Common variable immunodeficiency in a horse

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Infectious conditions that are not responsive to medical treatment may be associated with immune disorders.

Measurement of serum immunoglobulin concentrations, protein electrophoresis, and response to vaccination are valuable clinical tests for detection and characterization of deficiencies in antibody production.

Immunologic tests, including flow cytometric analysis of blood lymphocyte subpopulations, lymphocyte proliferation assays, and immunohistochemical analysis of tissue sections, may provide additional information regarding immune cell function and distribution.

Although characterization of primary and secondary immunodeficiencies can be complex, immunologic investigation of clinical cases may help to characterize specific diseases of the immune system.

A 12-year-old Quarter Horse mare was evaluated at The Ohio State University Veterinary Teaching Hospital for a history of lethargy and anorexia of 2 days’ duration and intermittent chronic nasal discharge of 3 weeks’ duration. The horse had moderate signs of depression, was mildly febrile (rectal temperature, 38.4°C [101.1°F]), and had left unilateral, yellow, opaque nasal discharge and mild mandibular lymphadenopathy. Endoscopy of the upper airways revealed severe pharyngitis and numerous chondroids in the medial and lateral compartments of the left auditory diverticulum (guttural pouch). The right guttural pouch did not have clinical signs of inflammation. The horse was kept in a stall in the hospital isolation facility until bacteriologic culture results of the exudate from the left guttural pouch were obtained. *Streptococcus zooepidemicus* was isolated and was susceptible to penicillin, ampicillin, potentiated sulfonylureas, and rifampin, among other commonly used antimicrobial drugs. The WBC count on admission was 19,200 cells/µL (reference range, 4,700 to 10,600 cells/µL) with 400 band neutrophils/µL (reference range, 0 to 100 cells/µL), 15,400 mature neutrophils/µL (reference range, 2,400 to 6,400 cells/µL), 2,100 lymphocytes/µL (reference range, 1,000 to 4,900 cells/µL), and 1,300 monocytes/µL (reference range, 0 to 500 cells/µL). The fibrinogen concentration was 301 mg/dL (reference range, 193 to 422 mg/dL). Serum biochemical analyses revealed high activities of alkaline phosphatase (ALP, 1,580 U/L; reference range, 80 to 187 U/L), aspartate aminotransferase (AST, 588 U/L; reference range, 170 to 370 U/L); and gamma glutamyltransferase (GGT, 935 U/L; reference range, 9 to 24 U/L), and sorbitol dehydrogenase (SDH, 11.3 U/L; reference range, 4 to 13 U/L) activities within reference ranges. Albumin concentration was 3.2 g/dL (reference range, 2.8 to 3.6 g/dL) and the globulin concentration was 2.5 g/dL (reference range, 3.6 to 4.3 g/dL). The high WBC count was consistent with the underlying infectious disease. However, the findings of fibrinogen and globulin concentrations within reference ranges were surprising in association with chronic inflammation.

The owner chose conservative medical treatment of the guttural pouch inflammation instead of surgical management. The horse remained hospitalized for treatment and endoscopic monitoring of the upper portions of the airways. Initial treatment included daily lavage of the left guttural pouch with saline (0.9% NaCl) solution, and administration of ampicillin sodium (10 mg/kg [4.5 mg/lb], IV, q 8 h) and phenylbutazone (1 g, IV, q 12 h). Amoxicillin was chosen over penicillin because the latter was commercially unavailable. After 2 weeks of treatment, the horse’s leukogram had improved (WBC count, 6,100 cells/µL; 200 band neutrophils/µL; 3,700 segmented neutrophils/µL; 1,600 lymphocytes/µL; 400 monocytes/µL; 100 cosinophils/µL; and 100 basophils/µL). However, the horse remained mildly febrile (38.6°C [101.5°F]), with occasional fever spikes (40°C [104°F]).

Because of poor resolution of the chondroids in response to conservative treatment, the owner authorized surgical exploration of the left guttural pouch, which was drained via a modified Whitehouse approach on day 20 after admission. A second bacteriologic culture of material from the left guttural pouch yielded *S. zooepidemicus* with unchanged antibiogram pattern. Antimicrobial treatment was changed to trimethoprim-sulfamethoxazole (SMZ-TMP, 20 mg/kg [9 mg/lb], PO, q 12 h). Empyema of the left guttural pouch resolved after surgery. The horse’s attitude improved moderately, and a chemistry profile revealed SDH activity (8.3 U/L) within reference range, decreased although still high liver enzyme activities (ALP, 656 U/L; AST, 277 U/L; GGT, 241 U/L), decreased albumin concentration (2.3 g/dL), and unchanged globulin concentration. The fibrinogen

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concentration had increased to 848 mg/dL. Although liver enzyme activities had improved, the cause of the persistently high GGT and ALP activities was not apparent. Resolving cholangio hepatitis, possibly due to primary dissemination of the streptococcal organism, was suspected. Assessment of a liver biopsy specimen would have been desirable; however, the owner repeatedly declined further diagnostic testing for hepatic disease. Phenylbutazone administration was reduced (1 g, PO, q 24 h) because of the decrease in albumin concentration. The anti-inflammatory and antipyretic treatments were still deemed necessary, and the horse did not have other clinical signs of toxicosis caused by nonsteroidal anti-inflammatory drugs.

Ten days after surgery, the horse still had intermittent fever and cough and purulent material drained from the incision site. The horse developed severe diffuse cellulitis and abscessation of the subcutaneous tissue of the ventral cervical region. A Penrose drain was placed ventrally, approximately 30 cm cranial to the thoracic inlet, to allow effective drainage. *Streptococcus zooepidemicus* was cultured from the affected cervical soft tissues. In addition, nasal discharge persisted despite the resolution of the guttural pouch chondroids. Therefore, thoracic and skull radiographs were obtained. Thoracic radiographs were unrewarding; however, bilateral maxillary sinusitis was evident. Both maxillary sinuses were flushed, and rifampin (5 mg/kg [2.3 mg/lb], PO, q 12 h) was added to the treatment regimen on the basis of the 2 former antibiogram results.

Treatment with SMZ-TMP, rifampin, phenylbutazone, daily flushing of the cervical draining tracts, and twice daily hydrotherapy of the neck were continued. Although the horse gained some weight and had modestly improved attitude, its overall clinical condition slowly deteriorated. The horse had intermittent signs of depression, poor appetite, and continued to have nasal discharge. Because of the persistent multifocal infection with *S. zooepidemicus*, serum immunoglobulin concentrations were measured by use of radial immunodiffusion. Only trace concentrations of IgG and IgM were detected, IgG(T) was undetectable, and IgA was markedly low at 25 mg/dL (reference range, 67 to 239 mg/dL). A diagnosis of acquired immunodeficiency of unknown cause was made, and the owner was informed that the horse had a poor prognosis for survival.

To better characterize the remarkable hypogammaglobulinemia and chronicity of the clinical signs, blood in heparinized tubes and serum samples were shipped overnight for immunologic testing to the College of Veterinary Medicine, Cornell University, Ithaca, NY. Serum samples were tested via protein electrophoresis at the Cornell Diagnostic Laboratory. Results confirmed severe hypogammaglobulinemia and revealed the distribution of the inflammatory proteins, which were within reference ranges despite the inflammatory processes in this patient (Table 1). Most acute-phase proteins are synthesized and stored in the liver in response to inflammatory cytokines released at the site of tissue injury. Although hyperglobulinemia is a common finding even in hepatobiliary diseases, it was possible, although unusual, that liver impairment had resulted in abnormal protein synthesis.

### Table 1—Serum protein concentrations (g/dL) in a horse with combined variable immunodeficiency. Notice the low values for α- and β-globulins, whereas the concentrations of the inflammatory α- and β-globulins are within reference ranges.

<table>
<thead>
<tr>
<th>Protein</th>
<th>Affected horse</th>
<th>Reference range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albumin</td>
<td>2.4</td>
<td>2.3–3.5</td>
</tr>
<tr>
<td>α1</td>
<td>0.33</td>
<td>0.3–0.8</td>
</tr>
<tr>
<td>α2</td>
<td>1.14</td>
<td>0.7–1.3</td>
</tr>
<tr>
<td>Total α</td>
<td>1.47</td>
<td>1.2–1.7</td>
</tr>
<tr>
<td>β1</td>
<td>0.46</td>
<td>0.2–1.1</td>
</tr>
<tr>
<td>β2</td>
<td>0.23</td>
<td>0.3–0.8</td>
</tr>
<tr>
<td>Total β</td>
<td>0.69</td>
<td>0.7–1.6</td>
</tr>
<tr>
<td>γ-globulin</td>
<td>0.14</td>
<td>0.7–1.8</td>
</tr>
<tr>
<td>Total globulin</td>
<td>2.3</td>
<td>2.8–4.7</td>
</tr>
<tr>
<td>Albumin/globulin</td>
<td>1.04</td>
<td>0.6–1.1</td>
</tr>
<tr>
<td>Total protein</td>
<td>4.7</td>
<td>5.7–7.8</td>
</tr>
</tbody>
</table>

### Table 2—Results of flow cytometric analysis of blood lymphocytes (%) from a horse with combined variable immunodeficiency and a healthy control horse

<table>
<thead>
<tr>
<th>Cell surface antigen</th>
<th>Control horse</th>
<th>Affected horse</th>
<th>Antibody</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>B cells</td>
<td>21.1</td>
<td>0.5</td>
<td>Mouse anti-parvovirus</td>
<td>1</td>
</tr>
<tr>
<td>MHC I</td>
<td>99.9</td>
<td>99.8</td>
<td>ELAW II No. 73</td>
<td>3, 4</td>
</tr>
<tr>
<td>MHC II</td>
<td>96.8</td>
<td>73.7</td>
<td>ELAW II No. 43</td>
<td>3</td>
</tr>
<tr>
<td>LFA-1</td>
<td>96.6</td>
<td>99.1</td>
<td>ELAW I No. 45</td>
<td>2</td>
</tr>
<tr>
<td>IgG</td>
<td>17.1</td>
<td>1.2</td>
<td>Goat anti-horse IgG</td>
<td></td>
</tr>
</tbody>
</table>

ELAW I = Equine Leukocyte Antigen Workshop I. ELAW II = Equine Leukocyte Antigen Workshop II. MHC = Major histocompatibility complex. LFA = Leukocyte-function associated antigen. Ig = Immunoglobulin.

At The James A. Baker Institute for Animal Health, blood lymphocytes were isolated from heparinized blood by use of gradient centrifugation and analyzed by use of flow cytometry for lymphocyte antigens with a panel of monoclonal antibodies characterized in the First (ELAW I) and Second (ELAW II) International Workshop on Equine Leukocyte Antigens (Table 2). Samples from healthy horses were processed in parallel to the patient’s samples as controls. Lymphocytes (n = 10) were incubated with each primary monoclonal antibody for 45 minutes at 4°C. A 20-minute blocking step was performed with 10% normal goat serum in buffer solution (0.01% sodium azide and 0.1% bovine serum albumin in phosphate buffered saline [PBS] solution). The secondary stage used was fluorescein isothiocyanate (FITC)-conjugated F(ab') fragment goat anti-mouse IgG (H + L) antibody. Cells were washed 3 times between steps with buffer solution. After staining, the cells were fixed with 2% paraformaldehyde in PBS solution. Samples were analyzed on a flow cytometer equipped with a 488-μm argon laser and computer software. Leukocyte subpopulations were displayed in a dot plot and gated according to size on the basis of forward light scatter and granularity on the basis of 90° side light scatter. A region was placed around lymphocytes, and data were collected on 10,000 gated cells (Table 2). Flow cytometric analysis revealed a high percentage of T lymphocytes.
with a CD4+ cell percentage within reference range and a high percentage of CD8+ cells. No B cells were detected in the patient’s blood with either of 2 independent markers. Expression of major histocompatibility complex (MHC) class I and II antigens and the leukocyte-function associated antigen-1 (LFA-1) were within reference ranges for the lymphocyte population.

To evaluate lymphocyte response to mitogens, lymphocyte proliferation assays were performed with isolated blood lymphocytes from the horse and from a control horse. Serial dilutions of blood lymphocytes were tested in triplicate in 96-well plates. The cells were isolated by use of gradient centrifugation and incubated in medium alone (medium plus 5% normal horse serum) or with the addition of different mitogens (phytohemagglutinin [PHA], 5 µg/mL for testing T-cell response; pokeweed mitogen [PWM], 2.5 µg/mL for testing T- and B-cell responses; and lipopolysaccharide [LPS], 12.5 µg/mL for testing B-cell response) in 5% CO2 at 37°C for 72 hours. Cells were treated with a pulse of 0.8 µCi of [3H]-thymidine/well for the last 8 hours of incubation. Well contents were harvested onto glass fiber filters, and [3H]-thymidine incorporation was measured by use of a liquid scintillation beta counter. No differences were observed between patient and control responses to PHA. Lymphocytes had a weak response to PWM. There was virtually no response to LPS (Fig 1). Lack of response to the B-cell mitogen LPS was consistent with the flow cytometric findings.

At the owner’s request, the horse was discharged 6 weeks after admission with instructions to continue administration of antimicrobials with SMZ-TMP (20 mg/kg, PO, q 12 h) and rifampin (5 mg/kg, PO, q 12 h), anti-inflammatory treatment with phenylbutazone (1 g, PO, q 24 h), and daily topical cleaning of the neck. Before discharge, serum samples were obtained, and the horse was vaccinated with tetanus toxoid to provide a marker of antibody production. Serum samples obtained before and 20 days after vaccination were analyzed for tetanus titers by the National Veterinary Science Laboratories, Ames, Iowa. Results revealed no specific immunoglobulin production, which confirmed the immunologic testing findings.

One month after release, the horse was again admitted to The Ohio State University Veterinary Teaching Hospital because of sudden deterioration of its clinical condition. The horse was extremely weak and hypothermic and had congested mucous membranes and signs of dementia. The cervical cellulitis was clinically resolved. The horse had severe leukocytosis (WBC count, 31,500 cells/µL) and neutrophilia (28,300 cells/µL). Liver enzyme activities were markedly increased (ALP, 2,155 U/L; GGT, 1,150 U/L; AST, 772 U/L; SDH, 36.5 U/L). Differential diagnoses included generalized septicemia and hepatic encephalopathy. On the basis of the clinical condition and for humane reasons, the mare was euthanatized and a necropsy was performed immediately.

Figure 1—Blood lymphocyte proliferation responses (mean ± SD values of triplicate readings of [3H]-thymidine incorporation [counts per minute (CPM)]) to mitogens (phytohemagglutinin [PHA], pokeweed mitogen [PWM], and lipopolysaccharide [LPS]) in cells (x axis [No. of lymphocytes per well]) from a horse with combined variable immunodeficiency (patient) and a healthy control horse. Notice that although no differences were observed in cell responses to PHA between the affected and control horse, lymphocytes from the affected horse had a weaker response to PWM and no response to LPS, which is consistent with the absence of B cells or abnormal B cell response.
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Gross necropsy revealed generalized lymphadenopathy (mandibular, mesenteric, and anterior mediastinal lymph nodes), diffusely firm, dark yellow-green liver, and diffuse fibrous thickening of subcutaneous tissues of the ventral cervical region. The left guttural pouch was unremarkable, and the right guttural pouch had mild focal empyema. Histologically, lymph nodes were characterized by the absence of lymphoid follicles (mediastinal and hilar nodes) and sinusoidal histiocytosis (mesenteric and hilar nodes). Focally extensive, mucopurulent, and suppurative bronchitis with marked fibrosis and lymphocytic, neutrophilic, and mild plasmacytic infiltration were seen in the lung. The liver had widespread micronodular cirrhosis with severe, widespread, dissecting fibrosis, lymphoplasmacytic inflammation, and multifocal nodular hepatocellular hyperplasia.

Fresh tissue samples from peripheral lymph nodes, spleen, and bone marrow were obtained at necropsy, snap frozen in embedding medium for optimal cutting temperature, and shipped to the Baker Institute for immunohistochemical analysis. Five-micron tissue sections were cut with a cryotome and fixed for 10 minutes in acetone and fixed for 10 minutes in acetone and fixed for 10 minutes in acetone and fixed for 10 minutes in acetone and fixed for 10 minutes in acetone. Immunohistochemical labeling was performed in a humidity chamber at room temperature (20°C). Tris buffer saline (TBS) solution was used for washing between steps. Blocking steps included individual 15-minute incubations with 0.1% sodium azide and 0.3% hydrogen peroxide in TBS solution, and 10% normal goat serum and 10% normal horse serum in TBS solution. Primary monoclonal antibody (anti-CD3 [ELAW II No. 98]), CD4 (ELAW II No. 72), CD5 (ELAW I No. 53), CD8 T cells (ELAW I No. 12), B cells (ELAW II No. 73), and IgM on B-cell surface (ELAW II No. 23 and 31), and secondary stage peroxidase-conjugated goat anti-mouse immunoglobulin were separately incubated for 30 minutes each. Substrate solution was prepared with 3-amino-9-ethylcarbazole, and counterstaining was performed with hematoxylin stain. Corresponding tissue specimens collected from a euthanatized, healthy horse were used as controls. The T lymphocyte subpopulations were identified in all tissues from our patient and control horses (Fig 2). No

![Figure 2](image-url)

**Figure 2**—Photomicrographs of bone marrow and lymph node specimens from a horse with common variable immunodeficiency (patient) and a healthy control horse. Cells were stained with a monoclonal antibody against a nonimmunoglobulin marker to detect B cells, a monoclonal antibody against surface IgM on B cells, and a monoclonal antibody against CD3+ T cells. Notice that no stained cells were detected in the affected horse’s bone marrow with the reagents for detection of B cells, compared with cells from the healthy control horse, whereas T cells were detected in all specimens. Occasional stained cells were detected in the affected horse’s lymph node with the 2 B-cell markers, and T cells were numerous. Notice the absence of a B-cell follicle in the affected horse’s lymph node. Immunohistochemical stains; bar = 20 µm.
positively stained cells were detected in the patient’s bone marrow or spleen with the B cell and surface IgM monoclonal antibodies, compared with positively stained cells in the control specimens. Occasional clustered, positively stained cells were found in the patient’s lymph nodes with anti-B cell and anti-IgM antibodies; however, those were uncommon, compared with the number found in the control specimen. The absence of B cells greatly affected the lymph node structure, which lacked the typical cortical (B cell)-paracortical (T cell) cell distribution, and the primary and secondary follicles (germinal centers) in the cortex. Instead, there was diffuse distribution of T cells throughout the node. Similar abnormalities were observed in the spleen, with absence of the expected B cells. Both findings were in agreement with the histopathologic findings.

Antibody deficiency is the most commonly diagnosed primary immunodeficiency in mammals. It is characterized by full or partial failure of B-cell development and differentiation into plasma cells that produce different immunoglobulin isotypes. Agammaglobulinemia with lack of mature B cells and plasma cells has been reported in genetically defined severe combined immunodeficiency in horses and in young male horses of various breeds. In the latter, clinical manifestations include bacterial infection of respiratory, digestive, and integumentary systems. The clinical manifestations include bacterial infection of respiratory, digestive, and integumentary systems. The syndrome in male horses appears similar to the X-linked agammaglobulinemia of male human patients and X-linked immunodeficiency mice. Affected humans and genetically modified mice carry a mutation of the btk gene on the X chromosome, which encodes Bruton tyrosine kinase. The absence of this protein affects sustained signaling in response to B cell receptor engagement, leading to defects in B-cell differentiation and proliferation. Typical X-linked agammaglobulinemia patients have recurrent bacterial respiratory infections early in life, the absence of B cells and plasma cells, and also have hypo- or agammaglobulinemia. Female carriers are healthy; nevertheless, they may have nonrandom X-chromosome inactivation in their B cells. Although the btk gene has not been mapped in horses and btk mutations have not been described in reports of affected horses, it is unlikely that the horse in this study carried this primary genetic mutation, because the horse was a mare and the clinical signs appeared late in life. The immunodeficiency of this horse is better classified as a type of common variable immunodeficiency (CVID).

Common variable immunodeficiency is a clinical primary antibody deficiency syndrome characterized by recurrent bacterial infection and impaired humoral response to vaccination. In affected humans, recurrent pyogenic sinopulmonary infection is common, and hepatobiliary disease that progresses to cirrhosis and death is a frequent complication. Affected humans are primarily susceptible to infections because of a combination of the humoral immunodeficiency and exposure to common pathogens. Although hepatobiliary conditions of unknown cause have been reported, hepatitis C virus, likely acquired during plasma transfusions, may contribute to more severe progression of hepatic disease in immunodeficient humans. In the horse reported here, respiratory and hepatobiliary systems were both affected and contributed to a fatal outcome. Similar to human patients, the cause of the severe liver disease in this horse was not apparent. The lesions were nonspecific and chronic, and their relationship to the underlying immunodeficiency was unknown. Bacteriologic culture of the liver before euthanasia might have been helpful in determining whether the streptococcal infection also involved the liver. However, the lesions detected at necropsy were not consistent with bacterial infection.

In humans, the incidence of this disorder is greater during the second and third decades of life. Both sexes are affected equally, and isolated and familial cases have been reported. Classification of CVID is complex because of its variable immunologic and clinical phenotypes. A method for classification of this heterogeneous disorder made on the basis of in vitro immunologic responses of purified B cells to sepharose-bound antibodies against surface IgM and interleukin-2 has been developed. Four groups of patients can be identified on the basis of the absence or presence of B cells. Our patient represented the type that involved the absence of B cells; therefore, no additional in vitro tests were possible.

To our knowledge, CVID with absence of B cells in blood, lymphoid tissues, and bone marrow has not been reported in adult horses. In domestic animals, there is 1 report of CVID in miniature Dachshunds infected with Neospora caninum and with altered B and T cell responses. Selective immunoglobulin deficiency with abnormal B-cell response has been reported in horses. In 1 case, a 10-month-old Arabian filly with recurrent gastrointestinal tract disease and syphilitis had undetectable serum concentrations of IgM, IgA, and IgG(T); typical concentrations of IgG, decreased response of B cells to LPS stimulation; and normal T cell response to concanavalin A. Another case involved a 3-month-old colt with recurrent respiratory disease, T- and B-cell blood counts within reference ranges, decreased B-cell response to LPS, and IgM deficiency. In addition, a 3-year-old colt with chronic diarrhea was reported to have hypogammaglobulinemia for IgG, IgA, and IgM; B cell lymphopenia; and poor response of T cells to stimulation with mitogens. These reports might have represented variations of CVID, and additional in vitro assays could have assisted in classification of the disease.

Immunologic testing was critical in identifying the immunodeficiency in the horse reported here. Abnormal B-cell development appears to have been the major cause of the severe hypoglobulinemia and immune system failure, which allowed a fatal septicemia to develop progressively. The immunohistochecmic results indicated that mature B cells were absent in the bone marrow, spleen, and blood, but were occasionally seen in lymph nodes by use of a monoclonal antibody directed against a nonimmunoglobulin antigen on the B-cell surface and others directed against equine IgM (tissue sections) or IgG (blood) expressed on cell surfaces. Although the monoclonal antibodies against IgM have not been specifically char-
acterized for immature B cells (expressing IgM on cell surface) and pre-B cells (expressing cytoplasmic μ chain) in the bone marrow, it is likely that they would recognize those cells. The absence of positively stained cells in bone marrow suggested the absence of immature and pre-B cells in this horse and, therefore, that the defect happened in the early stages of B-cell development. In addition, the presence of a few B cells in the lymph nodes may suggest that, originally, this mare may have had adequate B-cell development to populate secondary lymphoid organs. However, the progressive impairment of B-cell development may have led to insufficient supply of these cells, and few survived in the lymph nodes.

Primary B-cell defects in patients with CVID may result from distinct abnormalities in B-cell differentiation stages, resulting in corresponding abnormal expression of immunoglobulin genes and proteins. Described mutations in autosomal genes encoding early B-cell development in humans include the μ heavy chain, $\lambda_5/14$ surrogate light chain genes, the B cell receptor signaling molecule Igx, and the B-cell linker adapter protein. When B cells are present in the system, deficiencies of individual subclasses of immunoglobulins may result from isolated mutations in immunoglobulin heavy-chain genes, which lead to abnormal differentiation into plasma cells and isotype switching. In addition, T cell co-stimulation of B cells can be impaired by T cell lymphopenia, abnormal cytokine secretion, proliferation, and co-stimulatory signaling. Therefore, it is possible that other compromized segments of the immune system in patients with CVID lead to hypo- or agammaglobulinemia and a more generalized immunodeficiency.

Potential treatment of immunoglobulin deficiencies in horses is based on continuous antimicrobial treatment and replacement of immunoglobulins by use of IV administration of plasma or infusion of immunoglobulin preparations to maintain a minimum serum IgG concentration of 500 mg/dL. Subcutaneous administration of IgG provides serum IgG concentrations that are comparable to IV infusions in human patients, and this route of administration is indicated in patients that develop anaphylactoid reactions when administration is by the IV route. However, long-term plasma administration in horses is economically impractical. Bone marrow transplantation for regeneration of healthy B-cell precursors in lymphoblastic leukemia patients has been reported with limited but promising results in human patients.

It is not clear whether the horse reported here had a late-onset form of B-cell deficiency or whether a pre-existing marginal B-cell impairment became more profound as a result of unidentified factors. Identification and characterization of immunodeficiencies in horses have improved during the last decade, and accumulation of data may reveal specific diseases and genetic abnormalities of the immune system.

References


