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Case Report

Clinical case of lethal ended E. Coli O-157:H7 enteritis combined with liver failure in a newborn

Pekova L.[1*], G. Yosifova[1], M. Dimitrova[1], J. Ananiev[2], D. Petrov[3]

[1]Clinic of Infectious diseases, University Multiprofile Hospital for Active Treatment "Prof. d-r StoyanKirkovitch", Stara Zagora

[2]Department of Pathology, Medical faculty, Trakia University, Stara Zagora

[3]Department of Social medicine and Health management, Medical faculty, Trakia University, Stara Zagora

***Corresponding Author's E-mail: pe_kova@abv.bg**

Abstract

Infection with E. coli O 157 showed a wide clinical spectrum varying from hemorrhagic colitis to hemolytic-uremic syndrome and death. We present a 30-days aged patient who felt ill since 12 hours before the admittance with repeated diarrhea stool and hemorrhagic rash. In his mother, syphilis was proved. The child was admitted in a grave condition, languid and hypothermic. There was a data of heavy enteric toxicosis combined with DIC syndrome and icterus. Hemorrhagic syndrome was limited in some measure in 12 hours. Laboratorial findings showed elevated leukocytes 50.10^9/l with left shift, hepatic cytolysis with ASAT 506 U/l., ALAT 275 U/l, bilirubin 317 mcg/l with predominance of the conjugated fraction and hemostatic disturbances. Microbiological investigations of feces were positive for E. coli O 157:H7 - enterohemorrhagic strain. Antibodies against Toxoplasma gondii were proved. On the second day of hospital stay edema ascites syndrome was expressed with hypoproteinemia 34 g/l and hypoalbuminemia 19 g/l. Despite of applied therapy the child finished lethally with a picture of severe liver, respiratory and cardio-vascular failure. On the autopsy advanced cirrhosis with portal hypertension, ascites, pleurisy and pericarditis as well as hemorrhagic alterations on the gastrointestinal mucosa were determined. We assumed that the diagnosis was severe hemorrhagic E. coli O-157:H7 enteritis founded on a congenital Toxoplasma gondii hepatitis with decompensated cirrhosis, conducted to DIC syndrome and edema ascites syndrome.

Keywords: Ecoli O157:H7, Hepatic cirrhosis, Newborn

INTRODUCTION

E.coli O157: H7 is entero-hemorrhagic strain of the genus *Escherichia*. Typical for this genus are high contagious and low infective dose (Greig et al., 2010). The clinical course of the disease varies from asymptomatic to severe hemorrhagic diarrhea with acute renal failure in the course of hemolytic uremic syndrome (Greig et al., 2010; Fong et al., 1987). Haemolytic uremic syndrome is a heterogeneous group of vasculitic disorders characterized with microvascular angiopathy, hemolytic anemia andrenal disorders. Classical hemolytic uremic syndrome is the basis of most cases of kidney failure in children. Although correct and prompt treatment 14-31% of them have severe course and unfavorable outcome (Martin et al., 1990; Milford et al., 1990). It is etiologically associated with intestinal infections caused by Escherichia coli O157 and other verotoxin-producing E.coli (VTEC) (Bitzan et al., 1991).

P.G.A. is a 30days old infant of gypsy origin, born in term per viasnaturales, third normally passed pregnancy, full-term weighing 3500g. It was breastfed only one week due to hypogalactia of the mother, then was fed with leavened milk. It was grown in poor living conditions. Parents are with low health literacy, which is why medical history was insufficient. By their data it gets ill acutely about 12 hours before hospitalization on February 16th, 2013 with multiple diarrheal stools with mucus and

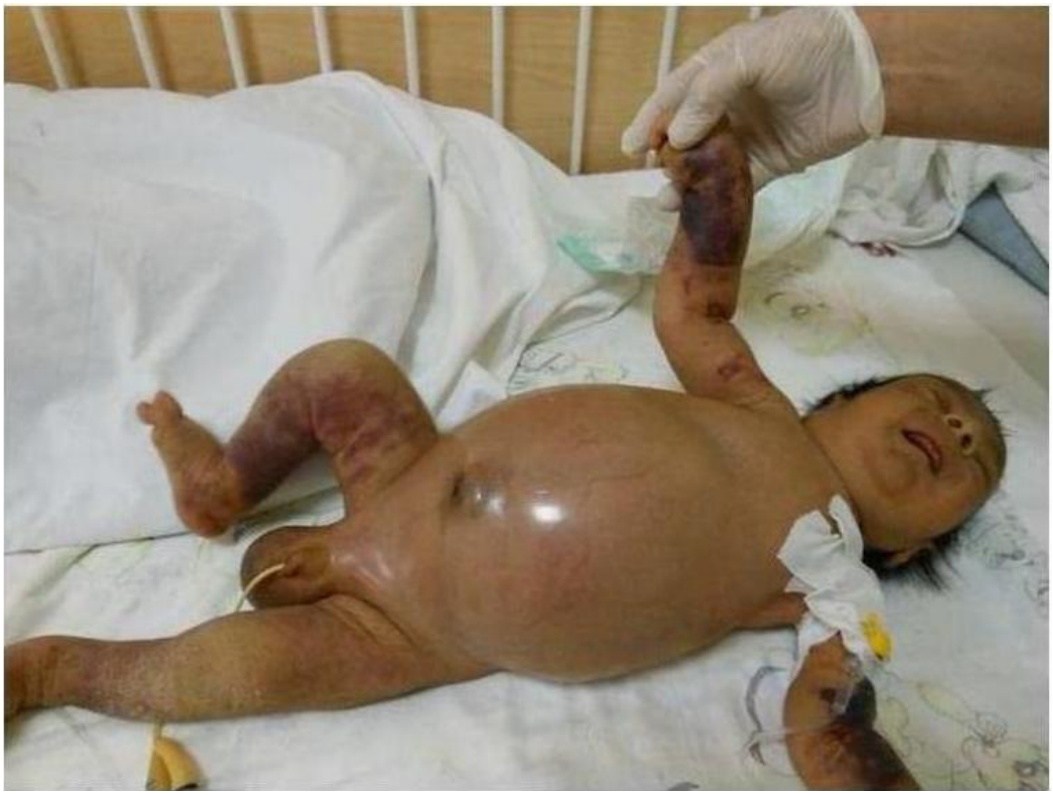

FIgure 1. Edematous skin with hemorrhagic rash as rigor mortis

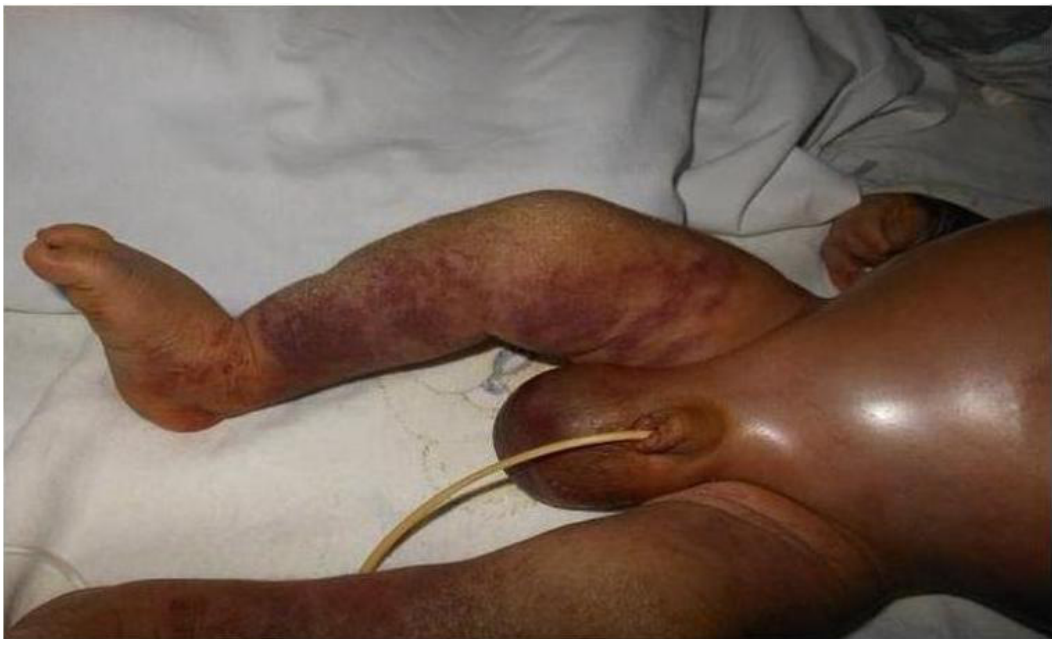

Figure 2. Swollen scrotum with reducible right-sided inguino-scrotal hernia

impurities from streaks of blood. The temperature in the home was not measured. In the last 2hours hemorrhagic rashes appeared on the body. There was no epidemiological evidence of intestinal infection in the family. The mother was diagnosed with Syphilis a month before birth. The patient entered in very poor general condition with data for severe enteral toxicosis, hypovolemic shock and DIC. It was intoxicated, relaxed, hypothermic T33ºC. The skin was with intense jaundice, poor turgor ande lasticity. Petechial rash was visible on

Table 1. Changes in CBC

Date	CYE	Hb	Er	Ht	Leuc	J	St	Sg	Ly	Mo	Tr
16.02.	22	113	3,2	0,32	50,0						115
17.02.		94	2,3	0,27	39,2	2	34	52	11	1	67
19.02.		64	2,0	0,21	36,1						53

Table 2. Laboratorial biochemical changes

Date	Blood sugar	Urea	Creatinine	Total protein	Albumin
16.02.	0,56	6,8	64	34	19
17.02.	0,58	10,3	84		
18.02.	5,33	19,1	138	36	20

Table 3. Laboratorial changes in liver biochemistry

Date	ASAT	ALAT	GGT	Total bilirubin	Direct bilirubin	Fibrinogen	PT	APTT	d-dimer
16.02.	506	275	21	317,2	178,2	1,0	>100s	>300s	3,08
17.02.									
18.02						1,36	13,7 s	98,8s	6,72

the face and trunk, and on limbs was having the character of suffusion, in places with a view of rigor mortis (Figure 1). Scleras were icteric, throat was moderate hemorrhagic. Fontanel was on the level of the skull with dimensions 20x20mm. Enlarged peripheral lymph nodes were missing. There were no lung abnormalities. Heart sounds were rhythmic, HR136/min, slight systolic murmur 2/6 was detectable with pm on apex cordis. Belly was tense, above the chest level. Hepatosplenomegaly could not be refined. Peristalsis was weak. The scrotum was swollen; there was reducible inguino-scrotal hernia on the right (Figure 2). Through the indwelling catheter expired bloody urine. Signs of meningoradicular irritation were not detected. Besides macroscopic hematuria, child demonstrates signs of parenchymal and cavity hemorrhage with rectorrhagia and hematemesis. After enemas, through the gas pipe expired melanosis at the beginning, but then scarce amount of green, watery stools with mucus abundance. On the second day of hospitalization was expressed generalized edema type of anasarca. Belly increased volume-bloated with flat flanks, shiny, tensioned skin and weak to absent peristalsis, with significant venouscol laterals by type on caputmedusae. Using ultrasound was confirmed the suspicion of fluid in the abdominal cavity. From February 19th was observed manifestation of brain edema with the indicative neck rigidity in the final stage of cervical flexion and slight prominence of the large fontanelle above the skull. Due to the severe thrombocytopeniaa lumbar puncture was not performed. Evidence of severe bacterial inflammation, dysfunction of the liver and kidneys were visible by laboratory tests. Tables 1, 2, 3. Data about hypoelectrolytemia, dehydration and metabolic acidosis was established. On February 20th during lung auscultation was detected weakened vesicular breathing with bilateral diffuse scattered small wetland wheezing. X-Ray confirmed the presence of bilateral pneumonia. In order to relax the difficult breathing, abdominal puncture with evacuation of ascites fluid was performed (Figure 3). The condition did not change. The condition did not change. Although adequate therapy, hemorrhagic diathesis persists with the emergence of new petechial rashes and formation of large hematomas in places of manipulations.

Child died on February 21st at 1am with the picture of severe respiratory and cardiovascular insufficiency.

From the performed feces and serological tests on the third day of hospitalization was obtained result for E. coli O-157 -H7. Table 4. Therapy with glucose-saline solutions, Medaxon, corticosteroids, haemostatics and blood substitutes, bioproducts, osmodiuretics and immunostimulatory agents was conducted.

It is known that upon infection with E.coli O-157 -H7 use of antibiotics and corticosteroids is not desirable, because they did not support, and also can worsen the condition of the patient. Thinking in the direction of sepsis of unknown etiology motivates us for their application. According to literature review the amount of lethality, even with intensive care implemented, is 3-5%.

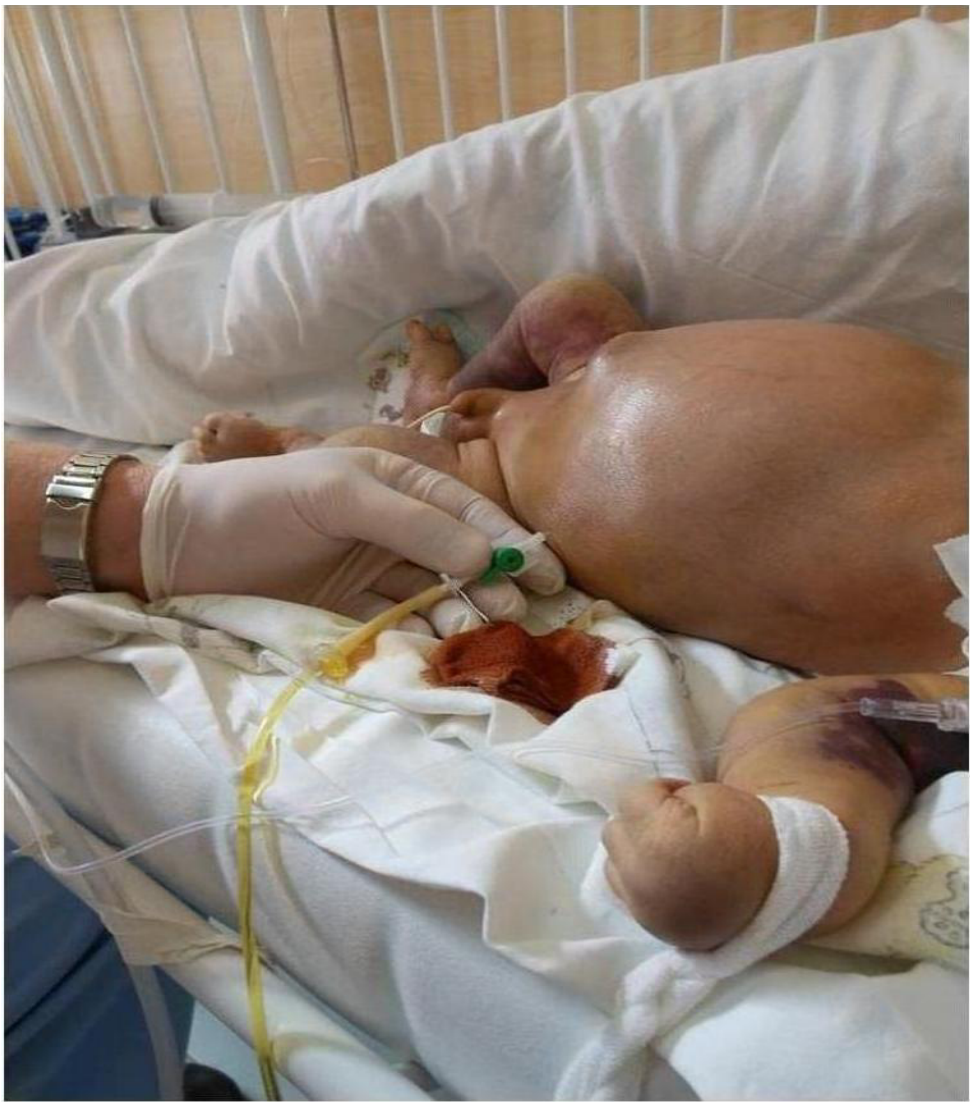

Figure 3. Abdominal puncture with evacuation of ascites fluid

Table 4. Microbiological and serological tests

Date/ Test	Anti HIV1/2	HBsAg	Anti HCV	Wasserman	Anti-T. pallidum IgM (-) neg.	anti-T.GondiiIgM	Coprocultures Shigella Salmonella E.coli Campilobacter	16.02. (-) neg.
16.02.	(-) neg.	(-) neg.	(-) neg.	(-) neg.			(-) neg.	
17.02.								
18.02.					(-) neg.	>1:500		
19.02							E.coli O_{157}. (+) poss.	

DISCUSSION

Considered case of hemorrhagic colitis with hemolytic-uremic syndrome caused by enterohemorrhagic strain of E.coli -O157: H7 occur at severe clinical form with enteral toxemia and sepsis, multiple organ failure and thrombotic thrombocytopenicpurpura, led to death. Contamination most likely occurred in the family, but the lack of commitment by parents to the child's condition and unwillingness to cooperate lead to impossibility to conduct microbiological testing. The reason for the prolonged neonatal jaundice was innate hepatitis with

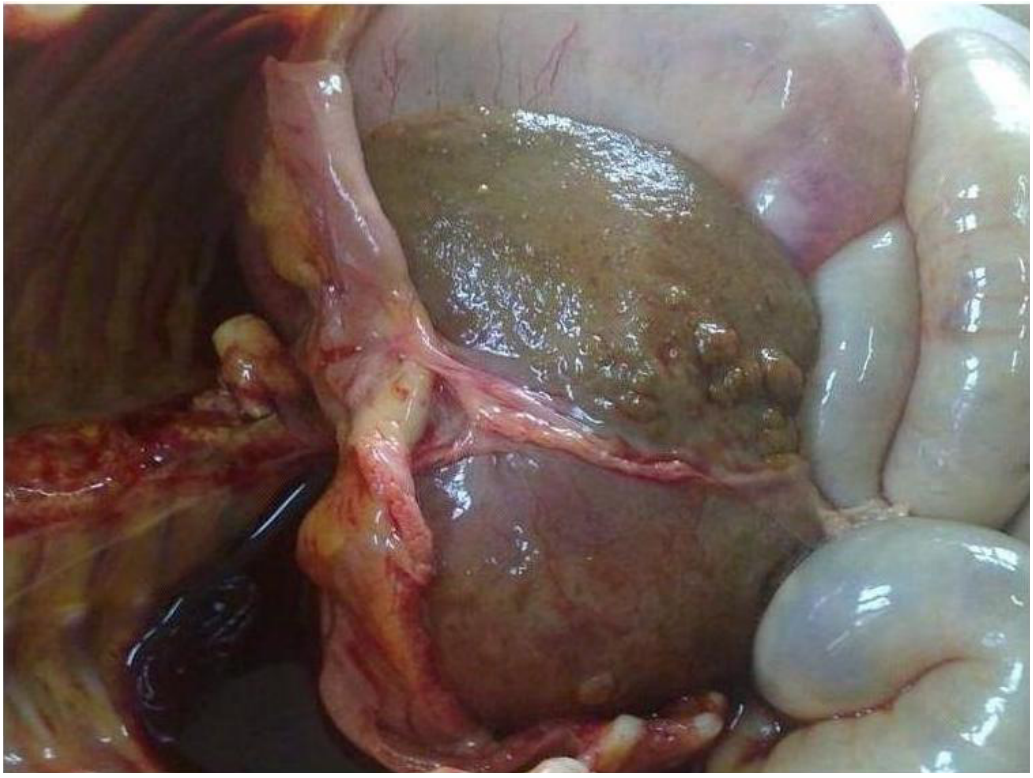

Figure 4. Macroscopic changes of decompensated liver cirrhosis

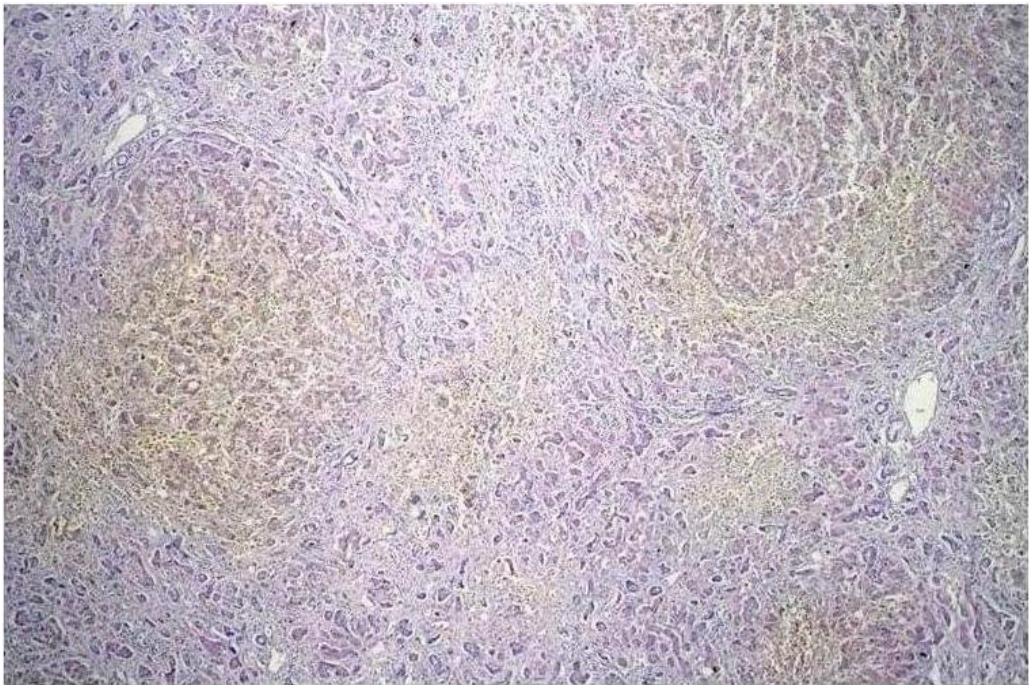

Figure 5. Liver - disruption in architecture of the liver, areas of necrosis, bridging fibrous septas and rounded parenchymal nodules of regenerating hepatocytes (liver necrosis and cirrhosis) (H&E stain).

etiologic agent Toxoplasma Gondii. It has led to liver failure ascites, and hemostatic disorders based oncirrhosis. We discussed the possibility of congenital syphilis because the mother was serologically confirmed

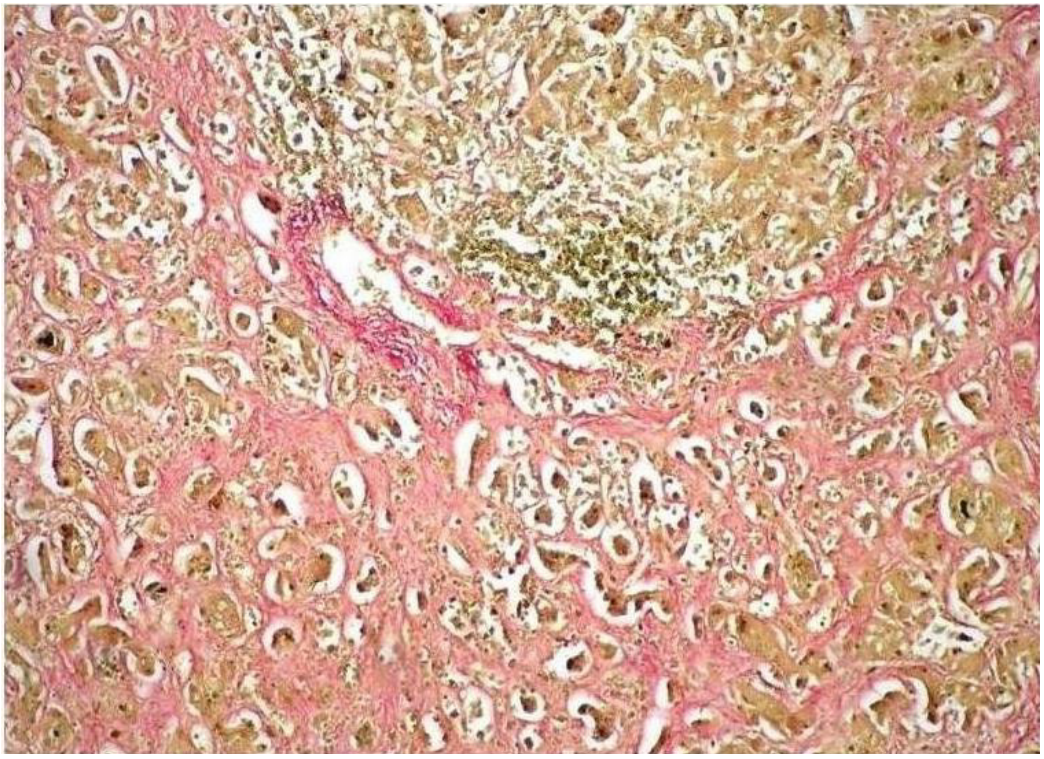

Figure 6. Liver - fragmented specimens with rounded edges containing connective tissue (liver cirrhosis) (Van Gieson's stain).

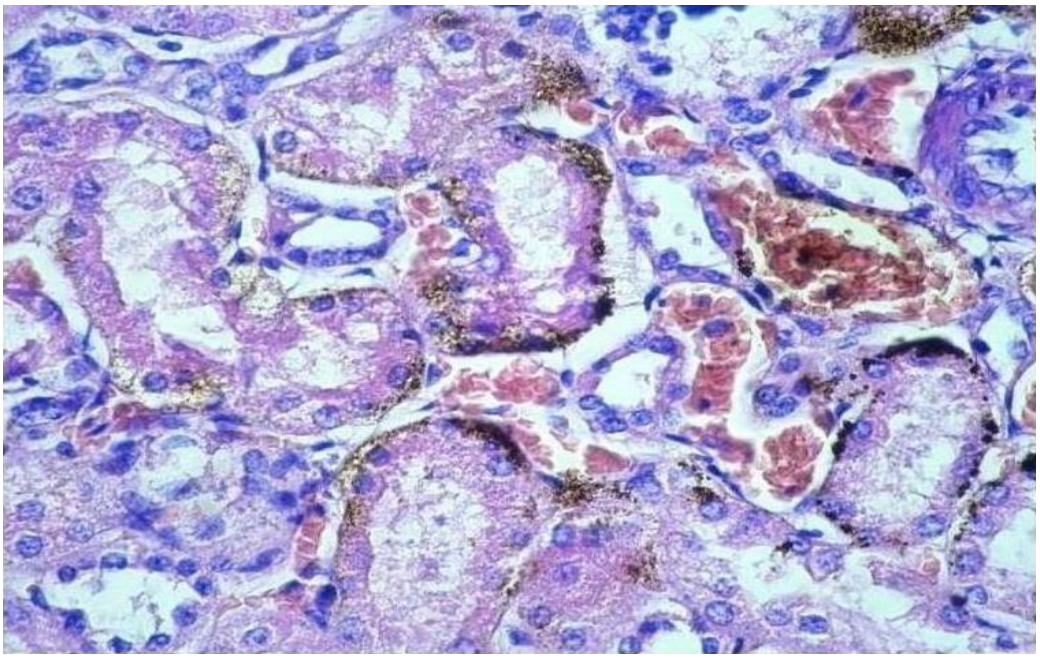

Figure 7. Kidney – dystrophy of epithelial cells of the proximal convoluted tubules and small changes of capillary wall thickening, endothelial cell swelling, and narrowing or thrombosis of the capillary lumen.

with such. It may proceed with such prolonged jaundice of the newborn, with hepatosplenomegaly, syphilitic pemphigus, thrombocytopenia and hemorrhagic syndrome. Negative serological testing of the child can be explained by the young age, insufficient to build its own antibodies. Intestinal infection with E. Coli O157: H7

has led to renal involvement with manifestation on hemolytic-uremic syndrome and thrombotic thrombocytopenicpurpura (Elliott and Nichols, 2001). Involvement of the liver, which is subject to damage by T. Gondii, and perhaps by T. pallida, as well as the lungs and brain are consequential during septic disease state. Hypotrophy and immature immunological status clearly contributed for the poor outcome (Belongia et al., 1993). Pathologic findings revealed the presence of macroscopic (Figure 4), and microscopic changes in decompensated liver cirrhosis based on severe intrauterine hepatitis: Figure 5. Liver - disruption in architecture of the liver, areas of necrosis, bridging fibrous septas and rounded parenchymal nodules of regenerating hepatocytes (liver necrosis and cirrhosis) (H&E stain) (Figure 6). Liver - fragmented specimens with rounded edges containing connective tissue (liver cirrhosis) (Van Gieson's stain) (Figure 7). Kidney – dystrophy of epithelial cells of the proximal convoluted tubules and small changes of capillary wall thickening, endothelial cell swelling, and narrowing or thrombosis of the capillary lumen.

CONCLUSION

This case illustrated a fatal outcome, due to hemolytic-uremic syndrome provoked by E. coli -157:H7. This illness was demonstrated on the bad back-ground consisted of decompensated cirrhosis caused by Toxoplama Gondii hepatitis and innatelues. In spite of all medical efforts, the therapy was ineffectiveness. Hypotrophy and poor social-hygiene conditions of live were additive factors, contributing to the lethal end.

REFFERENCES

Belongia EA, Osterholm MT, Soler JT, Ammend DA, Braun JE, MacDonald KL (1993). Transmission of Escherichia coli0157: H7 Infection in Minnesota Child Day-care Facilities. JAMA.; 269(7): 883-888.

Bitzan M, Moebius E, Ludwig K, Müller-Wiefel DE, Heesemann J, Karch H (1991). High incidence of serum antibodies to Escherichia coli O157 lipopolysaccharide in children with hemolytic-uremic syndrome The J. Pediatrics, vol119(3): 380-385

Elliott MA, Nichols WL (2001). Thrombotic thrombocytopenic purpura and hemolytic uremic syndrome.Mayo Clin Proc.;76:1154-1162.

Fong JSC, de Chadarevian JP, Kaplan BS (1987). Hemolyticuremic syndrome. Current concepts and management. PediatrClinNorthAm1982;29:83556. Kaplan BS, Proesmans W. The hemolyticuremie syndrome of childhood and its variants. SemHematol;24:148-60.

Greig JD, E.C.D. Todd, C. Bartleson, and B. Michaels (2010). Infective Doses and Pathen Carriage USDA Food Safety Education Conference: 19-20

Martin DL, MacDonald KL, White KE, Soler JT, Osterholm MT (1990). The epidemiology and clinical aspects of the hemolytic uremic syndrome in Minnesota. New EnglJMed;323:1161-7.

Milford DV, Tavlor CM, Guttridge B, Hall SM, Rowe B, Kleanthous H (1990). Haemolyticuraemic syndromes in the British Isles 1985-8: association with Verocytotoxin-producing Escherichia coli. Part 1. Clinical and epidemiological aspects. Arch Dis Child;65:716-21.