Concurrent juvenile primary acquired hypothyroidism in a young Maltese with glycogen storage disease – a case report

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Received October 10, 2025
Accepted March 2, 2026

Abstract

A 1-year-old Maltese dog weighing 1.6 kg was referred for evaluation of a persistent comatose mentation that developed within one day following treatments for seizures and hypoglycaemia. At presentation, the dog exhibited signs of mild dehydration (approximately 5%), pinpoint pupils, and hypothermia. Initial differential diagnoses included portosystemic shunt, glycogen storage disease (GSD), and congenital hypothyroidism based on clinical presentation and history. The bile acid and ammonia concentrations were within the reference intervals, thereby ruling out a portosystemic shunt. A thyroid panel was conducted, with the results revealing low serum total thyroxine concentrations with elevated thyroid-stimulating hormone concentrations, and subsequent thyroid-stimulating hormone stimulation test confirmed hypothyroidism. Euthanasia was performed at the owner's request. Histopathologic examination revealed idiopathic thyroid gland atrophy, diffuse vacuolar degeneration with glycogen accumulation in the liver and kidneys. The diagnosis was GSD type Ia and concurrent juvenile primary acquired hypothyroidism. This case highlights the importance of a comprehensive evaluation for concurrent congenital and acquired endocrine disorders in young dogs presenting with neurological and metabolic abnormalities.

Canine, hyperlipidaemia, hypoglycaemia, seizure, thyroxine

Glycogen storage disease (GSD) type I is an inherited disorder resulting from a deficiency in specific enzymes within the glycogen metabolism pathway. It comprises two major subtypes, GSD Ia and GSD Ib (Kishnani et al. 2019). GSD type Ia, also known as von Gierke disease, arises from a deficiency in glucose 6-phosphatase-α, a crucial enzyme involved in gluconeogenesis and glycogenolysis. Notably, the Maltese breed exhibits a predisposition to GSD type Ia, which often expresses the carrier (heterozygous) state for this naturally occurring mutation (Brix et al. 1995; Specht et al. 2011). This enzyme deficiency impairs the body's ability to elevate blood glucose concentrations in response to positive glucoregulatory stimuli, resulting in fasting hypoglycaemia and the accumulation of glycogen and lipid deposits, primarily within hepatic and renal tissues.

In humans, concurrent hypothyroidism has been reported in GSD type I, but there is no evidence of thyroid abnormalities in other classes of GSDs (Rossi et al. 2024). Hypothyroidism and GSD type I have similar clinical characteristics, such as retarded growth, hyperlipidaemia, and mental depression. Therefore, a clinical diagnosis of hypothyroidism could be missed in some patients with GSD type I.

This case report is the first to describe concurrent juvenile primary acquired hypothyroidism in a Maltese dog with GSD type Ia.

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Case description

A 1-year-old castrated male Maltese dog was referred for investigation and treatment of coma following generalized seizure and hypoglycaemia (blood glucose concentration = 40 mg/dl; reference interval [RI], 65–118 mg/dl) at the referring animal hospital. According to the owner, the dog had exhibited poor growth and lethargy since early life; however, acute neurological deterioration – including seizures and altered mentation – developed within two days prior to referral. At the referring hospital, the patient received an intravenous infusion of 1 ml/kg of 50% dextrose over 30 min, followed by crystalloid fluid therapy at a shock dose. Despite these treatments, the patient remained comatose. At the time of presentation at our hospital, the dog exhibited hypothermia (36 °C) without shivering, hypotension (systolic blood pressure measured by Doppler method = 115 mmHg), mild dehydration (approximately 5%), pinpoint pupils, and a comatose state. Additionally, the dog was poorly grown, with a body condition score of 2/9, weighing 1.6 kg, with no history of toxin or foreign body ingestion. Other physical examination findings were unremarkable. Considering the patient's clinical presentation and history, portosystemic shunt (PSS), GSD, and congenital hypothyroidism were the most likely differential diagnoses.

Haematology and serum biochemistry tests were performed using samples collected from the cephalic vein. Haematological indicators were obtained using the IDEXX ProCyte Dx analyser (IDEXX Laboratories, Inc., Westbrook, Maine, USA). Serum biochemical profiles were analysed with a biochemical analyser (Hitachi 7020, Hitachi High-Technologies Co., Tokyo, Japan), and venous blood gas analysis was performed using an ABL9 blood gas analyser (Radiometer, Copenhagen, Denmark). The reference intervals presented in this manuscript were based on those provided by the respective analysers. A summary of the patient's haematology, serum biochemistry, and venous blood gas results is presented in Table 1. Notably, fasting bile acid and ammonia concentrations were within normal limits, making PSS less likely. Venous blood gas analysis revealed marked hyperchloraemic metabolic acidosis. The elevated partial pressure of carbon dioxide indicated impaired respiratory compensation, likely secondary to the patient's comatose state and associated hypoventilation. Furthermore, a portable ketometer (FreeStyle Optium Neo, Abbott, IL, USA) revealed normal ketone concentrations.

Hormonal assessments of the hypothalamic–pituitary-thyroid axis were performed due to poor growth and marked hyperlipidaemia. Serum concentration of serum total thyroxine (T4) and thyroid-stimulating hormone (TSH) concentrations were measured using an enzyme immunoassay analyser (Immulite 2000; Siemens Healthcare Diagnostics, USA). The results revealed low serum total thyroxine (T4) (0.5 μg/dl; RI = 1.0–4.0 μg/dl), low free T4 (< 0.3 ng/dl; RI, 0.6–3.7 ng/dl), and increased thyroid-stimulating hormone (TSH) concentrations (1.10 ng/ml; RI = 0.05–0.42 ng/ml). A TSH stimulation test was performed to differentiate euthyroid sick syndrome. Total thyroxine concentrations were undetectable before and 6 h after stimulation with recombinant human TSH (Thyrogen; Zenzyme, Cambridge, MA, USA) (< 0.5 μg/dl, both). Thyroglobulin autoantibodies (TgAA) were not detected, leading to the diagnosis of primary hypothyroidism.

Thoracic radiography revealed no abnormalities, whereas abdominal radiography showed mildly decreased overall serosal detail in the abdominal cavity, likely attributable to the patient's markedly lean body condition, as well as a fluid-filled, moderately dilated stomach. During hospitalization, the dog received intravenous fluid therapy with PlasmaLyte A (Baxter, Gurgaon, India) at a rate of 10 ml/kg/h, along with continuous infusion of sodium bicarbonate at a dose of 1 mEq/kg over 2 h to correct metabolic acidosis and dehydration. Additionally, maropitant citrate was administered at a dosage of 1 mg/kg i.v. q24h and esomeprazole at a dosage of 1 mg/kg i.v. q12h as the patient was considered at risk of gastrointestinal

Table 1. Clinicopathology data included in the present study.

Indicator	Result	Reference interval
Complete blood count		
Total white blood cell count	7.10 ×10³/µl	5.05–16.76 ×10³/µl
Segmented neutrophils	5.93 ×10³/µl (83.5%)	2.95–11.64 ×10³/µl
Lymphocytes	1.02 ×10³/µl (14.4%)	1.05–5.10 ×10³/µl
Monocytes	0.12 ×10³/µl (1.7%)	0.16–1.12 ×10³/µl
Eosinophils	0.01 ×10³/µl (0.1%)	0.06–1.23 ×10³/µl
Basophils	0.02 ×10³/µl (0.3%)	0.00–0.10 ×10³/µl
Serum biochemistry		
Total cholesterol	333 mg/dl	135–270 mg/dl
Triglycerides	1 681 mg/dl	21–116 mg/dl
Blood glucose	89 mg/dl	65–118 mg/dl
Lactic acid	1.29 mmol/l	0.5–2.5 mmol/l
Creatine kinase	12 810 IU/l	42–530 IU/l
Aspartate aminotransferase	173 IU/l	23–66 IU/l
Total protein	4.9 g/dl	5.4–7.1 g/dl
Albumin	2.5 g/dl	2.6–3.3 g/dl
Blood urea nitrogen	110.1 mg/dl	7–25 mg/dl
Creatinine	2.0 mg/dl	0.5–1.5 mg/dl
Fasting bile acid	0 µmol/l	0–25 µmol/l
Ammonia	58 µmol/l	0–98 µmol/l
Venous blood gas		
pH	6.76	7.31–7.46
Bicarbonate	7.1 mmol/l	21–28 mmol/l
Base excess	−32.3 mmol/l	−2–3 mmol/l
Carbon dioxide pressure	53.6 mmHg	27.0–50.0 mmHg
Chloride	121 mmol/l	110–119 mmol/l
Sodium	147 mmol/l	144–151 mmol/l
Potassium	4.11 mmol/l	3.9–5.1 mmol/l
Anion gap	19.1 mmol/l	7–16 mmol/l

ulceration secondary to uraemic toxins and to safeguard against vomiting because the altered level of consciousness was considered high risk for aspiration. The dog exhibited intermittent tonic–clonic seizures and muscle twitching, accompanied by hypoglycaemia and hyperlactataemia. To manage these clinical signs, diazepam (0.5 mg/kg i.v.) was administered to control seizure activity, and hypoglycaemia was corrected with a bolus of 50% dextrose solution followed by a continuous rate infusion (1 ml/h for 6 h) to maintain blood glucose concentrations above 60 mg/dl. At presentation, blood glucose and lactate concentrations were within normal limits, likely due to prior dextrose administration at the referring hospital. However, during hospitalization, the patient developed recurrent hypoglycaemia, with a nadir of 34 mg/dl, and progressive hyperlactataemia reaching 5.4 mmol/l, indicating ongoing metabolic instability despite continuous dextrose supplementation. Despite appropriate correction of hypoglycaemia and other metabolic disturbances, the dog's comatose mentation persisted for over 24 h after admission. Given the persistent coma and poor prognosis, the owner elected euthanasia to prevent further suffering.

With the owner's consent, a histopathological examination was performed on the liver, kidneys, and thyroid glands. Microscopic sections stained with haematoxylin and

eosin revealed severe vacuoles of hepatocytes. Hepatocytes exhibited diffuse expansion characterized by large amounts of lacy to vesicular and occasionally vacuolated cytoplasm, resulting in compression and obstruction of adjacent sinusoidal spaces (Plate IV, Fig. 1). In addition, microscopic examination of kidney tissue revealed diffuse epithelial expansion in proximal renal tubules, with moderate to marked enlargement due to elevated levels of lacy to vesicular and occasionally vacuolated cytoplasm (Plate IV, Fig. 2). Microscopic examination of thyroid sections revealed that approximately 40% of thyroid follicles exhibit small/atrophied, collapsed morphology with the absence of colloid. No inflammatory changes were observed, indicating idiopathic thyroid atrophy (Plate V, Fig. 3).

Periodic acid-Schiff (PAS) staining was performed to detect glycogen in vacuolated hepatocytes. Coarse granules were observed within the cytoplasmic vacuoles and vacuolated nuclei of hepatocytes, confirming the presence of glycogen (Plate V, Fig. 4). Consequently, the dog was diagnosed with GSD type Ia concurrent with juvenile primary acquired hypothyroidism based on clinical observations, thyroid hormone tests, and histopathologic examinations.

Discussion

Glycogen storage diseases present with distinct clinical features, including growth retardation, hypoglycaemia, hepatomegaly, nephromegaly, hyperlipidaemia, hyperuricaemia, and lactic acidaemia (Specht et al. 2011). Initially, the dog showed normal blood glucose and lactate concentrations at our hospital due to the correction of blood glucose concentration at the referring hospital. However, low blood glucose and high lactate concentrations were detected during treatments at our hospital. Unlike other GSD classes, only GSD type Ia and Ib are non-ketotic which was compatible in this case based on the normal β-hydroxybutyrate concentration. Furthermore, the hallmark of GSD-1b is neutropaenia (Ozen 2007). Considering these characteristics, the diagnosis of GSD type Ia was established in the present case based on signalment, clinical signs, and histopathological findings, which revealed excessive glycogen storage within the liver and kidney.

In addition to GSD, the possibility of PSS was considered based on the patient's signalment and clinical signs, which included young age, poor growth, and hypoglycaemia. However, fasting bile acid and ammonia concentrations were found to be within normal limit. Using the upper limit of the laboratory reference intervals for serum fasting bile acid (20 μmol/l), the sensitivity of this test for diagnosing PSS in dogs is reported to be 93% (Ruland et al. 2010). Moreover, combined measurement of fasting bile acid and ammonia concentrations may represent the most specific diagnostic approach for identifying PSS in dogs (van Straten et al. 2015). In this case, the serum fasting bile acid concentration was 0 μmol/l and fasting ammonia concentration was within reference interval, suggesting that the possibility of PSS was very low. In addition, histopathological examination of the liver did not reveal the characteristic pattern associated with PSS, which includes a lack of portal veins, an increased number of arterioles (often tortuous) in the hepatic triads, hepatocyte atrophy with lipogranuloma formation, and occasional sinusoidal dilatation around the portal areas (Sobczak-Filipiak et al. 2019). Therefore, PSS was ruled out in the present case.

Congenital hypothyroidism was also considered based on the patient's age, poor growth, and severe hyperlipidaemia. Although rare in dogs (Feldman and Nelson 2015), congenital hypothyroidism can occur. The exact pathogenesis of this condition in dogs remains unclear, but the proposed mechanisms include central dyshormonogenesis and synthesis failure at the level of the thyroid gland. Central congenital hypothyroidism is typically diagnosed in patients presenting with relevant clinical signs, low T4 levels,

and concurrently low TSH level (Bojanic et al. 2011). In this case, central congenital hypothyroidism was initially excluded due to the increased TSH level.

To better understand the underlying cause of hypothyroidism in this juvenile Maltese, a TgAA assay was performed, yielding a non-detectable result. Additionally, histopathological analysis revealed thyroid follicle atrophy without inflammatory changes. Based on the results of the TgAA assay and histopathological examination, the dog was diagnosed with acquired primary hypothyroidism due to idiopathic thyroid atrophy. Acquired primary hypothyroidism is the most common cause of naturally occurring thyroid failure in adult dogs, accounting for over 95% of cases (Mooney 2011; Feldman and Nelson 2015). The diagnosis of acquired hypothyroidism is characterized by diminished concentrations of both total and free T4, along with elevation in TSH concentration (Mooney 2011). In dogs, primary hypothyroidism presents as two distinct histological types: lymphocytic thyroiditis and idiopathic thyroid atrophy (Feldman and Nelson 2015). Typically, thyroid atrophy progresses gradually and is often diagnosed in later stages of life. Histologically, lymphocytic thyroiditis is characterized by the diffuse infiltration of lymphocytes, plasma cells, and macrophages into the thyroid gland, leading to gradual follicle breakdown and subsequent fibrosis. Although negative titres may be observed in the later stages of the disease, circulating TgAA is a valuable indicator of lymphocytic thyroiditis. Conversely, idiopathic atrophy of the thyroid gland is characterized by a gradual decrease in the size of thyroid follicles on histopathological examination. No signs of inflammatory infiltration were observed, even in regions containing small follicles or their remnants, and assessments for lymphocytic thyroiditis yielded negative results (Feldman and Nelson 2015). Hence, the dog was diagnosed with idiopathic thyroid atrophy.

While glycogen accumulation has occasionally been reported as a secondary feature of hypothyroidism in humans and experimental animals, such changes are generally mild, focal, and limited in extent (Fariduddin et al. 2025). In contrast, the extensive and diffuse vacuolar degeneration observed in both hepatic and renal tissues in this dog is inconsistent with changes expected from hypothyroidism alone. Moreover, the clinical phenotype – fasting hypoglycaemia, a non-ketotic state, and marked hyperlipidaemia – is more characteristic of GSD type Ia than of hypothyroidism. The histopathological findings in this case closely resemble those described in genetically confirmed canine GSD Ia cases (Brix et al. 1995; Specht et al. 2011), further supporting this interpretation. Therefore, this case suggests that the dog suffered from two concurrent but distinct conditions: idiopathic acquired primary hypothyroidism and presumptive GSD type Ia. Although such coexistence is rare, multiple endocrine disorders have been reported in dogs and represent a unique diagnostic challenge (Blois et al. 2011). In human medicine, cases of coexisting GSD type I and hypothyroidism have been reported (Melis et al. 2007). However, in humans, 1 out of 15 GSD type Ia cases had subclinical hypothyroidism, and clinical hypothyroidism was reported only in GSD type Ib (Melis et al. 2007; Ön et al. 2020). Several hypotheses could be proposed regarding the potential impact of GSD type 1 on the hypothalamus-pituitary axis and thyroid damage (Melis et al. 2007). These include glycogen accumulation in thyroid tissue, potential toxic effects of excessive lactic acid on thyroid tissue, impairment in endogenous glucose production within thyroid glands, and increased thyroid autoimmunity (Pomorski et al. 2002; Melis et al. 2007). In the present case, histopathological examination of thyroid tissue did not reveal glycogen accumulation, and blood lactic acid concentrations were increased, although the levels were initially within normal limits, suggesting risk of lactic acid toxicity. Furthermore, the cause of hypothyroidism was idiopathic thyroid atrophy. However, the presence of idiopathic thyroid atrophy without glycogen accumulation or inflammatory changes further supports the interpretation that these two conditions developed independently.

Therefore, impairment of endogenous glucose production or the toxic effects of excessive lactic acid on thyroid tissue can be considered the likely cause in the present case.

A limitation of this report is the inability to perform genetic mutation analysis or glucose-6-phosphatase activity testing, as only formalin-fixed tissues were available, precluding molecular and enzymatic confirmation. The absence of such confirmatory analyses represents a diagnostic limitation; therefore, the diagnosis of GSD type Ia in this case remains presumptive. However, histopathological examination of the Maltese dog revealed characteristic diffuse hepatocellular and proximal renal tubular vacuolar degeneration with glycogen accumulation. These findings, together with the clinical signs and breed predisposition, were consistent with previously reported cases of presumptive GSD type Ia in Maltese dogs. Additionally, the patient's signalment (1-year-old Maltese), clinical histories, examination findings, and laboratory data collectively support the diagnosis of GSD type Ia. Furthermore, due to limitations in tissue availability at the time of postmortem examination, other endocrine organs – including the pituitary gland, adrenal glands, and pancreas – were not histologically evaluated. While this restricts our ability to fully exclude additional endocrine abnormalities, no clinical signs or laboratory findings during hospitalization suggested dysfunction of these organs. Specifically, the patient did not exhibit neurological signs indicative of pituitary disease, nor electrolyte or haemodynamic abnormalities suggestive of adrenal insufficiency, and there was no evidence of diabetes mellitus or pancreatitis. Additionally, although PAS staining demonstrated cytoplasmic granules consistent with glycogen, PAS-diastase staining was not performed, limiting definitive histologic confirmation of glycogen accumulation.

When a dog exhibits multiple biochemical and physical abnormalities, conducting a thorough assessment becomes imperative to optimize the patient's health because both congenital and acquired conditions can occur concurrently. Clinicians should remain mindful that dogs diagnosed with a single acquired disease often harbour concurrent congenital illnesses, prompting exploration when appropriate clinicopathological abnormalities are identified. Considering the possibility of concurrent hypothyroidism in young Maltese dogs suspected of GSD type Ia may facilitate earlier diagnosis and treatment, potentially improving clinical outcomes. If clinicians consider that primary hypothyroidism could occur concurrently in young Maltese dogs with suspected GSD type Ia, we believe that this will lead to faster diagnosis and treatment in other similar cases, thereby improving the patients' prognosis. Notably, despite the restoration of hypoglycaemia, the dog exhibited persistent comatose mentation, raising the question of whether the mentation status could be attributed to myxoedema coma resulting from hypothyroidism. Although hypothyroidism is not typically considered a cause of hypoglycaemia, retarded growth and severe hyperlipidaemia could be possible characteristics of concurrent hypothyroidism in young Maltese dogs. Furthermore, GSD type Ia is considered as a poor prognostic disease in dogs. Additional treatments for primary hypothyroidism could potentially improve the prognosis in some dogs of the GSD type Ia with concurrent primary hypothyroidism, although our report did not explore this aspect. Therefore, investigating concurrent hypothyroidism in dogs with GSD type Ia and administering treatment for primary hypothyroidism could provide valuable insight into potential therapeutic benefits.

This report describes the co-occurrence of primary hypothyroidism in a young Maltese suspected of GSD type Ia, offering valuable insights for prompt diagnosis and treatments in subsequent cases. Similar to the current case, expedited diagnosis and treatment could lead to a more favourable prognosis.

Acknowledgements

This work was supported by the National Research Foundation of Korea (NRF) grant funded by the Korea government (MSIT) (No. RS-2025-24683314).

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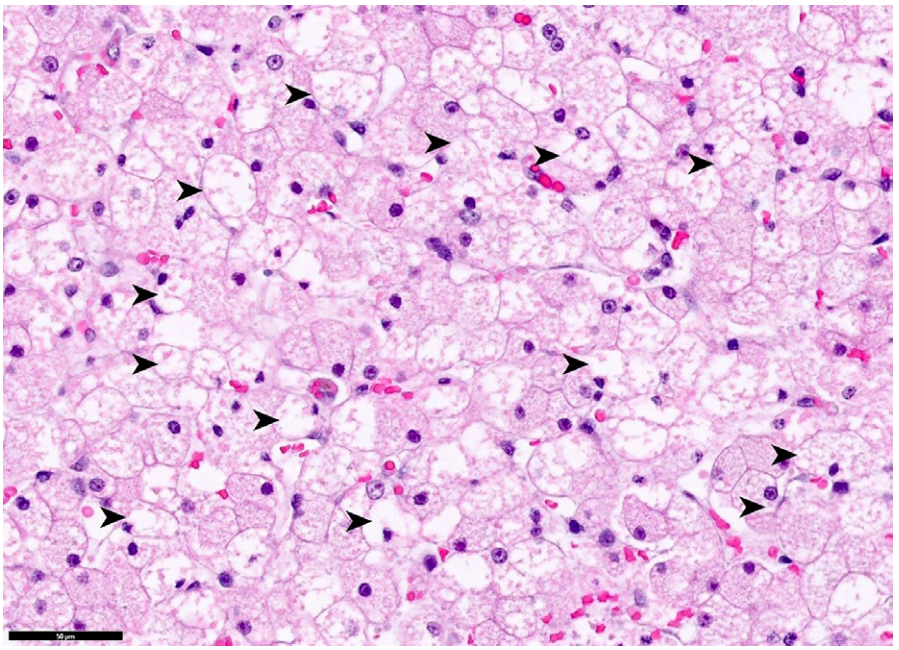

Fig. 1. Microscopic section of the liver illustrating severe vacuolization of hepatocytes. The hepatocytes exhibit diffuse expansion with abundant lacy to vesicular cytoplasm, occasionally containing vacuoles (glycogen and lipid-type), leading to compression and obstruction of adjacent sinusoidal spaces (arrowheads). Staining: Haematoxylin and eosin. Scale bar: 50 µm.

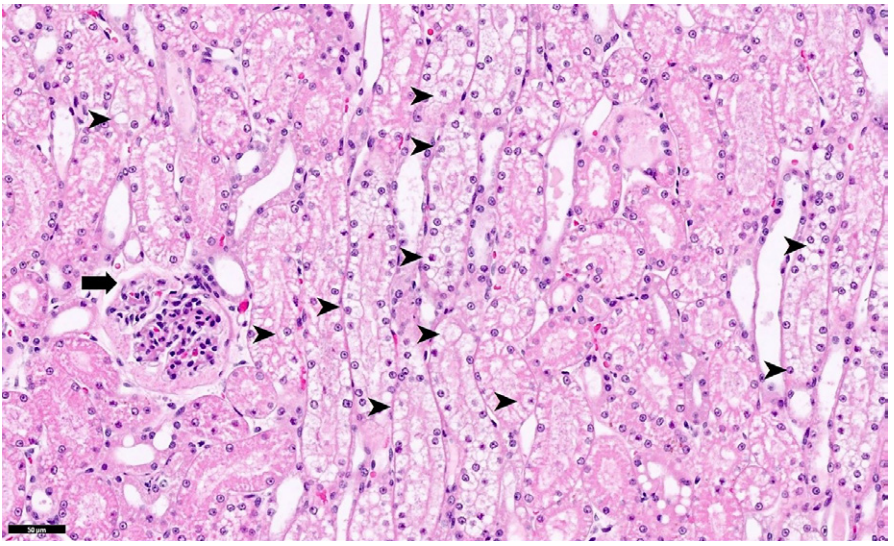

Fig. 2. The epithelium of proximal renal tubules exhibits diffuse expansion, characterized by moderate to marked enlargement with increased amounts of lacy to vesicular cytoplasm, occasionally containing vacuoles (arrowheads). Additionally, scattered renal corpuscles show segmental thickening and sclerosis of their basement membranes (arrow). Staining: Haematoxylin and eosin. Scale bar: 50 µm

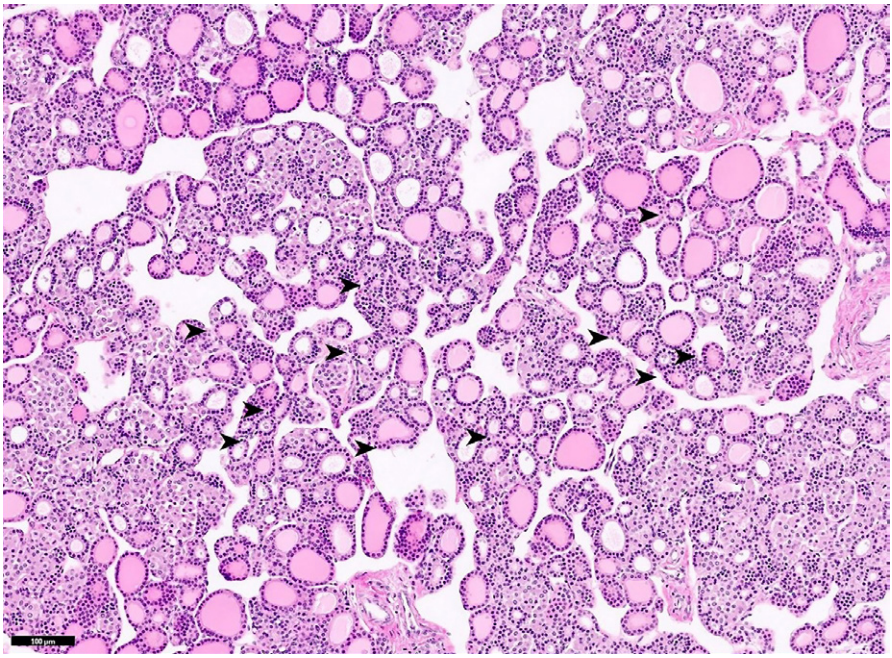

Fig. 3. Microscopic section of the thyroid revealing approximately 40% of thyroid follicles as small or atrophied (arrowheads), collapsed, and lacking colloid. No inflammatory changes are observed, ruling out lymphocytic thyroiditis. Staining: Haematoxylin and eosin. Scale bar: 100 µm.

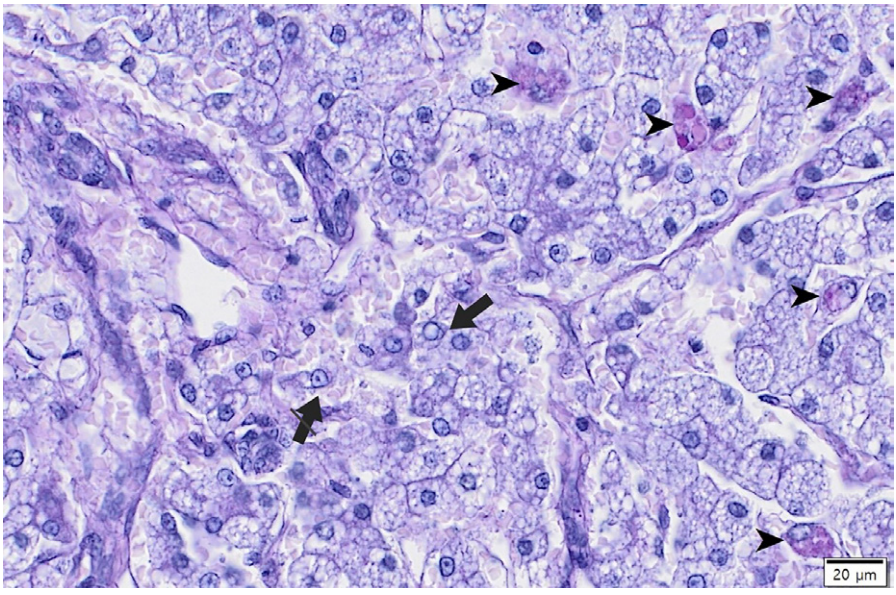

Fig. 4. Periodic acid-Schiff staining of the formalin-fixed liver reveals coarse granules of stored glycogen within vacuoles, highlighted by haematoxylin and eosin staining (arrowheads). Additionally, glycogenised nuclei are observed (arrow). Staining: Periodic acid–Schiff. Scale bar: 20 µm.