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# CASE REPORT: AORTIC THROMBOEMBOLISM RELATED TO POLYCYSTIC KIDNEY DISEASE IN A CAT

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***Abstract***

*A cross-breed female cat was presented with clinical signs of abdominal pain, paralysis of both hind limbs, tachycardia, tachypnea and bilateral absence of the femoral pulse. Necropsy confirmed an aortic thrombus at 2 cm above the adrenal glands, the heart with left atrial dilation and hypertrophied left ventricle, multiple bilateral kidney cysts and ectopic thyroid tissue. Histopathological examination reveals the kidney with multiple cysts, diffuse loss of renal structural details without inflammatory cells, hypertrophic cardiomyopathy, and no ectopic thyroid tissue changes. This communication describes a case of aortic thromboembolism associated with hypertrophic cardiomyopathy due to autosomal dominant polycystic kidney disease and ectopic thyroid tissue.*

***Key words****: aortic thromboembolism, hypertrophic cardiomyopathy, cardiorenal syndrome, polycystic kidney.*

## INTRODUCTION

In cats, arterial thromboembolism (ATE) is a common clinical syndrome. It is caused by the unexpected migration of a thrombus or a thrombus fragment from the left atrium (LA) (Vezzosi et al., 2020).

This syndrome was reported for the first time by Collet in 1930 (Smith & Tobias, 2004; Tomaiuolo, Litvinov, Weisel, & Stalker, 2020).

It is, most often, a consequence of the heart diseases, such as hypertrophic cardiomyopathy (HCM), dilated cardiomyopathy (DCM), restrictive cardiomyopathy (CMR), unclassified ischemic cardiomyopathy (Molina, Estrada G, Salas, & González, 2012; Schoeman, 1999), chronic myocardial infarction (Tsujino et al., 2005).

In 50% of cats with HCM, ATE has been reported (Michaud, Herbert, Elkins, & Gozalo, 2017). Generally, LA is the source of emboli.

This statement is supported by the fact that 21% of cats with HCM examined postmortem have left atrial thrombi.

Moreover, ultrasound examination in cats with heart diseases showed that intracardiac thrombi are quite commonly identified (Hogan, 2017; Smith & Tobias, 2004).

In cats without heart damage, ATE had been, sometimes, associated with neoplasms or hyperthyroidism. Neoplasia, especially lung carcinoma, is a risk factor for ATE, although in this situation there is a tumour embolism and less thromboembolism. In cats with thyroid disease and thyrotoxic cardiomyopathy ATE has been reported as being associated (Silva et al., 2016; Smith & Tobias, 2004). Dilated LA and intraatrial blood stasis are the main risk factors for thrombus development. In cats, the increasing of LA volume occurs most often in HCM, being concurrent with concentric hypertrophy of the left ventricle. Although it is known that in humans the improper functioning of the affected left ventricle may be a predictor of the onset of ATE than the increase in the volume of AS, in cats the systolic function has not been evaluated as a risk factor for ATE (Acierno et al., 2020; Baty et al., 2001). Male are more affected than female, with a male:female ratio of 2.5: 1, due to their predisposition to develop HCM (O'Dwyer, 2015; Tomaiuolo et al., 2020).

In the present case report we describe the clinical, pathological and histological features in a cat with aortic thromboembolism associated with hypertrophic cardiomyopathy

due to autosomal dominant polycystic kidney disease (PKD) and ectopic thyroid tissue. No other cases of cats that present these types of associated injuries were found in studied databases.

## MATERIALS AND METHODS

A mixed-breed long-haired cat, female, aged seven years, weighing 3.4 kg, was presented with paraplegia, tachypnea, cyanotic mucous membranes, hypersalivation, vomiting, vocalizations. The cat was under medical observation for hypertrophic cardiomyopathy with left atrial dilation, severe mitral regurgitation and pleural effusion. Before recommending the therapeutic protocol, a cytopathological examination of the thoracic fluid was performed after previuos centrifugation for 5 minutes at 1500 rpm. Almost all of the surnatant was discarded and the obtained cell pellet was resuspended with the small amount of surnatant left. Cytological impression smears of the resuspended centrifuged pellet cell sample were performed, air-dried and stained with May Grunwald Giemsa (MGG) for light microscopic evaluation. Due to the rapid worsening of the clinical condition, the cat was euthanized. Post-mortem examination was performed and samples were collected for histopathological examination, from all modified organs. The selected specimens were fixed in 10% neutral buffered formalin, processed routinely, embedded in paraffin wax, cut into 3 µm sections, and stained with hematoxylin and eosin (HE).

## RESULTS AND DISCUSSIONS

At the clinical examination were observed abdominal pain, paralysis of both hind limbs, tachycardia, and tachypnea. Lack of superficial and deep sensitivity, absence of femoral pulse, hypothermia, and cyanosis of the footpads was observed at the level of the hind limbs. The body temperature was 38°C. At the neurological evaluation, depression was present without neural reflexes. The thoracic fluid revealed grossly a pinkish, clear, watery appearance and the microscopical analysis showed inflammatory cells in approximately equal proportions: neutrophils, active macrophages, lymphocytes, plasma cells and eosinophils. A suspicion of modified transudate with chronic inflammatory process was suspected. The diagnosis of lumboaortic thromboembolism was established by the clinical investigation. Supportive therapy was instituted with anticoagulant, diuretic, steroidal anti-inflammatory drugs, antihypertensive drugs and fluids. Antibiotic was given to prevent secondary infections. The post-mortem examination confirmed the bilateral hydrothorax, and approximately 150 ml of pinkish fluid was evacuated. The lungs presented, bilaterally, diffuse oedema and congestion, a suggestive aspect of cardiogenic oedema. At the level of the dorsal, anterior mediastinum, an ectopic thyroid tissue of approximately 3 mm in diameter was found (Figure 1).

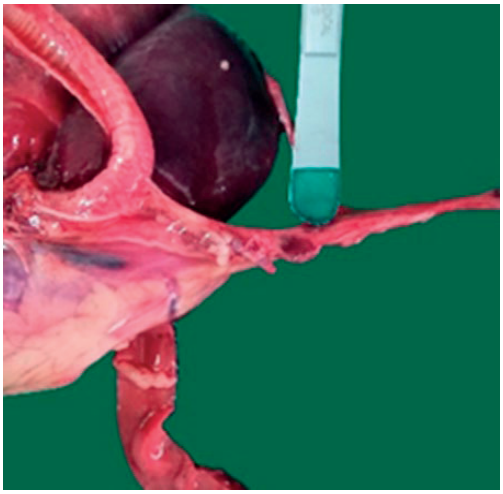

Figure 1. Ectopic thyroid tissue at the anterior mediastinum of the cat: the ectopic tissue is 3 mm diameter, dark red

0.8 ml of pinkish fluid were extracted from the pericardial sac, and that fits within the average values of 0.25 ± 0.15 ml/kg (Davies & Forrester, 1996; Holt, 1970). The left atrial dilation (Figures 2 and 3.), the thickening of the free left myocardial wall and the interventricular septum (Figure 4) were confirmed. The free left ventricular wall measured 9.82 mm, and the interventricular septum was 7.75 mm. Those dimensions are correspondent for HCM. Pathological

hypertrophy is defined by thicknesses of the left free ventricular wall and the interventricular septum ≥6 mm. At least one of these two myocardial segments must exceed the value mentioned above to establish the HCM diagnosis (Baty et al., 2001; Gouni et al., 2008) The values obtained following the ratio g heart/kg body was 0.88%, the cat heart having 0.46% ("Appendix 1: Normal Organ Weights - Percentage Body Weight", 2017).

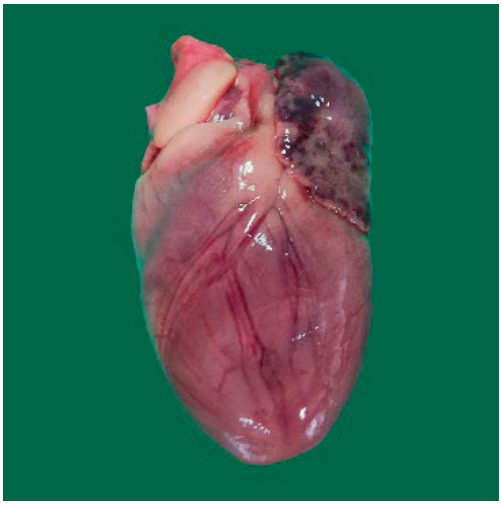

Figure 2. Gross features of the heart with HCM: LA dilation and diffusely enlargement heart volume

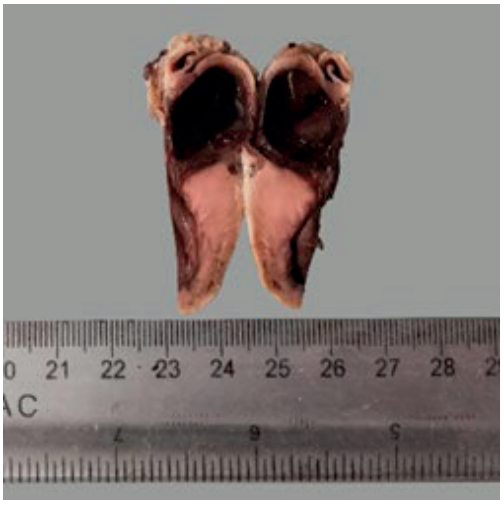


Figure 3. Gross left atrial chamber section view of the heart with HCM: dilation of left atrium

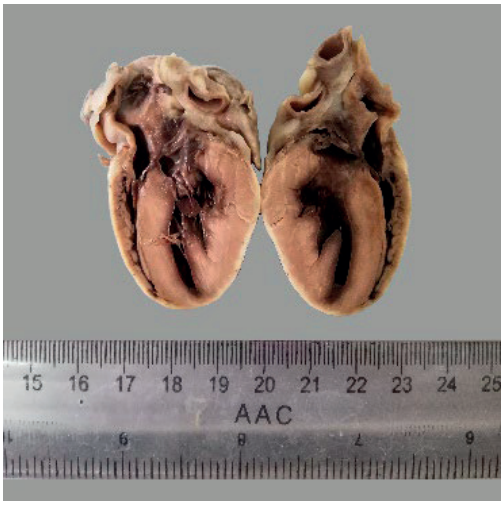


Figure 4. Four-chamber cross-section view of the heart with HCM in cat: disproportion between ventricular septum and free left ventricular wall hypertrophy with associated reduction in left ventricular cavity size

The liver has the appearance of passive congestion and areas of multifocal, subcapsular haemorrhage. The kidneys bilaterally showed numerous cysts and areas of fibrosis. Those modifications replaced more than half of the renal parenchyma (Figure 5). Multifocal necrosis of the renal papillae was also noted. The renal cortico-medullary area showed congestion, suggestive of prerenal ischemia. Yellow, transparent liquid leaked when the cysts were sectioned.

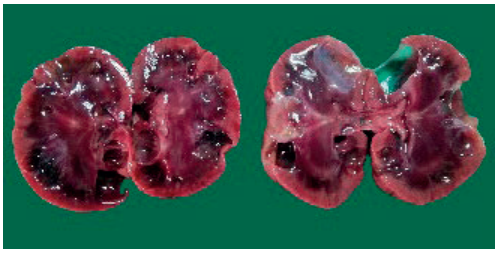

Figure 5. Longitudinal section of polycystic dysplastic kidneys: irregular external perimeter; the parenchyma was replaced by multiple cysts filled with fluid, varying in size; corticomedullary congestion suggestive of hypoperfusion

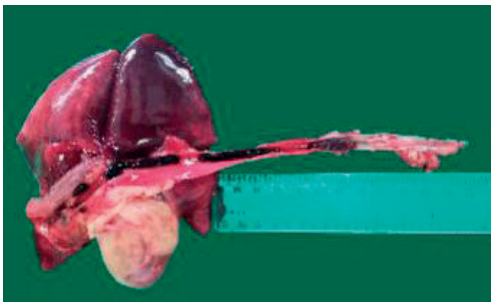

Figure 6. Longitudinal section of the aorta with thrombus, post-mortem prethrombotic haemoglobin imbibition, suggestive of obliterating thrombosis

The abdominal aorta showed a dilated reddish colour shape, at 2 cm above the adrenal glands (Figure 6). After dissection, a 2 cm long dark red, dry, friable, granular, obliterating, adherent to endothelium thrombus. The tail of the thrombus is extended downstream from the area of vascular attachment, in this case showed the cardiac origin. The cause of the complete obstruction of the lumen was found, confirming the clinical diagnosis of aortic thromboembolism (Figure 7).

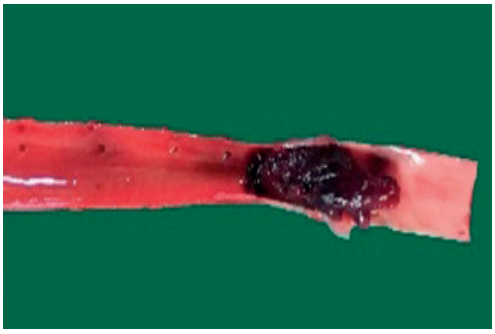

Figure 7. Longitudinal section of the aorta with thrombus obliteration: solid dark red mass, rough, matte, adherent to wall

Histopathological examination of the obliterated aortic segment revealed a massive accumulation of layers of fibrin, erythrocytes and organized cell debris, also an altered endothelium with partially denuded basement membrane (Figure 8).

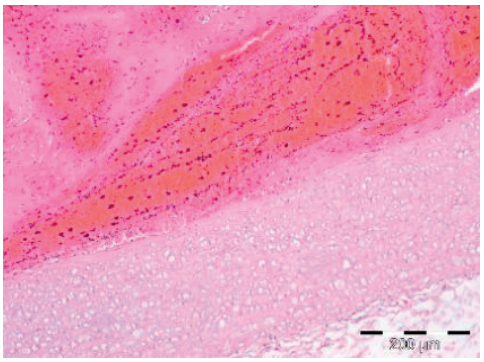

Figure 8. Thrombus details: layers of fibrin with erythrocytes; continuous adhesion to the intima of the aorta, ob 100, HE

Renal parenchyma presented multiple bilateral cysts with homogeneous liquid, with diffuse loss of renal structural details. The cysts were lined by a single layer of epithelial cells. (Figure 9).

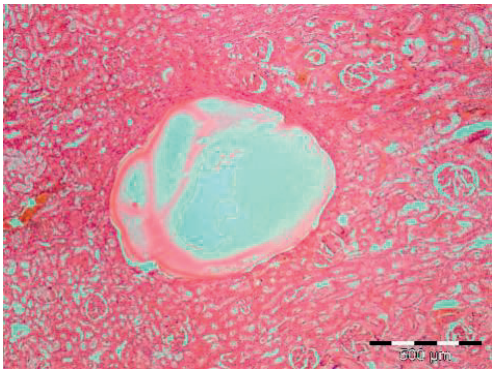

Figure 9. Renal cortex: large simple cyst lined by flattened cells, that contain granular eosinophilic material (probably protein). The glomeruli, are compressed, ob 100, HE

Disorganisation and hypertrophy of cardiomyocytes, as well as interstitial fibrosis, were revealed in the heart (Figure 10). Ectopic thyroid tissue did not show any changes in histopathology (Figure 11). Spherical and oval follicles filled with eosinophilic colloid could be observed in different functional stages, bordered by simple cuboidal epithelial tissue. Restricted areas of clustered cells with vesicular nuclei, small nucleoli and eosinophilic cytoplasm can be observed. No parathyroid tissue was observed.

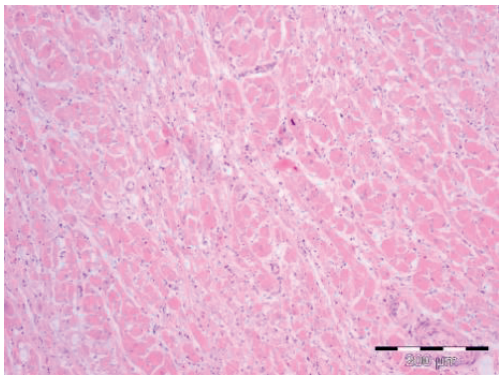

Figure 10. Ventricular septum myocardium with HCM: myocyte hypertrophy and disarray characterized by the interweaving of myofibers, associated with proliferation of the interstitial fibrous connective tissue (fibrosis), ob 100, HE

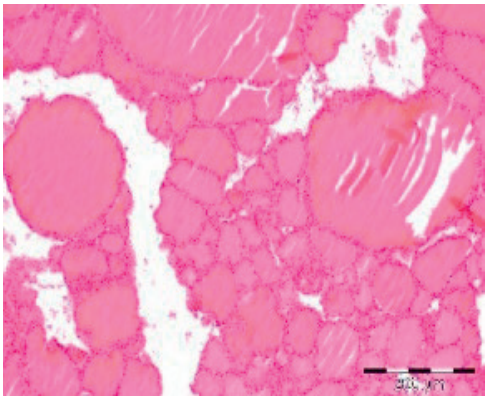

Figure 11. Ectopic thyroid tissue: follicles in different functional stages, bordered by simple cuboidal epithelial tissue, filled with eosinophilic colloid, ob 100, HE

The diagnosis of aortic embolism/aortic thromboembolism or natural aortic occlusion was made based on clinical signs shown by cat (Carter, amp, & Van Heerden, 1994; Silva et al., 2016). These clinical signs associated with ATE were due to acute ischemia of the tissue served by the occluded artery. The vocalisations of the cat were attributed to pain, the cats responding to pain through self-mutilation and vocalisations (Smith & Tobias, 2004). It is a well-known fact that cats with heart disease have high-risk condition for thrombus formation. Risk factors include changes in blood flow, endothelial cell lesions and hypercoagulopathy. Those modifications are known as Virchow's triad (Molina et al., 2012; Schoeman, 1999). Knowing that the cat was antemortem diagnosed with HCM and LA dilation, it may be considered that formation of the thrombus that led to the aortic embolism took place at the level of LA (Vezzosi et al., 2020). It is known that maximum speed of the blood in the left atrial auricle is lower in cats with cardiomyopathy (0.31 m/sec) compared to healthy cats (0.46 m/sec). In cats with concomitant left atrial thrombus or ATE, the maximum known velocity of blood flow is 0.14 m/sec, suggesting that stasis may indeed contribute to left atrial thrombus formation. These events are classified as cardiogenic because the source of the thrombotic material comes from a cardiac chamber, usually from LA (BUTLER, 1971; Hogan, 2017; Silva et al., 2016). As no endothelial lesions were observed at the AS level, and the cat was undergoing anti-platelet therapy, we can consider that the thrombus was formed following blood stasis at this level. The emboli are dislocated, reaching the aorta or one of its major branches. The blockage will occur when size exceeds the diameter of the vessel, thus obliterating the blood circulation. Subsequent consequence involves the absence of blood supply of one or more limbs. This cat had only aortic thromboembolism. It is known that thromboembolism can rarely affect the cerebral, renal and mesenteric arteries (Smith & Tobias, 2004; Tomaiuolo et al., 2020),(Hogan, 2017). The infarction of the area served by the blocked blood vessel is not exclusively due to the loss of blood flow. Experimental models have shown that when the activation of the coagulation system occurs, similar to the situation when an embolus obliterates the terminal aorta, the collateral circulatory network builds up in vasoconstriction, probably due to the release of vasoactive substances. Experimental studies have shown that complete ligation of the terminal abdominal aorta does not impede arterial flow to the pelvic limbs, nor result in clinical signs, as there is a collateral circulatory network in the vertebral and hepatic systems that maintain arterial flow around the ligature. Aortic ligation does not affect locomotion either, paralysis depending on the presence of blood thrombus in the aorta (BUTLER, 1971; Hogan, 2017; Săvulescu-Fiedler, Gherghiceanu, Militaru, Brăslașu, & Bruckner, 2014; Silva et al., 2016).

Corroborating the anamnesis with the result of clinical and necropsy investigations of this case was concluded that ATE was the consequence of HCM. It is known that certain breeds have a high risk to develop HCM. In Maine Coon and Ragdoll breeds, HCM is an inherited autosomal dominant condition. It is caused by a mutation of the gene, which is encoding cardiac myosin-binding protein C (MYBPC3). In these breeds, genetic testing for reproductive acceptance is recommended (Juliana Mariotti Guerra et al., 2020; Luis Fuentes et al., 2020).

In common breed cats cases of HCM have been reported, but that has not previously been diagnosed with an infectious or metabolic disease leading to this disease. It has not been shown that the disease was genetic. (Baty et al., 2001) The cat from the presented case does not belong to the breeds prone to developing HCM, but it has not been possible to determine if being a cross-breed of one of them. HCM, the most common cardiomyopathic phenotype in cats, is characterised by thickening of the interventricular septum and/or left ventricular wall, diffuse or asymmetric, without dilation of the right ventricular chamber. HCM is a condition with a high risk of morbidity and mortality, featured by lesions corresponding to congestive heart failure and consecutive ATE, being the most common causes of clinical signs of heart disease and sudden death in this species (Juliana Mariotti Guerra et al., 2020; Luis Fuentes et al., 2020).

The association between PKD and HCM, also observed in this case, has rarely been described in cats. In humans, 89% of patients with autosomal dominant polycystic kidney disease (ADPKD) who died of the cardiac disease had, as in the present case, left ventricular wall hypertrophy (LVH). In humans, 89% of patients with ADPKD who died of the cardiac disease had LVH, as in the presented case. Expansion of renal cysts and local hypoperfusion activates the intrarenal renin-angiotensin system, causing hypertension, while increased intracardiac pressure stimulates myocardial remodelling (Juliana Mariotti Guerra et al., 2020). The increase in blood pressure is also mediated by the increase in cardiac output. The result is compensatory left ventricular hypertrophy (Luis Fuentes et al., 2020)·(Sim Lam et al., 2020; Spencer, Wheeler-Jones, & Elliott, 2020), injury reported in this case as well. The interrelationship between heart and kidney diseases is quite common in cats; the association between the two diseases having a poor prognosis (Bongartz et al., 2012). In the presented case, it could be observed that the necrotic pressure caused by renal cysts of different sizes caused the denaturation of over 60% of the normal renal parenchyma. Loss of renal architecture has been associated with impaired renal function. Polycystic kidney disease (PKD) is one of the most common inherited disorders of the cat. It is common in the Persian breed or related breeds, and inconsistent in their mixed breed. An inherited dominant autosomal mutation caused PKD, and the animals may be asymptomatic for years. The cause of PKD is a mutation in the PKD1 gene. It has been identified as heterozygous in 48 different breeds of cats including Persians, Siamese and short-haired cats. Grossly, PKD is characterised by the presence of one or more fluid-filled cysts, of various sizes, in the cortex, medulla, or both renal areas. They can occur in a single kidney or bilaterally. Renal cysts tend to multiply in number and increase in size over time as the animal ages. This leads to progressive deterioration and necrosis of kidney tissue due to pressure, causing chronic renal failure and eventually cat death. The disease is usually subclinical until middle age or older. The diagnosis of PKD can also be established by genetic tests for the mutation of the PKD1 gene (Bilgen et al., 2020; Guerra, Cardoso, Daniel, Onuchic, & Cogliati, 2020; Sim Lam et al., 2020; Yu et al., 2019).

Knowing that PKD is a genetic condition with the potential to cause HCM, it was concluded that, in this case, HCM is secondary to chronic kidney disease induced by PKD. This lesion is known as being responsible for cardio-renal/reno-cardiac syndrome. Disorders of the heart and kidneys, manifested by acute or chronic dysfunction of one organ, could induce acute or chronic dysfunction in the other organ. The presented case is included in type 4 of this syndrome being known as five types in total. Type four represents chronic renocardiac syndrome consisting of chronic kidney disease, which causes injury, illness and/or heart dysfunction (Damman, Voors, Navis, van Veldhuisen, & Hillege, 2011; Orvalho &

Cowgill, 2017; Pouchelon et al., 2015). In the presented case, chronic kidney disease led to HCM.

It was concluded, at the histopathological diagnosis, there are no changes in the ectopic thyroid tissue, so it was possible to exclude the hyperthyroidism given by hypersecretion of thyroid hormones at this level (Peterson, 2012). Unfortunately, no blood tests were performed antemortem to confirm the hyperthyroidism. Corroborating the histological result with the body fur and muscles well-maintained appearance, it has been chosen to exclude this condition. It is known that hyperthyroidism leads to HCM in cats (Luis Fuentes et al., 2020).

It is known that ectopic thyroid tissue rarely undergoes pathological transformations, such as hyperplasia, adenomas or adenocarcinomas. Accessory thyroid tissue can occur anywhere from the larynx to the diaphragm. In cats, calcitonin-containing C cells appear at 38 days of gestation as single scattered cells and migrate into interfollicular spaces on the 50th day of gestation. The examined tissue showed the presence of C cells so we could conclude that ectopia could have appeared after the 38th day of gestation (Knowles, Uhl, Blas-Machado, & Butler, 2010; Patnaik, Peterson, & Hidgon, 2000).

## CONCLUSIONS

The case showed that PKD leads to chronic kidney disease, renal-cardiac syndrome and hypertension. In addition, the onset of left atrial dilation is a consequence of hypertension, producing subsequent disturbances of blood circulation and blood flow deceleration. Thus, predisposing factors for ATE are created, being considered a syndrome with a severe prognosis.

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