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CASE REPORT

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When *E. coli* strikes: a necropsy analysis of a juvenile giraffe's fatal infection

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Abstract

Background As bacterial infections pose a major health risk to captive populations, disease prevention and management play a crucial role in the ex situ conservation of giraffes (*Giraffa camelopardalis*). This study describes the case of a giraffe that developed septicemia after an umbilical cord infection caused by *Escherichia coli*. To our knowledge, pathological changes in diseased giraffes caused by *E. coli*, which is an opportunistic pathogenic organism, have not been reported. This is the first report presenting an analysis of necropsies and subsequent microbiological investigations.

Case presentation The baby giraffe's mother died shortly after birth, so it had to be fed milk powder. The giraffe was healthy at first but developed symptoms like depression, loss of appetite, and lameness at 8 days old. At 14 days of age, the juvenile giraffe showed astasia and gradually died, with a disease course of 7 days. Postmortem examination revealed opisthotonus and navel swelling. Serofibrinous arthritis, serofibrinous necrotizing inflammation of periarticular soft tissue, serous omphalitis, and severe adventitia hemorrhage of the umbilical artery were observed. Severe serofibrinous pericarditis, pleuritis, and peritonitis were also observed. The interstitium of the pulmonary lobule widened because it was filled with a pale yellow translucent gelatinous exudate. Histopathologically, the calf had diffuse serous interstitial pneumonia, serous necrotizing umbilical arteritis, degenerative hepatitis with mild fibrosis, degenerative nephritis, hemorrhagic lymphadenitis, necrotizing enteritis, and necrotizing thyroiditis. Blue-stained clumps of bacteria of varying sizes and neutrophil infiltration were scattered or diffused in the interstitial connective tissue and edematous serosa of all tissues and organs, as well as in small vessels and lymphatic vessels, which were filled with many neutrophils (lymphatic spread). Single gram-negative *Escherichia coli* were cultured from all tissues of the animal. Polymerase chain reaction results of 16S rRNA of the isolated *Escherichia coli* had 99.79% homology to KJB03889.1.

Conclusions The gross, histopathologic, microscopic, and polymerase chain reaction sequencing features reported in a juvenile giraffe were consistent with colibacillosis, which is a rare disease of giraffes. The gross, histopathologic, microscopic, and polymerase chain reaction sequencing features reported in a juvenile giraffe. This case serves as a paradigmatic illustration of a giraffe suffering from neglect and inadequate treatment, leading to severe consequences. In instances of giraffe Escherichia coli septicemia, it is imperative to thoroughly assess for underlying diseases, particularly in the absence of obvious predisposing factors. The rise of multidrug resistant organisms

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has constrained the efficacy of empirical antibiotic treatment, highlighting the importance of promptly conducting culture and sensitivity testing and employing antibiotic therapy guided by susceptibility results.

Keywords Giraffe, Colibacillosis, Escherichia coli, Pathology, 16S rRNA

Background

Escherichia coli (*E. coli*) is the one of prevalent gram-negative species. The following three broad categories of *E. coli* strains are of biological significance to mammals: commensal, intestinal pathogenic (InPEC), and extraintestinal pathogenic (ExPEC) [1, 2]. Although *E. coli* is a benign commensal colonizing the mammalian intestine, some strains or pathotypes can cause a variety of intestinal and diarrheal disorders [3]. For example, a minimum of the following six pathotypes have been described: enterohemorrhagic, enteropathogenic, enterotoxigenic, enteroaggregative, diffusely adherent, and enteroinvasive *E. coli*, respectively [3]. Moreover, ExPEC can cause diseases such as urinary tract infections, bacteremia, septicemia, and meningitis [3–6]. It is unclear how E. coli genetic diversity, virulence, and antimicrobial resistance affect biodiversity and wild animal conservation [7]. Wild animals may get exposed to antimicrobial compounds and antimicrobial resistance bacteria by interaction with anthropogenic sources such as human waste (garbage and sewage) and polluted waterways [8–10], livestock activities [11, 12], or predation on impacted prey, including livestock corpses [13, 14].

Giraffes (*Giraffa camelopardalis*) are the tallest living animals and are kept in many zoos worldwide. Despite the passionate interest in keeping captive giraffes healthy, the health management of the giraffe presents a significant challenge. Despite being routinely bred in zoos, giraffes continue to provide a problem, particularly when it comes to food. Because of the high risk of maternal rejection and death among both mother-reared and hand-reared calves [15, 16]. Although success rates have increased over time, intensive care therapy of compromised calves remains under documented [15]. There are still no definitive feeding standards, predicted weight increase, or suggestions for veterinarian assistance. In addition, little research has been conducted on diseases affecting giraffes, which are primarily associated with its hoofs and musculoskeletal system [17, 18]. However, there are few reports of *E. coli* disease in young giraffes.

ExPEC infections are a serious threat to public health worldwide [19, 20]. Urinary tract infections, severe newborn meningitis, major intra-abdominal infections, and, less frequently, pneumonia, intravascular device infections, osteomyelitis, soft tissue infections, or bacteremia are the most troublesome illnesses. Bacteremia can result in sepsis, which is defined as life-threatening organ dysfunction caused by an unregulated immune response to infection [5]. In this study, we describe the case of a giraffe that developed septicemia after an umbilical cord infection caused by E. coli. This case study may serve as a valuable reference and caution for veterinarians in zoos.

Case presentation

Clinical history

A female giraffe's mother died of severe trauma approximately 5 h after delivery; hence, the juvenile giraffe could not feed colostrum and had to be artificially administered milk powder (Holstein milk + 10% colostrum). The juvenile giraffe was able to stand on its own 3 days after birth and was in a good condition. However, on the eight day after birth, the juvenile giraffe began to show clinical signs of losing appetite, slow walking, and depression. Lactasin (Lactaid®, Johnson & Johnson Inc., Guelp, Canada; Take 3 caplets with their bite of daily food.) was administered orally twice a day for 4 days during the course of the disease, and the treatment was ineffective. On the 12th day after birth, the juvenile giraffe showed anorexia, tarsal joint swelling of the right hind limb, claudication, unwillingness to move, the presence of a small amount of dirty yellow loose stool around the anus, and eventually lying down, and died on the 14th day after birth.

Necropsy

A postmortem examination was performed within 2 h of the animal's death. According to the naked eye observation, dark, red, and swollen umbilicus (Fig. 1A); and a small amount of dirty yellow sticky feces on the perianal coat. Serofibrinous arthritis and periarticular serous necrotizing inflammation: the swollen hock joint of the hind limb and the subcutaneous tissue near it was light yellow gelatinous material due to inflammatory edema, and the local skin is attached to the subcutaneous tissue and muscle (Fig. 1B). A cystic necrotic focus was formed at the adhesion site, with a red inflammatory response zone at the margin and yellow necrotic tissue in the central area. A large amount of pale yellow translucent inflammatory fluid and yellow flocculent fibrinous exudate accumulated in the joint cavity of the wrist, hock, and hip joints (Fig. 1C). Serous omphicitis with severe gelatinous swelling of the umbilical pore was obvious. The umbilical veins and bilateral umbilical arteries were

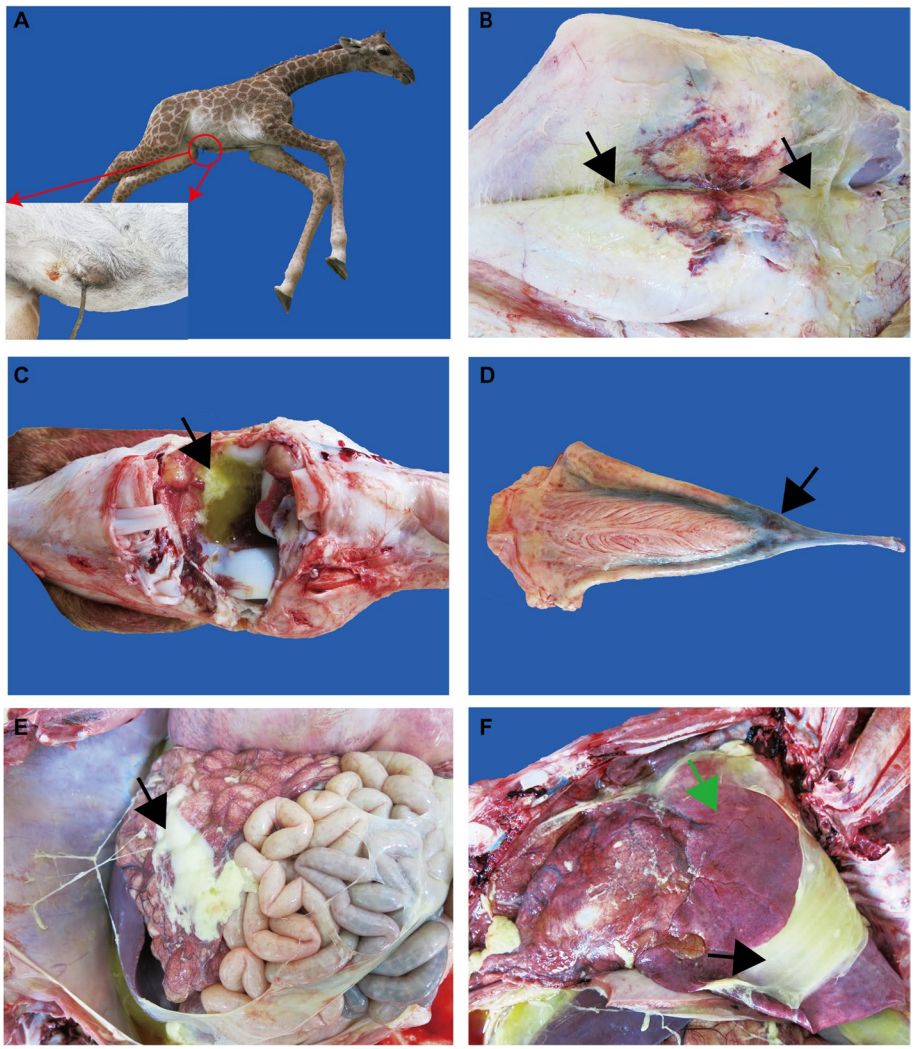

Fig. 1 Gross pathology findings. **A** marked bulging in the umbilical region; **B** umbilical arteritis; The black arrow represents the swollen hock joint of the hind limb and the subcutaneous tissue near it was light yellow gelatinous material due to inflammatory edema. **C** the knee joint of the right hind leg (edema and focal necrosis); The black arrow represents a large amount of pale yellow translucent inflammatory fluid and yellow flocculent fibrinous exudate accumulated in the joint cavity. **D** the umbilical arteries; The black arrow represents the umbilical arteries were full of dirty dark red necrosis. **E** fibrinous peritonitis; The black arrow represents yellow white flocculent fibrinous exudates in the abdominal cavities. **F** fibrinous pleurisy; The black arrow represents yellow white flocculent fibrinous exudates in the chest. The green arrow represents the lungs were enlarged, dark red in color

thickened significantly, with black and red adventitia and gelatinous edema of the surrounding connective tissue. The umbilical arteries were full of dirty dark red necrosis, and the intima was rough (Fig. 1D).

Severe serofibrinous pericarditis, pleuritis, and peritonitis: A large amount of pale-yellow translucent fluid and yellow white flocculent fibrinous exudates in the pericardial, chest, and abdominal cavities, and slight adhesion of the local serous membrane were observed (Fig. 1E and F). The kidneys and liver were swollen and dark red, with moist and glossy surfaces, and the submucosa of the renal pelvis was thickened and showed yellowish gelatinous edema. The lungs were enlarged, dark red in color, covered with flocculent fibrinous exudates, and the interstitium of the pulmonary lobule was generally widened and full of yellow translucent gelatinous exudate (Fig. 2A). The transverse diameter of the heart was significantly widened, and the epicardial membrane was attached to a flocculent yellowish-white fibrinous exudate. Hyperemia and edema of the abomasum mucosa and intestinal pneumatosis were observed.

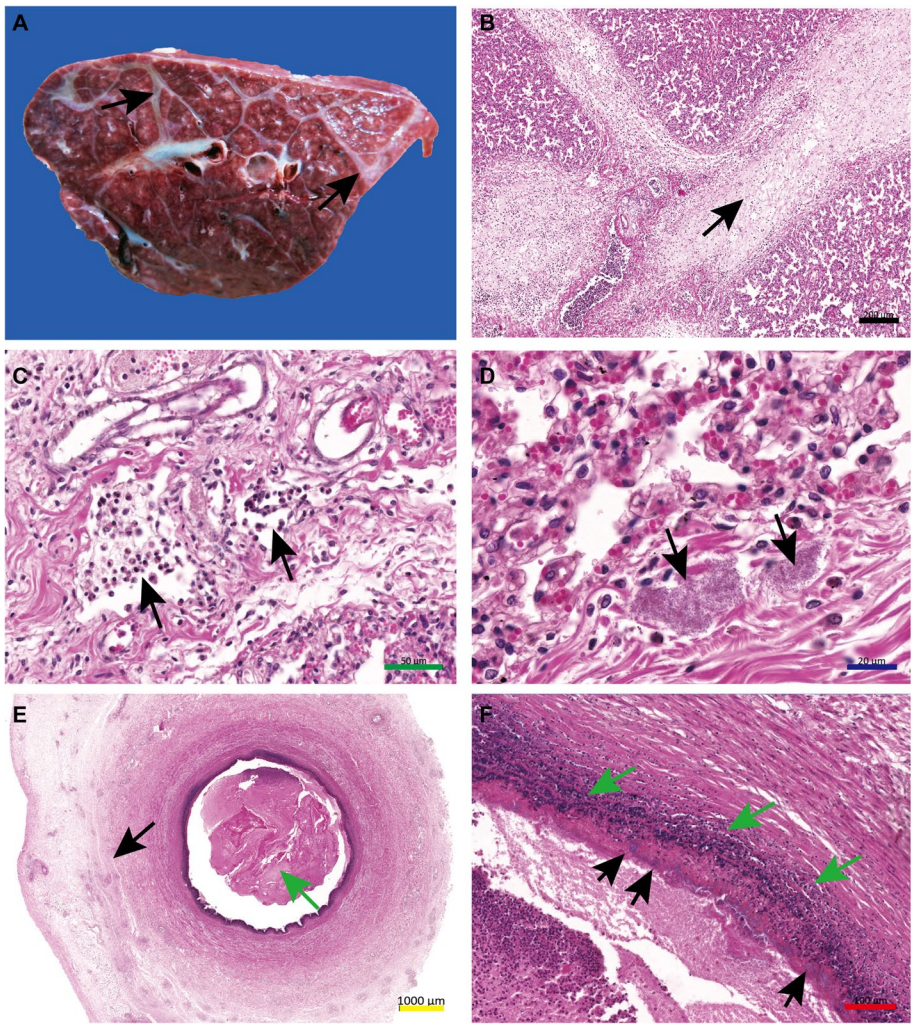

Fig. 2 Histopathological findings. **A** serous interstitial pneumonia; The black arrow represents the interstitium of the pulmonary lobule was generally widened. **B** microscopy showing distinct widening of the alveolar septum, filled with massive neutrophils (black bar = 200 μm); Arrowhead shows position of the alveolar septum. **C** the accumulation of neutrophils in pulmonary vessels (green bar = 50 μm); Arrowhead shows position of the neutrophils. **D** hematoxylin–eosin staining of pulmonary interstitium intravascular bacterial clumps (blue bar = 20 μm); Arrowhead shows position of the bacterial clumps. **E** necrotizing umbilical arteritis (yellow bar = 1000 μm); The black arrow indicates the position of the tunica adventitia of the umbilical artery, and the green arrow indicate the pink serous fluid positions. **F** diffuse infiltration of neutrophils in umbilical artery; bacterial clumps were observed abundantly (red bar = 100 μm); The black arrow indicates the position of bacterial clumps, and the green arrow indicate the neutrophils positions

Histopathology

Serous interstitial pneumonia and lobular interstitial pneumonia were significantly widened and filled with homogeneous pink stained serous fluid (Fig. 2A). A small amount of fibrous protein, diffused neutrophils, scattered or clustered small blue bacilli, and a large number of neutrophils within lymphatic vessels at all levels were observed (Fig. 2B). Pulmonary hyperemia and sporadic serous fluid, erythrocytes, and neutrophils were found in the alveolar and bronchial lumens near the lobular interstitium (Fig. 2C and D). Serous necrotizing umbilical arteritis with hyperemia, edema, and marked thickening of the tunica adventitia of the umbilical artery filled with homogeneous pink serous fluid, scattered or diffused infiltrating neutrophils, and scattered or clustered small blue-stained bacilli were observed (Fig. 2E and F). Necrosis of the tunica intima and partial tunica media with diffused neutrophils and increased blue-stained bacterial clusters of varying sizes were observed; there was a large amount of serous fluid, necrotic neutrophils, and erythrocytes in the lumen of the artery (Fig. 2F). Mild hepatic sclerosis: hepatic interstitial connective

tissue proliferated and widened mildly, with small bile duct increase; liver edema, obvious Disse space, incomplete wall of hepatic sinusoid, hemolysis, and hepatocytes separated from each other were seen. Mild steatosis and scattered necrosis of hepatocytes in the central area of the hepatic lobule were observed. Renal hyperemia and edema, mild to moderate cell swelling of the renal tubular epithelia, occasional necrosis of the renal tubular epithelia in some renal tubules, and increased neutrophil content in the pelvis were observed. Hyperemia and edema, loose capsules with scattered infiltrating neutrophils, and cells in the zona fasciculata separated from each other were observed in the adrenal glands. Lymphocyte reduction, fewer lymph nodules with inconspicuous germinal centers, and diffuse hemorrhage of the medulla were observed in the lymph nodes. Hyperemia and edema, significantly reduced lymphocytes, white pulp lymphocyte nodules with sparse lymphocytes of white pulp were observed in the spleen. Mild to moderate cellular swelling of cardiomyocytes was observed. Serous necrotizing enteritis: significant edema and thickening of the small intestine wall, large amount of serous fluid, diffuse infiltrating neutrophils, and necrotic mucosal layer were observed in the small intestine. The marginal acinar epithelial cells of the thyroid gland were partially necrotic. Blue-stained bacterial clusters of varying sizes or diffuse blue-stained small bacilli were present in the interstitium and serous membranes of most tissues and organs as well

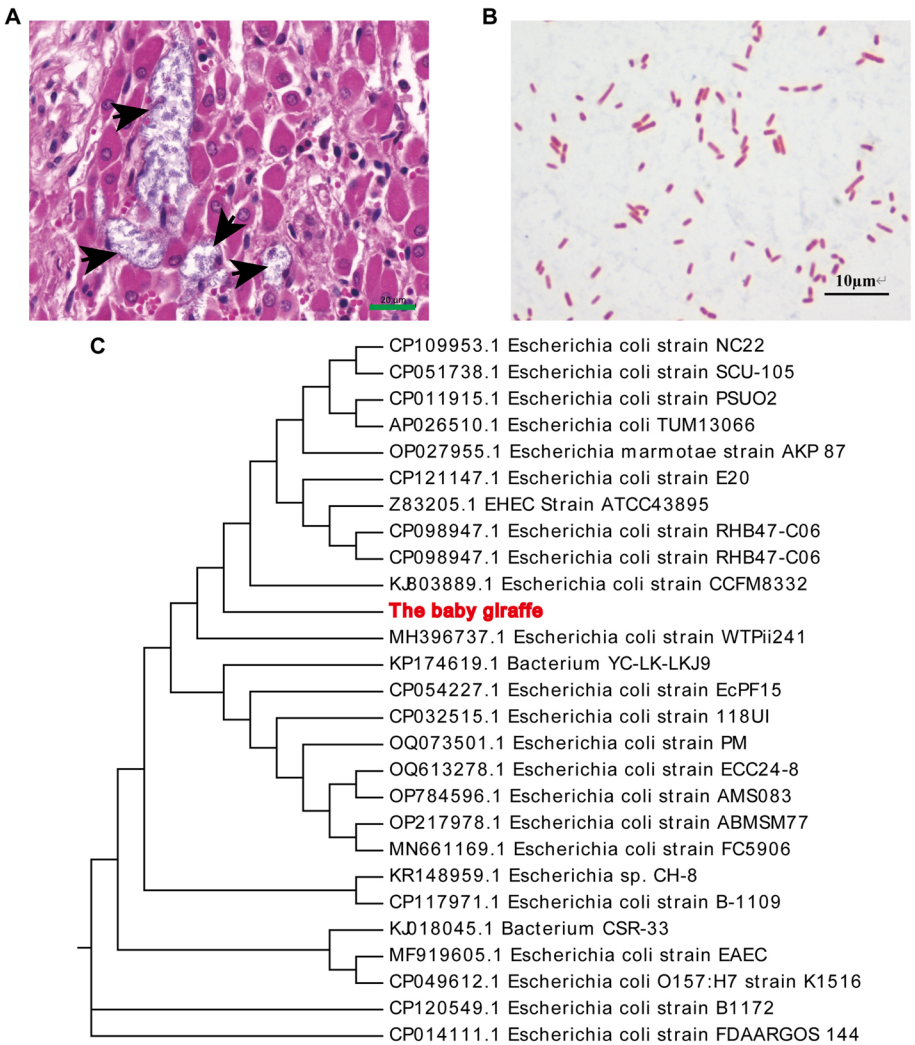

Fig. 3 Morphology of the *Escherichia coli* isolate analyzed using staining. **A** adrenal glands; bacterial clumps (black arrowhead) are observed abundantly (green bar = 20 μm). **B** Gram staining of *Escherichia coli*. The Gram stain of a positive culture demonstrates gram-negative bacilli on microscopy (× 100) (Black bar = 20 μm). **C** Phylogenetic analysis of 16S rRNA sequences of the isolated strain. The tree was constructed using the neighbor-joining method with 1000 replicates for bootstrap values using MEGA 6

as in small blood vessels and lymphatic vessels (Fig. 3A). This was accompanied by scattered or diffuse infiltrating neutrophils, particularly in the lymphatic vessels of tissues filled with neutrophils (lymphatic spread). The endothelial cells separated severely from the media of the small vessels because of edema.

Bacterial isolation and molecular identification

Pleural fluid, pericardial exudate, ascites, joint fluid, lung, liver, and umbilical artery wall were aseptically collected with an inoculation loop and inoculated on MacConkey and eosin-methylene blue (EMB) medium and cultivated at 37 °C for 24 h. Many small pink colonies grew on the MacConkey medium. The EMB medium grew many small, round, shiny black colonies characteristic of *E. coli*. Using an inoculation loop, a small amount of the organism was collected to prepare a smear. Simple gram-negative small rods having the same morphology as that of *E. coli* were detected using Gram staining (Fig. 3B).

In this study, the 16S rRNA of the cultured bacteria was sequenced. We selected ten colonies from each plate (total 70 colonies) for polymerase chain reaction (PCR) detection and sequencing. General primer sets (10Fx:5′-AGAGTTTGATCCTGGCTCAG-3′; 1509R:5′-GTTACCTTGTTACGACTTCAC-3′) were selected to amplify the 16S rRNA from all the colonies isolated from the baby giraffe samples [12, 13]. For amplification, the following conditions were used: initial denaturation at 95 °C for 3 min; 30 cycles of denaturation (30 s at 94 °C), annealing (30 s at 55 °C), extension (1.5 min at 72 °C), and final extension at 72 °C for 5 min. The amplified PCR products were analyzed on 1.5% agarose gels, purified, and sequenced. Through BLAST searches, the sequences were compared with those in the NCBI database. The results indicated that all the 70 colonies were of *E. coli*; they also revealed a nucleotide sequence similarity of 99.16–99.79% to strains from human feces (CCFM8332), Yuncheng Salt Lake (YC-LK-LKJ9), poultry droppings (AKP_87), marine (CSR-33, CSR-59), wetland (CH-8), and wastewater treatment plant (WTPii241) (Fig. 3C).

The phylogenetic groups of E. coli isolates were identified using a PCR-based method developed by Clermont et al. E. coli was classified into four main phylogenetic groups (A, B1, B2, and D) based on the presence of three markers (chuA, yjaA, and TSPE4.C2) in their DNA. Crude DNA was extracted from colonies by lysing them in sterile water at 100 °C for 15 min, followed by centrifugation. The lysis supernatant was utilized for the polymerase chain reaction, following the conditions outlined by Clermont et al. [21]. The primers utilized in this investigation are detailed in Supplementary Table 1. PCR analysis of the isolate indicated its classification within phylogenetic group B1 (Fig. 4A). A total of twenty-five virulence genes were identified, including PAI, *pap*A, *fm*H, *kps*MT III, *pap*EF, *ibe*A, *fyu*A, *bma*E, *sfa*/*foc*DE, *iut*A, *pap*G allele III, *hly*A, *rfc*, *nfa*E, *pap*G allele I, *kps*MT II, *pap*C, *gaf*D, *cva*C, *foc*G, *tra*T, *pap*G allele I, *pap*G allele II, *afa*/*dra*BC, *cnf*1, and *sfas*. Each virulence gene was amplified using specific primers in PCR. The primers utilized in this investigation are detailed in Supplementary Table 1. Thermal cycling conditions included an initial denaturation cycle at 94 °C for 2 min, followed by 35 cycles at 94 °C for 1 min, annealing at a specific temperature for 1 min, and extension at 72 °C for 1 min, with a final cycle at 72 °C for 2 min. In this strain, 6 virulence genes (PAI, *iut*A, *pap*G allele III, *cva*C, *sfas*, *afa*/*dra*BC) associated with adhesion, toxicity, and environmental response were identified (Fig. 4B). E. coli strains were tested for antibiotic susceptibility using CLSI guidelines and a disc diffusion method with 16 antibiotics [22]. The resistance profiles of the E. coli strains to the antibiotics tested are outlined in Table 1, with interpretation of all susceptibility results based on the CLSI guidelines [22]. The strains exhibited resistance to ceftazidime, ceftriaxone, ciprofloxacin, levofloxacin, amoxicillin, and azithromycin, while demonstrating susceptibility to penicillin, oxacillin, lincomycin, clindamycin, ampicillin, and cotrimoxazole.

Discussion and conclusions

Among neonatal hand-reared giraffes, failure of passive transfer of immunity (FPI) continues to be a problem [16]. The cotyledonary placentas in giraffes transfer negligible antibodies. Therefore, newborns rely on colostrum consumption and the absorption of maternal antibodies across the intestines during the first 24–48 h after birth [16, 23, 24]. FPI increases the risk of diarrhea, enteritis, septicemia, arthritis, omphalitis, and pneumonia in domestic ungulates [25–28]. Passive immunity transfer during the newborn's first week is crucial for the successful rearing of ruminant neonates.

To ensure optimal and steady growth, milk replacers must have a composition similar to that of giraffe milk. Bovine milk and colostrum have been effectively utilized and advised for hand-rearing giraffes despite the lower fat and protein contents of cow's milk and milk substitutes than that of giraffe milk [16]. Until the regular consumption of solid food, milk should be consumed daily in amounts of 7–10% of the body weight (19,000–25,000 kcal/day) [16]. A hand-fed giraffe calf (which did not receive colostrum) died of septicemia caused by *E. coli* in the present study. Septic arthritis and phlegmon are caused by trauma or systemic infection. No trauma was recorded in this giraffe pup. Therefore, systemic infection may have contributed to the septic polyarthritis and/or phlegmon observed in this study. Enteritis,

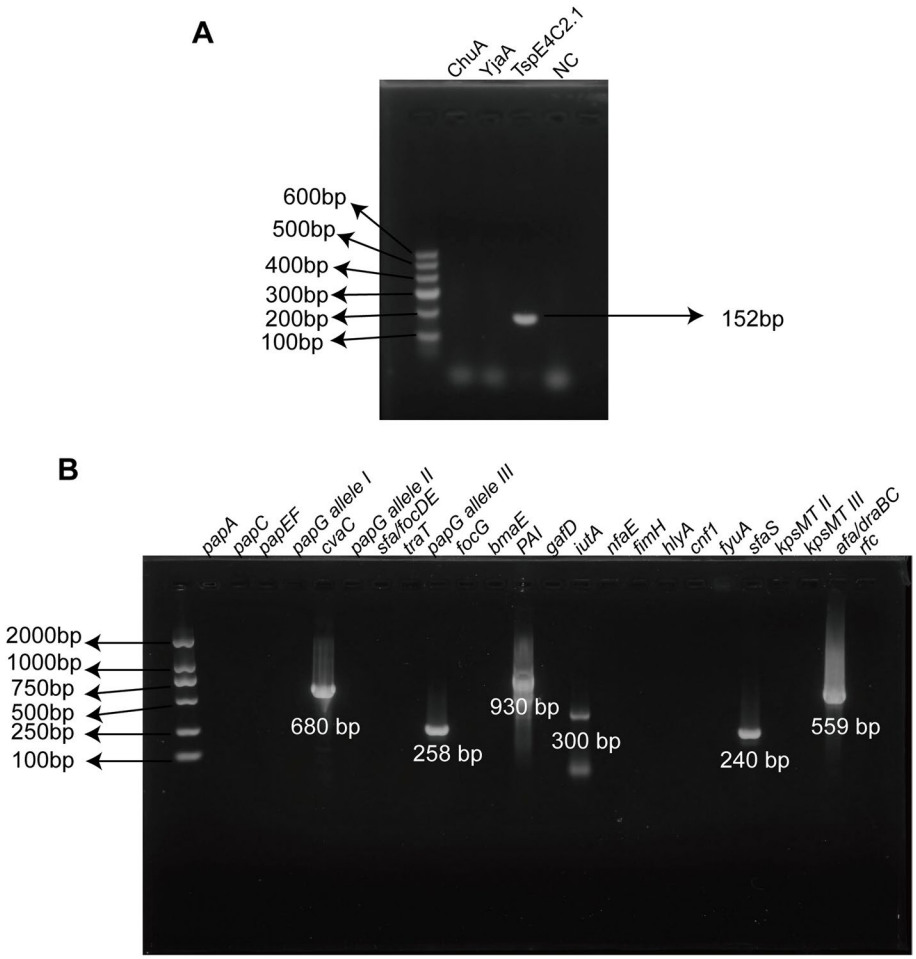

Fig. 4 The gel electrophoresis images of PCR product of the phylogenetic groups and virulence genes. **A** The PCR profiles were specific to the phylogenetic groups of *E. coli*. The determination of a strain's phylogenetic group was achieved through the amplification of the *chu*A and *yja*A genes, as well as the DNA fragment TSPE4.C2. The negative control was included in Lane NC. **B** PCR was used to detect virulence genes

pneumonia, and funisitis are common sources of infection in giraffe calves; enteritis and pneumonia were not recorded in giraffe calves before the development of arthritis [15, 29, 30]. Furthermore, the lack of immunocompetence might have put the calves at a risk of the infection spreading systemically through the umbilical cord. Septic polyarthritis and/or phlegmon may be caused by systemic infection. A PCR and sequence analysis confirmed that *E. coli* was the cause of bacteremia in the present case.

E. coli colonizes newborn pups' gastrointestinal tract shortly after birth and typically coexists with its host without causing disease. However, certain strains with specific virulence attributes can cause a range of illnesses in immunocompromised hosts or when gastrointestinal barriers are compromised. Extraintestinal pathogenic *E. coli* (ExPEC) are characterized primarily by their site of isolation, with the most clinically significant groups being uropathogenic *E. coli* (UPEC), neonatal meningitis-associated *E. coli* (NMEC), avian pathogenic *E. coli* (APEC), and septicemic *E. coli* (SEPEC) [29, 31]. ExPEC strains have the ability to cause infections in various extraintestinal locations. In the present case, the ExPEC strain resulted in pneumonia, umbilical arteritis, hepatitis, nephritis, hemorrhagic lymphadenitis, necrotizing enteritis, and necrotizing thyroiditis in the baby giraffe. There is no doubt that this is a direct result of E. coli bacteremia. In order to initiate bacteremia, the ExPEC strain must successfully infiltrate initial sites of infection or colonization, disseminate throughout the bloodstream, and persist within the blood. Nevertheless, the ExPEC strain has the capability to access the bloodstream through various pathways. Bacteremia lacking a discernible origin is classified as primary, while secondary bacteremia may result from dissemination originating from an existing infection, such as pneumonia or urinary tract

Table 1 Antimicrobial susceptibility profile of the *E. coli*

Antimicrobial agent (disc content)	Antibiotic Susceptibility
Penicillin G	Resistant
Ampicillin	Resistant
Amoxicillin/clavulanic acid	Sensitive
Ceftazidime	Sensitive
Ceftriaxone	Sensitive
Ciprofloxacin	Sensitive
Levofloxacin	Sensitive
Erythromycin	Intermediate
Azithromycin	Sensitive
Clindamycin	Resistant
Gentamicin	Sensitive
Levofloxacin	Sensitive
Tetracycline	Intermediate
Oxacillin	Resistant
Lincomycin	Resistant
cotrimoxazole	Resistant

infections, or from contaminated medical equipment [5]. In this case, however, the bacteremia was likely a result of an umbilical cord infection. Improper handling of the umbilical cord presents a potential risk of infection, as it serves as a significant entry point for pathogens in newborns. Therefore, it is strongly advised that veterinarians adhere to proper disinfection, sterilization, isolation, and other cleaning protocols to ensure optimal umbilical cord hygiene when handling neonates.

ExPEC uses various factors to cause disease in animals, including adhesins, invasins, protectins, iron acquisition systems, and toxins [32, 33]. These factors help ExPEC adhere, invade, evade the immune system, colonize, proliferate, and spread throughout the body, leading to infection in animals [33, 34]. Other bacterial factors such as secretion systems, quorum sensing systems, transcriptional regulators, and two-component systems also play a role in ExPEC pathogenesis [35–37]. In this study, the virulotyping revealed that the *E. coli* strain was positive for PAI, *iut*A, *pap*G allele III, *cva*C, *sfa*s, and *afa*/*dra*BC. Adhesins are bacterial components that help them stick to other cells or surfaces, increasing their virulence. Specific adhesins are adapted to colonize different environments. Virulence genes linked to adhesion include *pap*G allele III, *sfas*, and *afa*/*dra*BC. Iron is a crucial micronutrient necessary for the growth and proliferation of bacteria within the host following successful colonization and/or invasion. Among the most significant virulence plasmids associated with ExPEC virulence are ColV and ColBM, particularly those containing the aerobactin operon (*iut*A/*iuc*ABCD). This operon codes for high-affinity iron-transport systems that enable bacteria to acquire iron in low-iron environments, such as those found in host fluids and tissues. Our isolates carrying virulence genes were found to possess the *iut*A gene, which facilitates survival in low iron conditions.

Antibiotics are commonly utilized for the prevention and treatment of ExPEC infections. However, the widespread use of antibiotics has been linked to the development of multidrug-resistant bacteria. The high levels of antibiotic resistance observed in ExPEC strains present a significant risk to human health, as antibiotic-resistant bacteria and genes can be transmitted through the food chain. Previous research has shown that ExPEC isolates exhibit resistance to multiple antibiotics [38, 39], underscoring the importance of conducting antibiotic susceptibility testing to identify the most effective treatment option. In this particular instance, the *E. coli* strain exhibited broad-spectrum beta-lactamase production. β-Lactam antibiotics, particularly 3rd generation cephalosporins, are commonly prescribed for the treatment of serious community-onset or hospital-acquired infections caused by *E. coli*. Regrettably, β-lactamase production in E. coli continues to be a significant factor in the development of resistance to β-lactam antibiotics [33, 40]. β-lactamases are bacterial enzymes that render β-lactam antibiotics ineffective through hydrolysis.

This study presents findings on septic polyarthritis and/or septicemia in juvenile giraffes, potentially attributed to insufficient colostrum intake and *E. coli* infection via the umbilical cord. Furthermore, the study elucidates the diverse array of virulence factors exhibited by the *E. coli* strain and underscores the pathogenic significance of these pathogens in animal health. Continued research is warranted to identify additional virulence factors and elucidate the pathogenic mechanisms, ultimately aiding in the development of an effective diagnosis and treatment strategy for managing giraffe colibacillosis.

Abbreviations

E. coli	*Escherichia coli*
EIEC	Enteroinvasive *E. coli*
EMB medium	Eosin-methylene blue medium
ExPEC	Extraintestinal Pathogenic
FPI	Failure of Passive Transfer of Immunity
InPEC	Intestinal Pathogenic
PCR	Polymerase chain reaction

Supplementary Information

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Supplementary Material 1.

Supplementary Material 2.

Supplementary Material 3.

Acknowledgements
Not applicable.

Authors' contributions
JW, WG, ZZ, YD, WW, BZ, and G helped collect samples, isolate pathogens, perform histopathological and drug sensitivity tests, and identify 16S rRNA. YZ, JW, and WW assisted with data analysis. YZ, JW, and WG contributed to the study concept, wrote the major sections of the manuscript, and revised it. The final manuscript was reviewed and accepted by all authors.

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Data availability
Genome sequences described in the study were deposited in GenBank under the accession number OQ931882 (16S rRNA). The datasets used and/or analyzed in this study are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
The owners of the animals in the case report provided written consent to publish the case in public.

Competing interests
The authors declare no competing interests.

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