the farmer.

Competing interest

None

References

1. Delsart M, Pol F, Dufour B, Rose N, Fablet C. Pig farming in alternative systems: strengths and challenges in terms of animal welfare, biosecurity, animal health and pork safety. Agriculture 2020; doi:10/3390/agriculture10070261.
2. Lahrmann HK, Bremermann N, Kaufmann O, Dahms S. Health, growing performance and meat quality of pigs in indoor and outdoor housing–a controlled field trial. Dtsch Tierarztl Wochenschr. 2004;111:205–8.
3. Roepstorff A, Mejer H, Nejsum P, Thamsborg SM. Helminth parasites in pigs: new challenges in pig production and current research highlights. Vet Parasitol. 2011;180:72–81.
4. Roguet C, Neumeister D, Magdelaine P, Dockes AC. Les débats de société sur l'élevage au sein de l'Union européenne: thèmes, arguments et modes d'action des parties prenantes, conséquences sur les modes d'élevage. Journées de la Recherche Porcine. 2017;49:307–12.
5. Park HS, Min B, Oh SH. Research trends in outdoor pig production — A review. Asian-Australas J Anim Sci. 2017;30:1207–14.

6. Roinsard A: Valorisation des ressources fourragères par les porcins. In: Bordes A, Calvar C, Maupertuis F, Alibert L, Ferchaud S, Uzereau A, Roinsard A, Carriere J. Alimentation des porcins en agriculture biologique, eds ITAB, IBB, Chambre d'agriculture des pays de loire, IFIP, 2014, p. 29–33.
7. Vetter J. A biological hazard of our age: bracken fern [Pteridium aquilinum (L.) Kuhn]–a review. Acta Vet Hung. 2009;57:183–96.
8. Cortinovis C, Caloni F. Epidemiology of intoxication of domestic animals by plants in Europe. Vet J Lond Engl. 2013;197:163–8.
9. Scala C, Ortiz K, Catinaud J, Lemberger K. Hematuria and urinary bladder lesions compatible with bracken fern (*Pteridium aquilinum*) intoxication in captive fallow deer (*Dama dama*). J Zoo Wildl Med Off Publ Am Assoc Zoo Vet. 2014;45:380–5.
10. Stegelmeier BL. Overview of Bracken Fern Poisoning - Toxicology. Merck Veterinary Manual 2014. https://www.merckvetmanual.com/toxicology/bracken-fern-poisoning/overview-of-bracken-fern-poisoning. Accessed 03 July2020.
11. Wright B. L'intoxication des chevaux par la fougère d'aigle. http://www.omafra.gov.on.ca/french/livestock/horses/facts/09-050.htm. Accessed 03 July 2020.
12. Priymenko N. Végétox' root. http://www.vegetox.envt.fr/. Accessed 02 July 2020.
13. Gallet S. Le contrôle de la fougère-aigle, 2002. http://www.inforet.org/IMG/pdf/fougere.pdf. Accessed 03 July 2020.
14. Banrie. Bracken poisoning in pigs, 2013. The pig site. https://thepigsite.com/articles/bracken-poisoning-in-pigs. Accessed 08 September 2020.
15. Edwards BL. Poisoning by *Pteridium aquilinum* in pregnant sows. Vet Rec. 1983;112(19):459–60. doi:10.1136/vr.112.19.459.
16. Harwood DG, Palmer NM, Wessels ME, Woodger NG. Suspected bracken poisoning in pigs. Vet Rec. 2007;160(26):914–5. doi:10.1136/vr.160.26.914-c.
17. Evans A, Humphreys DJ, Goulden L, Thomas AJ, Evans WC. Effects of bracken rhizomes on the pig. J Comp Path. 1963;73:229–43.
18. Maxie G. (2015). Jubb, Kennedy & Palmer's Pathology of Domestic Animals-E-Book: Volume 2 (Vol. 2). Elsevier Health Sciences.
19. Follis RH, Miller MH, Wintrobe MM, Stein HJ. Development of Myocardial Necrosis and Absence of Nerve Degeneration in Thiamine Deficiency in Pigs. Am J Pathol. 1943;19:341–57.
20. Collongues JL. En Hautes-Pyrénées les sangliers ont occasionné 40 fois plus de dégâts aux cultures. *La Dépêche*, 21/08/2019. https://www.ladepeche.fr/2019/08/21/en-hautes-pyrenees-les-sangliers-ont-occasionne-40-fois-plus-de-degats-aux-cultures,8370150.php. Accessed 27 July 2020.
21. Hannah T, Michaud H. Contrôler le développement de la fougère aigle. Espaces naturels. 2006;14:30–1.
22. Jurado C, Martinez-avilez M, De la Torre A, Stukeli M, De Carvalho Ferreira HC, Cerioli M, Sanchez-Vizcaino JM, Bellini S. Relevant Measures to Prevent the Spread of African Swine Fever in the

European Union Domestic Pig Sector. Front Vet Sci. 2018;5:77.

23. Gagnon S. Essai et expérimentation sur la pollinisation et la réduction des herbicides dans la production du bleuet nain au Saguenay–Lac-Saint-Jean, 2009. https://www.agrireseau.net/petitsfruits/documents/75857/essai-et-experimentation-sur-la-pollinisation-et-la-reduction-des-herbicides-dans-la-production-de-bleuet-nain-au-saguenay. Accessed 03 July 2020.
24. Gama A. Utilisation des herbicides en forêt et gestion durable. Quae, 2006.

Figures

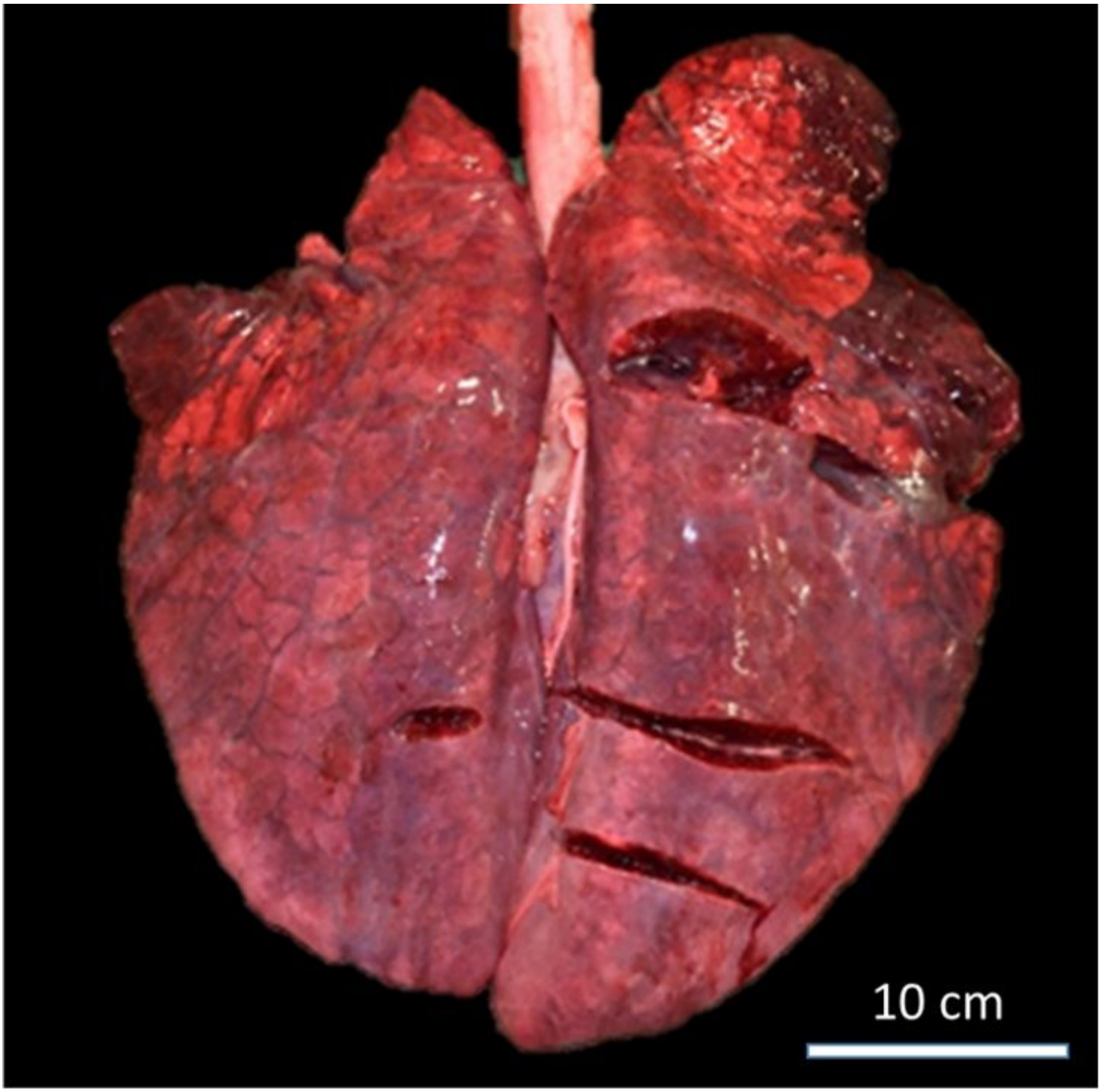

Figure 1

Lung with moderate cranio-ventral consolidation and marked diffuse interlobular oedema

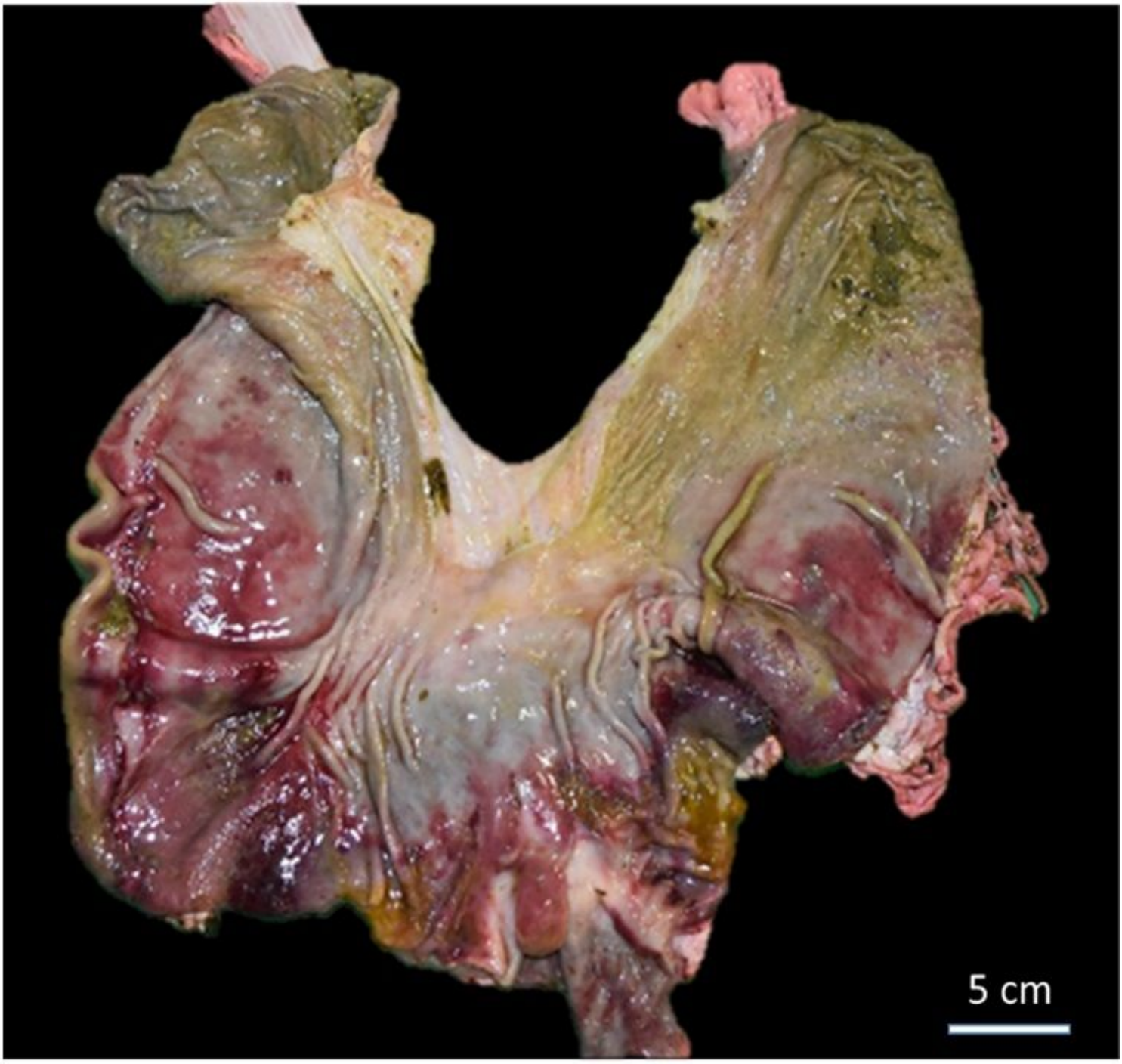

Figure 2

Stomach with extensive fundic congestion.

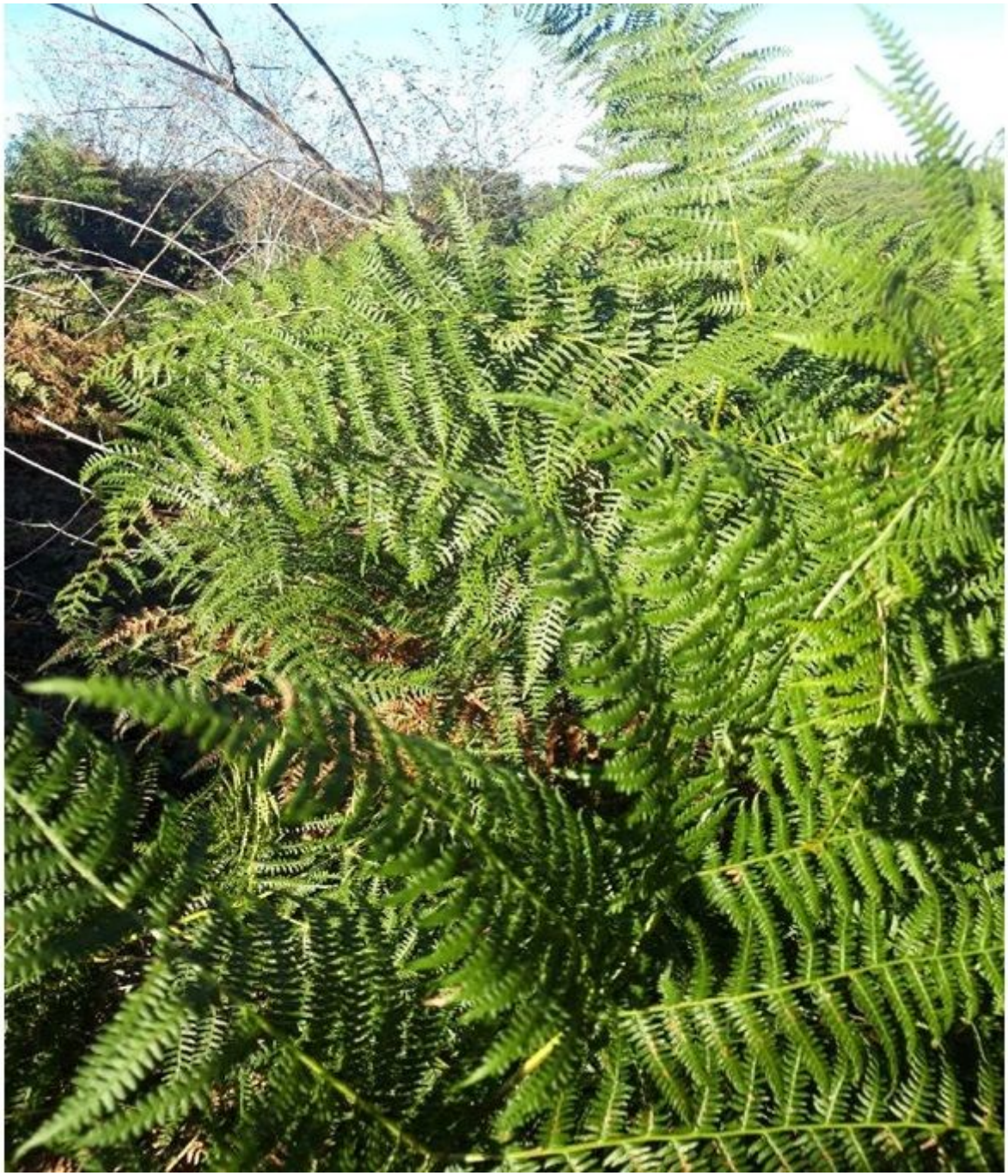

Figure 3

Great eagle fern (Pteridium aquilinum).

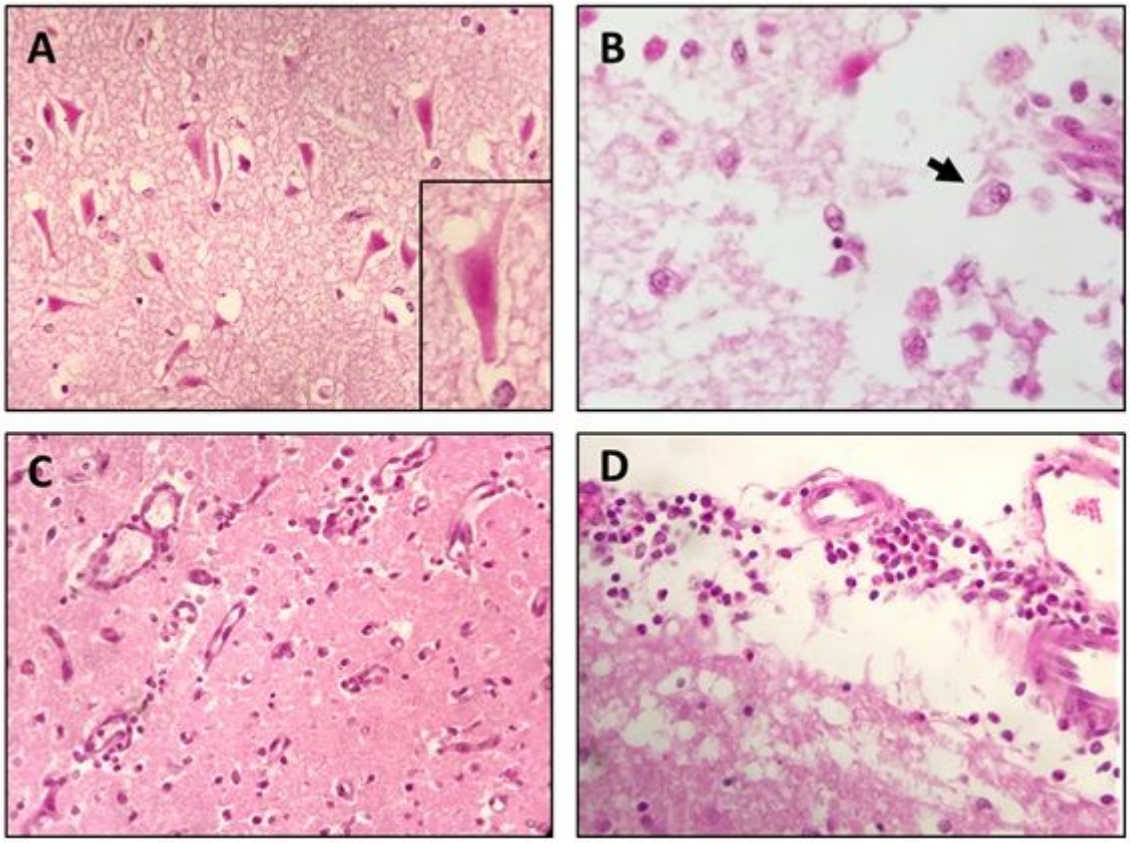

Figure 4

Brain histological pictures of the male pig. A. Cerebrocortical acidophilic neuronal necrosis (x400). B. Gitter cells (arrow) in the spongiotic area (x400). C. Endothelial activation with perivascular leukocytic infiltration (x200). D. Leptomeningeal leukocytic infiltration, including eosinophils (HE, x400).

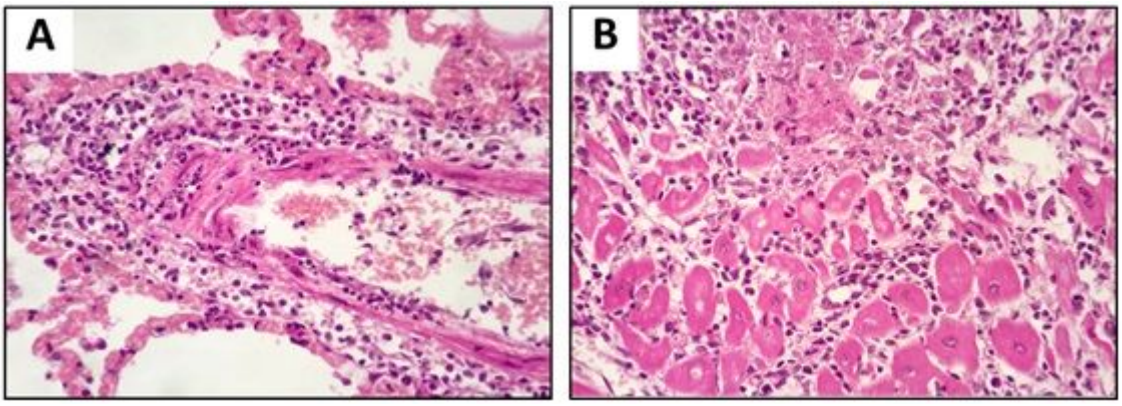

Figure 5

Lung and heart histological pictures of the female pig. A: Lung with necrotizing leukocytoclastic vasculitis. B: Heart with polyphasic necrotizing and leukocytic myocarditis (HE, x400).