# What's your Diagnosis?

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## Case presentation

Two suckler calves from an Angus herd were presented to the University of Zurich, Veterinary Hospital in September 2022 because of diarrhea and ventral edema. Both animals were born in spring 2022 in the canton of Zurich (around 416m above sea level) and housed on changing alpine pastures (all above 1589m) since June in the canton of Graubünden.

## Case one: 4-month-old steer

The first case was a 4-month-old steer, weighing 170 kg, with no prior health concerns. Two days prior to presentation, watery diarrhea was observed by the alpine herdsman and the steer was treated by the local veterinarian with oral electrolytes and oxytetracycline (Engemycin 10 % ad us vet., MSD Animal Health GmbH, Werftestrasse 4, 6005 Luzern, 10 mg/kg, i.m.). The owner decided to take the animal back to its original farm where he noticed a swelling at the brisket. The steer was then referred to the veterinary hospital without additional therapy.

Clinical examination showed a body condition score of 4/5, lethargy, spontaneous cough, edema from the submandibular area down to the brisket (Figure 1) and bilaterally distended jugular veins with positive venous pulsation. Body temperature was 37,9°C (reference range 38,5–39,5°C) and heart rate and respiratory rate were increased (104 beats and 36 breaths per minute, respectively, reference ranges 60–80 beats per minute and 10–30 breaths per minute, respectively).[5] Auscultation of the heart revealed no abnormalities; lung sounds were increased (both inspiratory and expiratory) and gastrointestinal motility absent. Feces were pasty, olive and of decreased amount.

**Figure 1:** Submandibular edema (arrow) of the 4-month-old steer (case one).

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Venous blood gas, hematology, and biochemistry analysis as well as thoracic and abdominal ultrasound examinations were performed.

Main abnormalities were an elevated concentration of erythrocytes with hypochromia and microcytosis and a non-degenerative left-shift, azotemia, hyperphosphatemia, hyperbilirubinemia, and increased activity of liver enzymes as well as hypoglycemia and hyperlactatemia (Table 1).

Ultrasonographic examination revealed a small amount of pleural effusion with atelectasis of both cranioventral pulmonary lobes and a round, distended caudal vena cava. The intrahepatic vessels appeared prominent, and the liver was mildly enlarged (Figure 2).

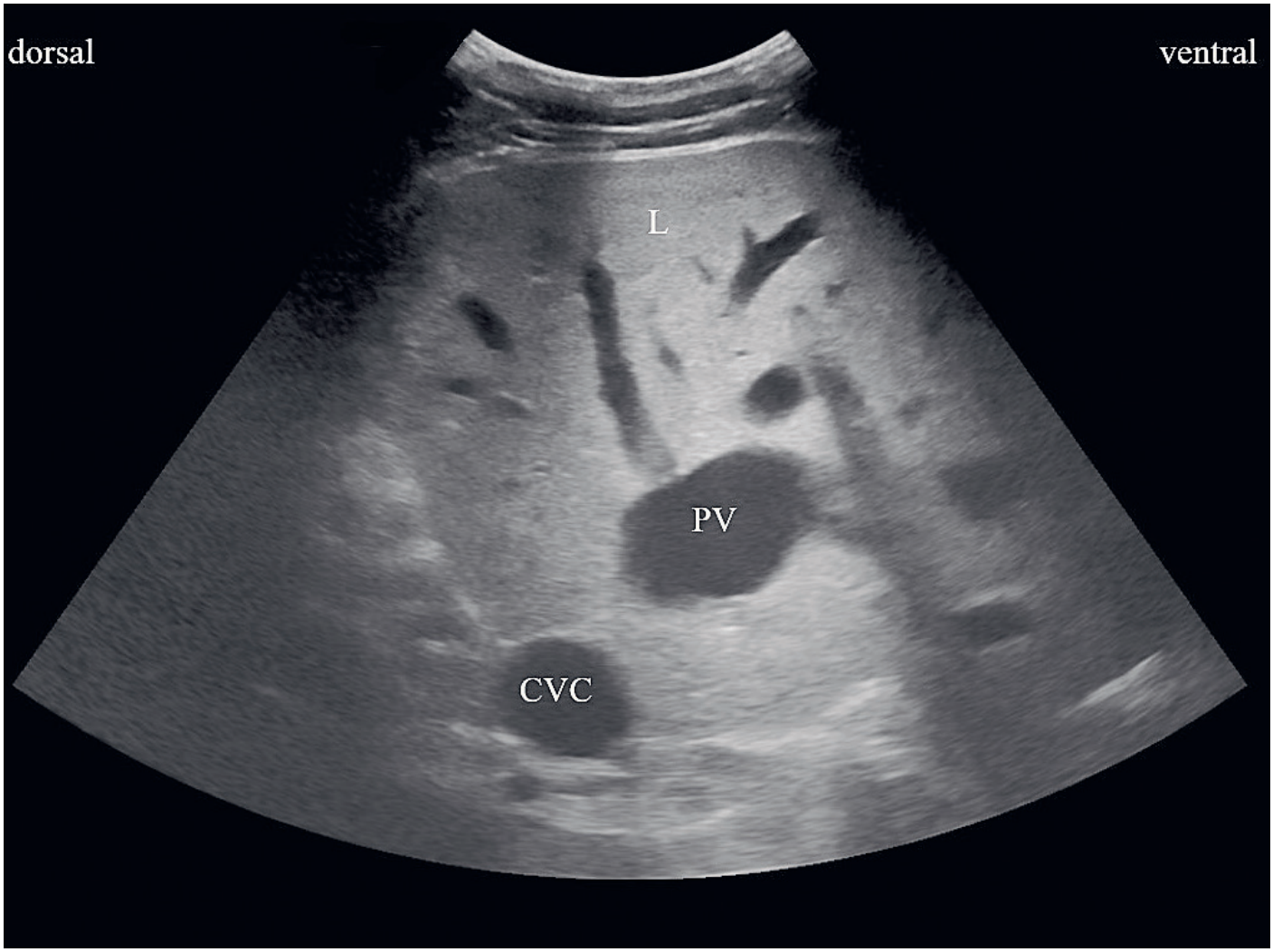


**Figure 2:** Ultrasonographic image of the liver of the 4-month-old steer (case one). The caudal vena cava is distended, and the other hepatic vessels are very prominent. L = liver tissue, PV = portal vein, CVC = caudal vena cava (normally a triangular shape).

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**Table 1:** Hematological and biochemical findings in two Angus calves.

| | | Steer (case one) | | | Heifer (case two) | Reference ranges[1] |
|---|---|---|---|---|---|---|
| | | day 1 | day 4 | day 13 | day 1 | |
| **Hematological parameters** | | | | | | |
| **PCV*** | % | **35** | – | – | **41** | 25–33 |
| **Hemoglobin** | g/dl | 10,9 | – | – | **12,9** | 8,7–11,8 |
| **Erythrocytes** | *10^6/µl | **10,26** | – | – | **11,33** | 4,9–6,9 |
| **MCH*** | pg | **11** | – | – | **11** | 15–20 |
| **MCHC*** | g/dl | **31** | – | – | **31** | 35–37 |
| **MCV*** | fl | **34** | – | – | **36** | 41–52 |
| **Leucocytes** | *10^3/µl | 8,1 | – | – | **13,3** | 4–8,8 |
| **Rod-shaped neutrophiles** | *10^3/µl | **0,12** | – | – | **0,27** | 0–0,07 |
| **Segmented neutrophiles** | *10^3/µl | **4,52** | – | – | **10,7** | 1,23–3,28 |
| **Basophiles** | *10^3/µl | 0 | – | – | **0,2** | 0–0,08 |
| **Monocytes** | *10^3/µl | **0,24** | – | – | **0,8** | 0–0,17 |
| **Lymphocytes** | *10^3/µl | 3,19 | – | – | **1,33** | 2,19–5,12 |
| **Thrombocytes** | *10^3/µl | 338 | – | – | **616** | 210–452 |
| **Biochemical parameters** | | | | | | |
| **Total protein** | g/l | 64 | **53** | 65 | 73 | 64–77 |
| **Urea** | mmol/l | **14,6** | 5,5 | 2,5 | **7,3** | 2,4–6,5 |
| **Creatinine** | µmol/l | **204** | **135** | **129** | **117** | 55–103 |
| **Total bilirubin** | µmol/l | **31,7** | **8,4** | 2,7 | **13,6** | 0,1–2,9 |
| **ASAT** | U/l | **249** | **129** | 87 | **221** | 57–103 |
| **γGT** | U/l | **319** | **302** | **290** | **188** | 13–32 |
| **GLDH** | U/l | **172,3** | **172,4** | **54,6** | **137** | 4–18,2 |
| **SDH** | U/l | **21** | **23,2** | **9** | **118,6** | 4–7,4 |
| **CK** | U/l | **1626** | **240** | **453** | **795** | 70–169 |
| **Sodium** | mmol/l | **137** | 140 | 140 | 142 | 138–145 |
| **Potassium** | mmol/l | 4,9 | **3,8** | 4,4 | 4 | 3,9–5 |
| **Chloride** | mmol/l | 97 | **105** | 101 | 102 | 94–102 |
| **Calcium** | mmol/l | **2,2** | **1,95** | **2,29** | 2,5 | 2,3–2,6 |
| **Magnesium** | mmol/l | 0,85 | **0,66** | 0,91 | **0,53** | 0,8–1 |
| **Phosphate** | mmol/l | **4,36** | **3,03** | 2,19 | 1,33 | 1,3–2,4 |
| **L-Lactate[2]** | mmol/l | **4,77** | **0,8** | – | **2,73** | 0–2 |

PCV = packed cell volume, MCH = mean corpuscular hemoglobin, MCHC = mean corpuscular hemoglobin concentration, MCV = mean corpuscular volume
ASAT = Aspartate transaminase, γGT = γ-Glutamyl transferase, GLDH = Glutamate dehydrogenase, SDH = Sorbid dehydrogenase, CK = Creatine Kinase.
Variables in bold are outside of the reference ranges.
[1]reference ranges established by the performing laboratory; [2]measured with RapidPoint 500, reference ranges from Constable et al.[5]

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## Case two: 6-month-old heifer

The second animal was a 6-month-old heifer weighing 140 kg. While it was coughing prior to alpine residence, according to the herdsman, it had appeared healthy over the course of the summer. Signs of diarrhea were noticed on the day of referral of the steer, so the owner elected to bring both animals to the university hospital.

The heifer showed a reduced body condition score (2,5/5), spontaneous cough, serous nasal discharge, and distended jugular veins (Figure 3). Body temperature (40,1°C) and heart rate (108 bpm) were elevated. Respiratory rate was 32 bpm, and expiratory wheezing was noticed on the left side. Gastrointestinal motility was reduced.

Main abnormalities on hematological and biochemical examination included elevated hematocrit and erythrocyte count with hypochromia and microcytosis, thrombocytosis, leukocytosis with a non-degenerative left shift, azotemia, hyperbilirubinemia, hyperlactatemia, and increased activity of liver enzymes (Table 1).

Ultrasonographic examination showed similar results as the one performed in the steer (moderate pleural effusion, atelectasis of pulmonary lobes, round caudal vena cava, enlarged portal vein), but also small, consolidated areas distributed over the entire lung field and the right ventricle of the heart seemed significantly larger than the left.

Based on the information above, formulate your differential diagnoses, further diagnostics to confirm or to disprove them and recommended therapy.

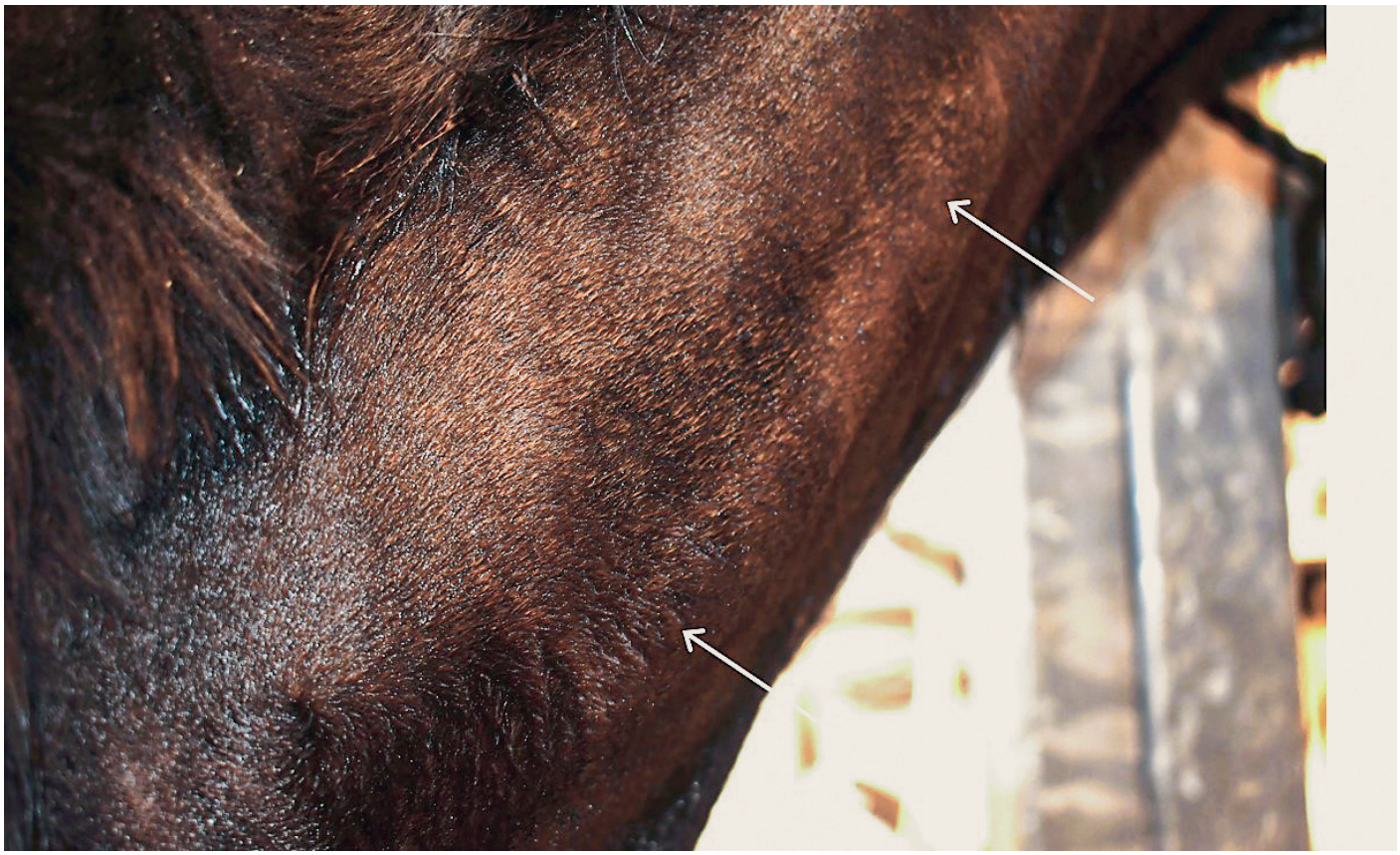

**Figure 3:** Distended jugular vein (arrow) of the 6-month-old heifer (case two).

## Differential diagnoses

Differential diagnoses for the historically observed diarrhea include gastrointestinal diseases such as ruminal acidosis, gastrointestinal parasites or bacterial enteritis, hypoproteinemia due to renal or gastrointestinal losses, or, less commonly, liver failure, and congestive heart failure.

Cough can be caused by inflammation of the respiratory tract such as enzootic bronchopneumonia, dictyocaulosis or because of pulmonary edema.

Possible reasons for peripheral edema are decreased oncotic pressure (hypoproteinemia), vasculitis, and elevated hydrostatic pressure due to congestive heart failure (globalized) as well as thrombosis or compression of vessels (localized).

Due to distended jugular and abdominal veins, pleural effusion and liver congestion, congestive right-sided heart failure due to hypoxia was suspected. Possible causes for right-sided heart failure are presented in Table 2.[2,3,4,5,31]

## Further diagnostics

The day after arrival to the hospital, arterial blood gas analysis was performed and demonstrated a decreased pCO2 and elevated pO2 in the steer, most probably due to hyperventilation, and in the heifer a decreased pO2 due to impaired diffusion or less likely due to hypoventilation. Calculated sO2 was decreased in both animals (Table 3). Radiographs of the thorax were performed, and echocardiography was repeated in both animals by a board-certified internal medicine specialist experienced in large animal cardiology.

Ultrasonographic examination of the heart was suggestive of pulmonary hypertension (dilated pulmonary artery with reduced distensibility), congestive right-sided heart failure (dilated right atrium and ventricle, right ventricular hypertrophy, increased septal thickening, bulging into the left ventricle and abnormal motion, Figure 4 and Figure 5), and secondary insufficiency of the tricuspid valve.

Thoracic radiographs of the steer showed normal lung patterns but a dilated caudal vena cava. The heifer had an alveolar lung pattern, most likely due to chronic bronchopneumonia.

Pressure measurements of the right atrium (RAP), right ventricle (RVP) and pulmonary artery (PAP) were conducted by a board-certified internal medicine specialist experienced in large animal cardiology and were elevated (Table 4). Cardiac Troponin I (cTnI) was measured and was 0,05 ng/ml for the steer and 0,03 ng/ml for the heifer on the day of admission (reference range 0- 0,05 ng/ml[15]).

Fecal samples of both calves were taken for parasitological examination at which small numbers of eggs of intestinal parasites (*Trichostrongylus spp.*) and no larvae of lung worms were found.



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**Table 2:** Differential diagnoses for congestive right-heart failure in cattle and diagnostic tools to investigate them further.

| | |
|---|---|
| **Myocardial injury (Cardiomyopathy)** | |
| Nutritional (Vitamin E- / Selenium-deficiency, copper-deficiency) | Cardiac Troponin I: elevated |
| Mechanical (perforating foreign bodies, turbulent blood flow) | |
| Cardiac lymphoma | |
| Infectious (bacteremia, viral, parasitic, sepsis) | |
| Toxic (monensin, vitamin D, oleander, snake bites) | |
| Idiopathic | |
| **Left-to-right shunt** | |
| Patent foramen ovale | Ultrasonographic examination |
| Ventricular septal defect | |
| Atrial septal defect | |
| PDA | |
| **Insufficiency of tricuspid valve** | |
| Congenital | Ultrasonographic examination |
| Endocarditis valvularis | |
| **Stenosis of the pulmonary valve or pulmonary artery** | |
| Congenital | Ultrasonographic examination |
| Endocarditis valvularis | |
| Thromboembolism | |
| **Pulmonary hypertension** | |
| Left-sided heart failure | Measurement of pulmonary artery pressure Ultrasonographic and radiologic examination, clinical history |
| Interstitial pulmonary disease (bronchopneumonia, pulmonary edema) | |
| Angiopathy (arterial sclerosis, intravascular parasites, thromboembolism) | |
| Alveolar hypoxia (bronchial obstruction, high altitude) | |
| Gram-negative sepsis | |
| **Restricted ventricular filling** | |
| Pericardial effusion (pericarditis, perforating foreign bodies) | Ultrasonographic examination |
| Pleural effusions (thoracic neoplasia, pleuritis) | |

**Table 3:** Temperature-corrected results of arterial blood gas analysis in two Angus calves.

| | | Steer (case one) | Heifer (case two) | Reference ranges[1] |
|---|---|---|---|---|
| **Temperature** | **°C** | 38,4 | 39,0 | – |
| **pH** | **–** | 7,408 | **7,477** | 7,35–7,45 |
| **pCO2** | **mmHg** | **32,7** | 36,7 | 35–45 |
| **pO2** | **mmHg** | **109,6** | **84,9** | 95–105 |
| **sO2[2]** | **%** | **97,9** | **96,4** | > 98 |

Variables in bold are outside of the reference ranges.
[1]Reference ranges from Constable et al.[5]; [2]calculated sO2 is not temperature-corrected.

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## Diagnosis

In both cases, the diagnosis of high-altitude disease was made based on history, breed disposition, clinical and further diagnostic findings.

**Table 4:** Mean pressures (in mmHg) in two Angus calves.

| | Steer (case one) | | Heifer (case two) | Reference ranges[1] |
|---|---|---|---|---|
| | day 3 | day 13 | day 3 | |
| **Pulmonary capillary wedge pressure (PCWP)** | 15 | 8 | **31** | 5–21 |
| **Pulmonary artery pressure (PAP)** | **Systolic 72**<br>**Diastolic 37**<br>**Mean 51** | **Systolic 52**<br>**Diastolic 30**<br>**Mean 41** | **Systolic 58**<br>**Diastolic 27**<br>**Mean 40** | 33–46<br>19–21<br>24–31 |
| **Right ventricle pressure (RVP)** | **Systolic 79**<br>**Diastolic –16**<br>**Mean 38** | Systolic 53<br>Diastolic 0<br>Mean 24 | **Systolic 57**<br>**Diastolic -2**<br>Mean 27 | 42–56<br>0–1<br>19–28 |
| **Right atrium pressure (RAP)** | **32** | **16** | **24** | <5 |

Variables in bold are outside of the reference ranges. [1]Reference ranges from Smith et al.[31]

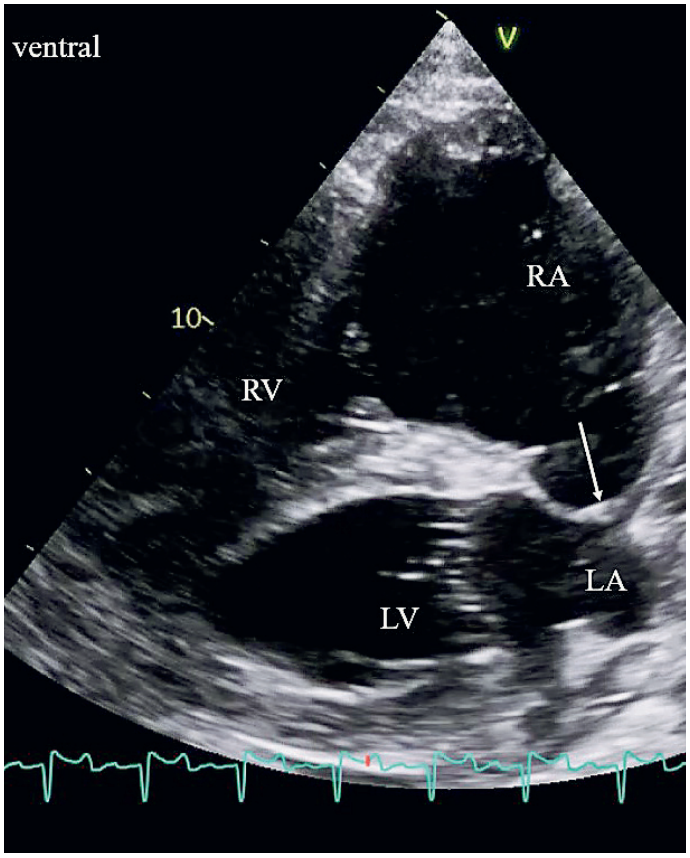


**Figure 4:** Echocardiographic image (4-chamber-view) of the heart of the 4-month-old steer (case one). The right atrium and right ventricle are larger than the left atrium and ventricle, respectively. The atrial septum (arrow) is bulging into the left atrium. RA = right atrium, RV = right ventricle, LA = left atrium, LV = left ventricle.

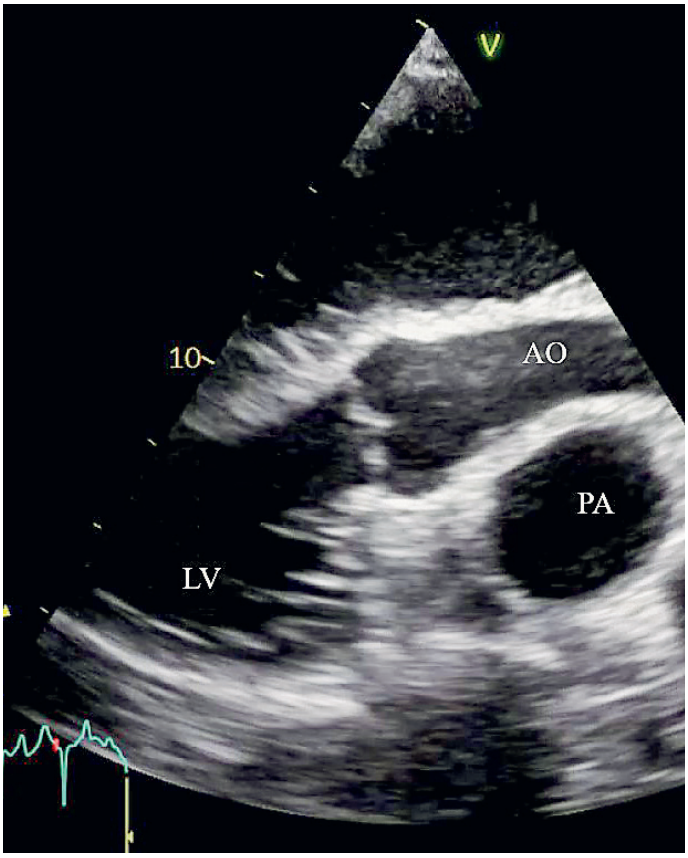


**Figure 5:** Echocardiographic image of the dilated pulmonary artery of the 6-month-old heifer (case two). The diameter of the pulmonary artery is bigger than the diameter of the aorta during maximal distension of the aorta. AO = aorta, PA = pulmonary artery, LV = left ventricle.

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## Case one: clinical progression

The steer was treated with intravenous fluids (Infusionskonzentrat Dr. Braun, Laboratorium Dr. G. Bichsel AG, Weissenaustrasse 73, 3800 Unterseen, NaCl 0,9 %, Glucose 5 %, 2 ml/kg/h) for three days, furosemide (Dimazon ad us vet, MSD Animal Health GmbH, Werftestrasse 4, 6005 Luzern, 1 mg/kg i.v.) once daily for a total of 8 days and vitamin E / selenium (Tocoselenit ad us vet, Dr. E. Graeub AG, Rehhagstrasse 83, 3018 Bern, 15 ml s.c.) once. Blood gas analysis was repeated every second day to monitor potassium concentration for the duration of furosemide treatment. The steer's general condition improved and the edema as well as the distension of the jugular veins diminished progressively.

Blood chemistry analysis was repeated on day four and 13 (Table 1). All altered parameters decreased towards the reference range, but stayed elevated (except for lactate, urea, bilirubin, ASAT, and phosphate).

On day eight post admission, the steer developed a fever (body temperature 39,9°C) and nasal discharge. Thoracic ultrasonographic examination revealed large consolidations of the apical pulmonary lobes and antibiotic and anti-inflammatory therapy was initiated with amoxicillin and clavulanic acid (Synulox Suspension ad us vet., Zoetis Schweiz GmbH, Rue de la Jeunesse 2, 2800 Delémont, 8,75 mg/kg i.m., for 7 days) and ketoprofen (Rifen 10 % ad us vet., Streuli Tiergesundheit AG, Bahnhofstrasse 7, 8730 Uznach, 3 mg/kg, i.m., for two days) and meloxicam (Inflacam 20 mg/ml ad us vet., Virbac Switzerland AG, Cherstrasse 4, 8152 Glattbrugg, 0,5 mg/kg, s.c., as needed in case of fever the following days).

On day 12, the central venous pressure measurements and ultrasonographic examination were repeated as well. All measured pressures had decreased (Table 4), and ultrasonographic signs of congestive right-heart failure (dilated right ventricle, distended caudal vena cava, round liver edges) and pneumonia (consolidation of pulmonary lobes, pleural effusion), were improved.

The animal was discharged after 18 days of hospitalization. At this time point, its general condition had greatly improved, appetite and feces were normal, and ventral edema and overt jugular vein distension were resolved. Mild tachycardia (88 to 108 per minute) and tachypnoea (44 to 56 per minute) were, however, still present.

Six months later, the clinical examination on farm was normal (jugular veins, heart and lung auscultation and rates, no edema), but the animal showed retarded growth compared to its herd mates.

## Case two: pathological findings

Due to additional pneumonia and the already retarded weight gain, the owner decided to euthanize the heifer. The body was submitted to the University of Zurich, Institute of Veterinary Pathology for necropsy and histopathological examination.

Heart weight was 1,38 kg, which correlated to 0,9 % of the body weight (reference ranges 0,3–0,7 %)[17,24] and relation of right to left ventricular free wall was 1:1.8 (1:2–1:3).[18,24] The (reference ranges 1:2–1:3) right atrium and ventricle as well as the pulmonary trunk were severely dilated (Figure 6) with a thickened wall of the right ventricle and the septum. The left atrioventricular valve showed a linear dysplasia separating the atrioventricular entry into two parts, but no sign of hemodynamic consequences (i.e., multifocal, subendocardial lesions and plaques, so called jet lesions)[17] were seen. No histological changes were found.

The cranioventral lobes of the lungs were darkened and consolidated, the right one with small (0,5 cm) nodular abscesses. Histological findings included infiltrates of lymphocytes, macrophages and neutrophiles, an increased amount of collagenic connective tissue within the interstitial space, around the vessels and in the adventitia of the vessels. Larger vessels also showed thickening of middle and inner layers. All pulmonary vessels were highly filled with erythrocytes (Figure 7). Bacteriological culture revealed small amounts of *Pasteurella multocida*, *Escherichia coli* and *Streptococcus* subspecies.

The liver was enlarged, red-brown, and histological examination demonstrated a thickened liver capsule and increased amounts of periportal connective tissue (positive Van Gieson stain).

Brain, kidneys, and spleen were macroscopically normal, but in the kidneys, histological signs of hyperemia (vessels filled with erythrocytes) were found while the spleen showed small amounts of extramedullary hematopoiesis due to hypoxemia.

Other organs, including the gastrointestinal tract, were macroscopically and histologically normal.

The pathological diagnoses were congestive heart failure secondary to high-altitude disease and purulent, secondary bronchopneumonia.

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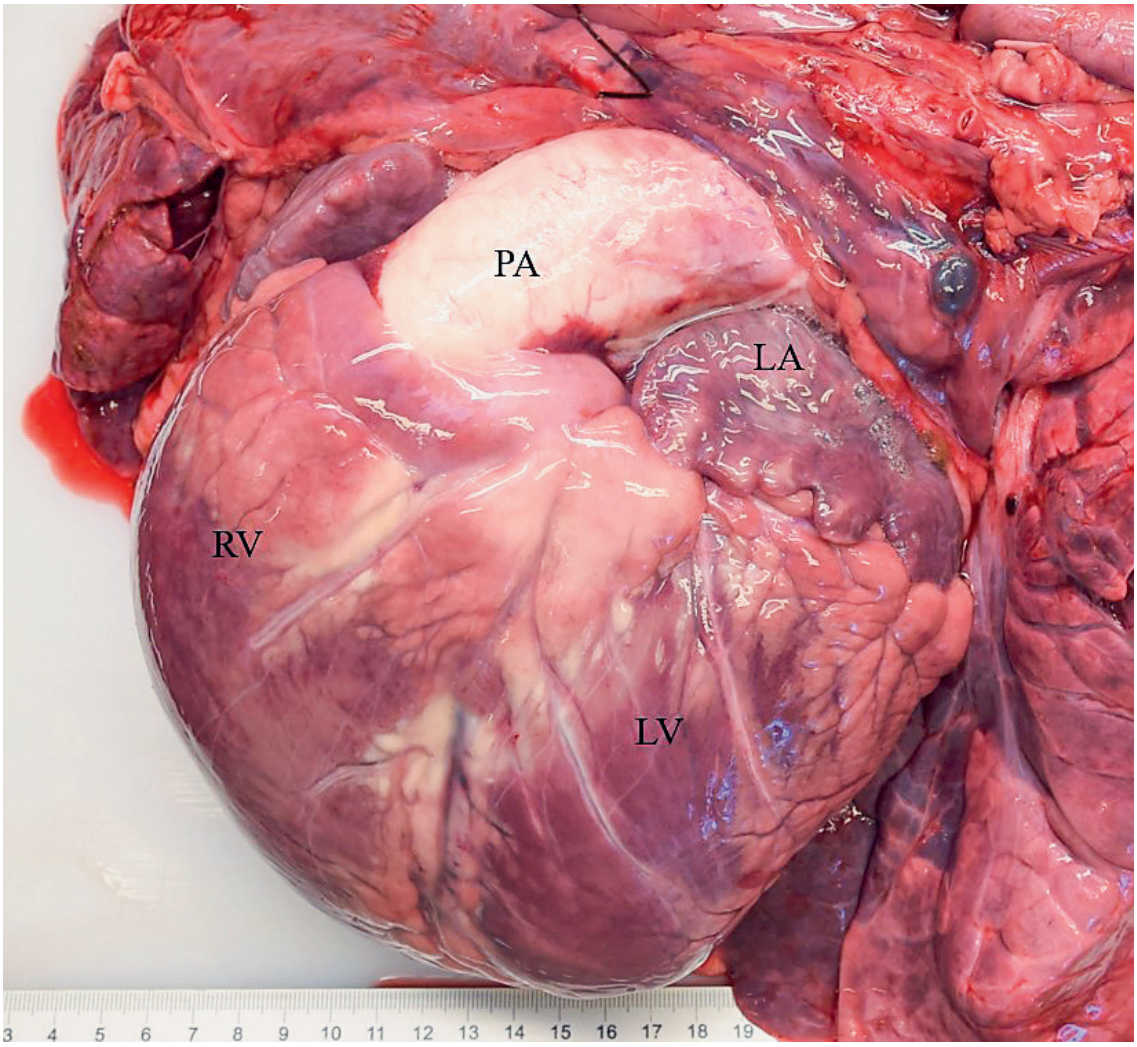


**Figure 6:** Gross pathology of the heart of the 6-month-old heifer (case two). The right ventricle and pulmonary artery are enlarged, and the apex of the heart is rounded. PA = pulmonary artery, RV = right ventricle, LA = left atrium, LV = left ventricle.

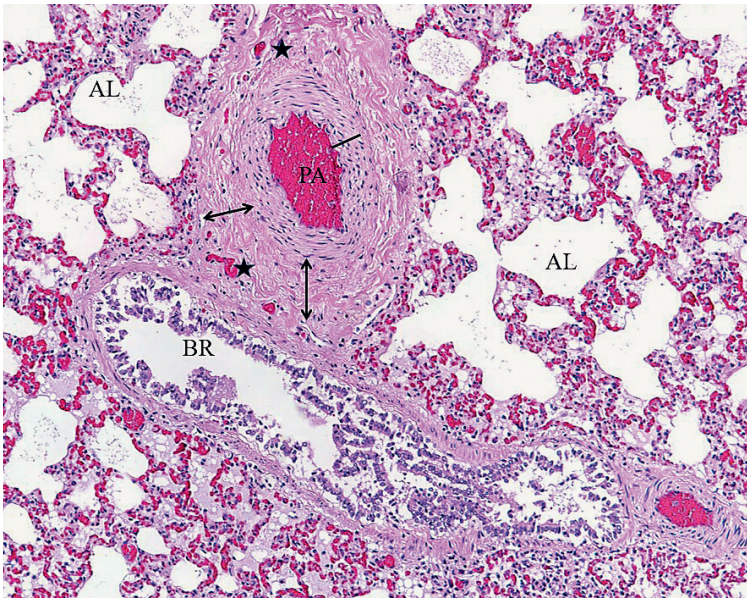


**Figure 7:** Histological image of the lung of the 6-month-old heifer (case two). The adventitia, the middle and the inner layer of the pulmonary artery are thickened, and the adventitia contains an increased amount of collagenic connective tissue. All pulmonary vessels are filled with erythrocytes. AL = alveolus, BR = bronchiolus, PA = pulmonary artery, – = middle layer, ↔ = adventitia, ★ = vasa vasorum.

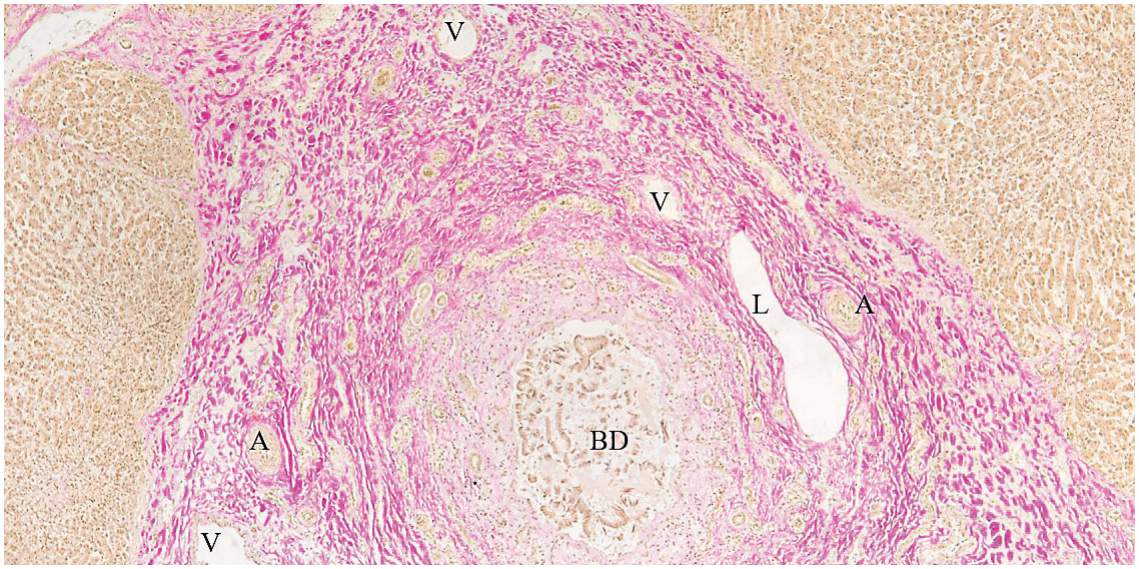


**Figure 8:** Histological image of the liver of the 6-month-old heifer (case two) (periportal field, Van Gieson stain). The increased content of collagenic connective tissue is painted pink. A = interlobular artery, BD = interlobular bile duct, L = lymphatic vessel, V = interlobular vein.

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## Discussion

### Pathogenesis and treatment

Both animals were presented with a primary complaint of diarrhea, a sign typically associated with gastrointestinal pathogenesis (infectious enteritis or ruminal acidosis being the most common reasons). However, other reasons include hypoproteinemia following high intestinal or renal losses as well as low synthesis due to malnutrition or liver failure, and congestive right heart failure with high venous blood pressure and therefore loss of water from intestinal capillaries into the lumen. In these two cases, distended jugular veins and brisket edema indicated congestive heart failure.

Right-sided heart failure in cattle most commonly originates from congenital malformations with left-to-right shunt (ventricular septal defect, patent foramen ovale), tricuspid valve insufficiency due to endocarditis, or pericarditis due to traumatic reticuloperitonitis.[5,26,31] Furthermore, pulmonary hypertension can be found secondary to left-sided heart failure,[32] pulmonary[1,19,21] or vascular[1,32] diseases (i.e. bronchopneumonia, arteriosclerosis, thrombosis), gram-negative sepsis,[10,16,27] or hypoxia due to low oxygen-content in the air due to high altitude, the latter called bovine high-altitude disease.[5,6,10,13,14,21,31,36]

First cases of bovine high-altitude disease were described in the early 20th century in Colorado, Wyoming, New Mexico, and Utah ("Mountain States") above 2000m above sea level as brisket disease, and hypoxic pulmonary hypertension was thought to be the underlying cause.[10,16,36] Although cases similar to the ones reported in this report were documented at moderate altitude above 1500m since the 1970th[19,21] in the USA, no cases were described in Europe to our knowledge to date, even though high alpine pastures have a long tradition in some countries. According to studies conducted in Colorado, a clear breed disposition could not be confirmed, even though the Angus breed seemed to be frequently affected.[10,19] Other reported breeds diagnosed with the disease are Hereford, Shorthorn, and Holstein.[16,28]

Hypoxia leads to constriction of pulmonary arteries[33] with the goal to lower blood flow in hypoxic alveoli and consequently higher blood flow with more efficient oxygen absorption in better ventilated alveoli.[10,13] Within days to weeks, persistent hypoxia results in vascular remodeling with hypertrophy of the arteriolar smooth muscles (medial hypertrophy) and adventitial tissue in the pulmonary artery wall.[10,11,33] This adaption is constructive in localized hypoxia found with bronchopneumonia (viral, bacterial, parasitic, or thrombo-embolic) but maladaptive in generalized hypoxia due to high altitude, leading to pulmonary hypertension followed by increased pressure on the right heart, which responds with hypertrophy and dilatation of the right ventricle. Depending on the duration of development, it can lead to congestive right heart failure over time with increased systemic venous blood pressure.[10,13]

Cattle are highly susceptible to hypoxic pulmonary hypertension because of their high amount of smooth muscle in the pulmonary arterial wall, the lobulated lung pattern without collateral ventilation and the narrow ratio of lung-size to body-weight with little reserve lung capacity.[9,13,19,28]

Case reports of congestive heart failure also exists in zoo mammals housed at levels higher than 2000m above sea level. In a retrospective study of 17 zoo animals (maras, capybaras, Bennet's wallaby, cotton-top tamarins, nilgai antelope and scimitar-horned oryx) with detailed pathology results, all had right heart hypertrophy and remodeling of pulmonary arteries as described in cattle.[12]

Clinical signs normally occur within several weeks and include weakness, apathy, reduced appetite, distended jugular veins, peripheral edema mainly in the brisket region, tachycardia, tachy- and/or dyspnea and diarrhea.[1,10,14,26] Other reported findings are pleural effusion, abdominal effusion with a distended ventral abdomen, enlarged liver with secondary intrahepatic cholestasis (in severe cases clinical icterus) and congestion of the kidneys.[1,19,26] In case of tricuspid valve insufficiency, jugular vein pulsation and a systolic heart murmur may be present.[10] In later stages, recumbency and death will occur.[10] Congestion and hypoxia of the liver lead to elevated activity of liver enzymes (SDH, GLDH, ASAT) and the cholestasis leads to elevated activity of GGT and hyperbilirubinemia.[1,3,4,8,30] Congestion and hypoxia of the kidney leads to renal insufficiency with azotemia and hyperphosphatemia.[5,7] All these changes were seen in both cases, except for hyperphosphatemia which was observed in the steer only.

Increased pulmonary pressure also reduces the blood flow in bronchial vessels, which supply the pulmonary tissue with oxygen and nutrients. Hence, animals with pulmonary hypertension are also highly susceptible for secondary bronchopneumonia as occurred in the steer.[13] The heifer showed signs of pneumonia on admission and had a history of chronic respiratory problems. It is impossible to know whether the pulmonary hypertension developed before or after the onset of pneumonia.

Additionally, generalized hypoxia leads to hypoxemia and therefore to erythropoiesis. In both cases, an increased hematocrit and erythrocyte count and a decreased MCV and MCH showed that long-time, exhausted erythropoiesis was evident. Hemoconcentration is exacerbated by water loss due to diarrhea, increased ventilation (leading to respiratory alkalosis) and urinary diuresis (to correct the respiratory alkalosis). As a consequence, blood viscosity and the pressure in the pulmonary capillaries increase.[1,20]

Reduced blood volume as well as pulmonary hypertension,[32] and bulging of the ventricular septum into the left ventricle all reduce left ventricular filling and stroke volume,[39] but cardiac output stays comparable to sea level because of persistent tachycardia.

Stopping the alveolar hypoxia as an inciting factor is the most important action. Breathing 100 % oxygen has been shown to decrease the pulmonary hypertension, but bringing the animal to lower altitude is required because of the impossibility of long-time oxygen-therapy.[14,31] Vasodilators show no effect on pulmonary hypertension[1,31] and are therefore not recommended. Symptomatic therapy includes diuretics to minimize the congestive heart failure,[4,31] correction of electrolyte imbalances (due to reduced feed intake or forced diuresis) to ensure contractility of the myocardium[4], correction of dehydration to ensure peripheral perfusion and broad-spectrum antibiotics due to the increased risk of secondary pneumonia.[13] The dose and frequency of diuretics administered (e.g. furosemide 1–5 mg/kg iv up to every 12 hours),[5,25] fluids and electrolyte supplementation should be re-evaluated clinically (e.g. edema, urine production and amount of abdominal or pleural effusion, distention of jugular veins, capillary refill time) and using laboratory variables (hematocrit, L-lactate, creatinine, electrolytes) at least once daily.

Clinical manifestations can disappear within 4 to 6 weeks, but return of pulmonary artery pressure to normal levels can take up to 10 months.[14,36]

## Diagnosis and prevention

Diagnosis is confirmed with a history of high-altitude residence, clinical signs of congestive right heart failure and pulmonary hypertension, as well as by ruling out other major differential diagnoses. Pulmonary hypertension can be confirmed by PAP. Pulmonary capillary wedge pressure (PCWP) normally reflects the left atrial pressure and therefore is increased in cases of left-sided heart failure. In pulmonary hypertension secondary to left-sided heart failure, the difference between PCWP and PAP is less than 10 mmHg. Otherwise, elevated PAP with normal PCWP exclude a left-sided heart failure as underlying pathogenesis of pulmonary hypertension.[32]

Mean PAP is influenced by many factors like breed, genetic background, pregnancy, age, body condition, concurrent illness leading to pulmonary hypoxia, elevation, environmental temperature, and ingestions of monocrotaline, ionophores or swainsonine, an indolizidine alkaloid occurring in some plants of species *Astragalus* and *Oxytropis*, also called locoweed and prevalent in North America.[1,10,29,38]

Some reports assume that high-altitude disease causes 5 % of the deaths and highly susceptible animals (elevated PAP without clinical signs) can make up around 30 % of a herd.[10,22] Therefore, a well-established preventative measure is to identify susceptible animals at low altitude. For that, preventive PAP-measuring at around 1500m is common in the Mountain States of the USA. Interpretation of PAP must consider other influencing factors, but a mean PAP higher than 49 mmHg usually indicates a high risk to develop high-altitude disease.[10] Normal mean PAP seems to be 27–29 mmHg at sea level and 31–41 mmHg at 2100m.[23] It is also important to remember that not all animals with increases in PAP will develop heart failure.[10]

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## Rindermedizin: Was ist Ihre Diagnose?

Zwei Angus-Kälber, die seit zwei Monaten in den Schweizer Bergen gealpt wurden, zeigten ein Trielödem, gestaute Jugularvenen und Durchfall. Die hämatologische und biochemische Untersuchung ergab eine erhöhte Erythrozytenkonzentration sowie eine erhöhte Aktivität der Leberenzyme. Die Ultraschalluntersuchung ergab einen geringgradigen Pleuraerguss, Hepatomegalie und eine gestaute kaudale Hohlvene. Die Diagnose einer kongestiven Herzinsuffizienz als Folge einer Höhenkrankheit wurde pathologisch bestätigt.

**Schlüsselwörter:** Kalb, Ödem, Herzinsuffizienz, Höhenkrankheit

## Bovine medicine: Whats´s your diagnosis?

Two Angus calves housed in the Swiss Alps for two months were presented with brisket edema, jugular distension, and diarrhea. Hematological and biochemical examination included elevated concentration of erythrocytes and increased activity of liver enzymes. Ultrasonography revealed small amount of pleural effusion hepatomegaly and congested caudal vena cava. The diagnosis of congestive heart failure secondary to high-altitude disease was confirmed in pathology.

**Keywords:** Calve, edema, congestive heart failure, high-altitude disease

## Médecine bovine: Quel est votre diagnostic?

Deux veaux Angus alpés dans les Alpes suisses depuis deux mois ont été présentés avec un œdème du poitrail, une stase jugulaire et de la diarrhée. Les examens hématologiques et biochimiques ont révélé une concentration élevée d'érythrocytes ainsi qu'une augmentation de l'activité des enzymes hépatiques. L'échographie a révélé un petit épanchement pleural, une hépatomégalie et une veine cave caudale congestionnée. Le diagnostic d'insuffisance cardiaque congestive secondaire à un mal des montagnes a été confirmé par la pathologie.

**Mots clés:** Veau, œdème, insuffisance cardiaque congestive, mal des montagnes

## Medicina dei bovini: qual è la vostra diagnosi?

Due vitelli di razza Angus alpeggiati nelle Alpi svizzere per due mesi mostravano un edema del petto, distensione giugulare e diarrea. Gli esami ematologici e biochimici hanno rilevato una concentrazione elevata di eritrociti e un aumento dell'attività degli enzimi epatici. L'ecografia ha rivelato una piccola quantità di versamento pleurico, epatomegalia e vena cava caudale congestionata. La diagnosi di insufficienza cardiaca congestizia secondaria alla malattia da alta quota è stata confermata dall'esame patologico.

**Parole chiave:** Vitello, edema, insufficienza cardiaca congestizia, malattia da alta quota