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Fatty liver and subcutaneous edema in a free-living bottlenose dolphin (*Tursiops truncatus*, Montagu 1821) from the Adriatic Sea; light- and electron-microscopical study

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ABSTRACT

Research was carried out on a 21-year-old, 288 cm-long free-living grand multigravida female bottlenose dolphin (*Tursiops truncatus*, Montagu 1821) with mass of 214 kg. The animal died on 8 October 1997 in the Adriatic Sea, near Šibenik, Croatia. Prior to death she showed some neurological symptoms. For control and comparison we used the carcasses and tissues of two presumably clinically healthy dolphins. Autopsy showed massive edema in the subcutaneous tissues (between blubber and musculature) on the dorsal side of the body of the diseased subject. The fluid in intercellular tissue spaces was clear and light-yellowish. Larger amounts of the same fluid were found in serous sacs of the animal, as well as slight brain edema. The most prominent structural change was found in the liver, which was

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normal in size but extremely yellow and fragile. Its structure was totally destroyed, without any traces of blood, and there was pronounced fatty degeneration of liver parenchyma. All hepatocytes were also destroyed and swollen mitochondria and dilated endoplasmic reticulum were evident. This massive hepatic degeneration and necrosis was probably due to some unknown acute biological or chemical agent that provoked hepatargic crisis and the ultimate death of this free-living female dolphin.

Key words: dolphin, bottlenose dolphin, *Tursiops truncatus*, fatty liver, subcutaneous edema, light-microscopy, electron-microscopy, histochemistry, Adriatic Sea

Introduction

The liver is one of the most important metabolic organs. It participates in transformation and synthesis of many biologically important substances. Its obvious function is transformation of substances that have entered the gastro-intestinal tract and that are absorbed in the gut (FORENBACHER, 1993). Some of those substances are often toxic and the liver renders them harmless in some way. Liver parenchyma, i.e., hepatocytes, are in close contact with blood, especially that arriving from gastrointestinal organs (DELLMANN and BROWN, 1976; BANKS, 1981; MOSIMANN and KOHLER, 1990; MIYAI, 1991; JUNQUEIRA et al., 1995). This means that hepatocytes are constantly burdened by various substances which arrive from gastrointestinal tract and which are often toxic. Some specific hepatotoxic substances also have an effect on hepatocytes, as well as some specific bacteria and viruses (CULLEN and RUEBNER, 1991; FORENBACHER, 1993). If the amount of those noxious substances is above the tolerable threshold of hepatocytes, or if they have a too frequent effect on hepatocytes, the hepatocytes will demonstrate recoverable changes in the beginning. If the stimulus is severe enough or lasts sufficiently long, the ability of cells to adapt may be exceeded and cellular functions become altered, and the cell is said to be injured. If the injury is sub-lethal the cell survives, even though it may function abnormally. Chronic sub-lethal injury or altered environmental factors may induce cells to undergo stable, structural and functional alterations that restore homeostasis. Sub-lethally injured cells may also continue to deteriorate structurally and functionally, reaching a point from which recovery is no longer possible. The so-called 'point of no return' is not yet precisely defined, either ultrastucturally or functionally (MARZELLA and TRUPM, 1991). In most cases the hepatocytes respond to intolerable noxious stimuli with accumulation of large amounts of fatty substances (fat infiltration and degeneration) and later on with apoptosis and necrosis of large numbers of hepatocytes. If the noxious stimulus lasted long enough prior to death there will also be cirrhosis of liver (MARZELLA and TRUPM, 1991). All these changes in liver tissue could be seen on the light or electron microscope.

Similar spontaneous changes in liver are often found in humans and other animal species as a result of various illnesses or poisoning, or as an accidental finding in individuals that died due to some other cause. In marine mammals, especially dolphins, fatty degeneration in liver is rarely found. When it is found, it is usually accidental and is not the cause of death. SIMPSON and GARDNER (1972) found fatty degeneration of hepatocytes in captive dolphins and GOMERČIĆ et al. (1998) mention it as an accidental finding. Parasitic cholangitis and pericholangitis were found in free-living harbour porpoises by SIEBERT et al. (1996) and KUIKEN et al. (1996). LIPSCOMB (1996) described liver fibrosis in an adult free-living dolphin, while BIRKUN (1996) described venous congestion, blood stasis, cell degeneration and necrosis in liver of captive bottlenose dolphins. However, we were unable to find data in the literature that mentioned fatty liver as a cause of death. Also, no mention of subcutaneous (sub-blubber) edema seems to have been made, especially in reference to the dorsal side of dolphin trunk. In this paper we describe a case of a free-living bottlenose dolphin from the Adriatic Sea whose most probable cause of death was pronounced and irreparable liver changes.

Materials and methods

Research was carried out on three specimens of bottlenose dolphin (*Tursiops truncatus*, Montagu 1821) that died along the eastern shores of the Adriatic Sea.

The diseased female dolphin seemed disoriented prior to death and was swimming near the coast of Pirovac, near Šibenik, Croatia (N43°48'30"; E15°40'30") in circles, swinging around her longitudinal axis. The animal died 8 October 1997 and was immediately recovered from the sea, frozen and transported to Zagreb.

For control and comparison we used the carcasses and tissues of clinically presumable healthy dolphins that had been caught in fishing nets and drowned. The control female dolphin was drowned near the west coast of the Istrian peninsula, Croatia (N45°03';E13°35') on 16 October 1990. The control male dolphin was drowned in the environs of the town of Šibenik, Croatia (N43°43'40";E15°53'30") on 21 December 1995.

The dolphins were measured according to PERRIN (1975) and aged by growth-layer groups in the dentine according to SLOOTEN (1991).

We performed dissection of all animals at the Department of Anatomy, Histology and Embryology, Faculty of Veterinary Medicine, Zagreb, Croatia, and took the samples for microscopic, bacterial, viral and heavy metal analysis. The liver samples were fixed in 4% formalin and

osmium tetroxide, stained with haematoxylin and eosin (H&E), method according Mallory, with PAS procedure and Sudan black B and Sudan III., and with uranyl acetate for electron microscopic examination (CULLING, 1974). Total mercury, lead and cadmium contents were analysed by standard procedures (HATCH and OTT, 1968; STANLEY and ELLIOT, 1976; CAPAR, 1977; BOYER, 1984) in the diseased female dolphin and the control male dolphin. Results are expressed as ppm (g of heavy metals in 1 g of wet mass of liver tissue). Slides were examined and photographed by light microscope Nikon Microphot-FXA and by electron microscope JEOL JEM-1200EX.

Results

All three examined dolphins were in good condition, without any signs of starvation.

The diseased female dolphin was 21 years old, 288 cm in length and had 214 kg. She had previously given birth many times.

The control female dolphin was 4 years old, 250 cm long, had 204 kg and had not given birth.

The control male dolphin was 9 years old, 278 cm long, had 237 kg and was sexually mature.

At the moment of death the diseased female dolphin had only a couple of small bones and otoliths of fish in her forestomach. No chyle was found in mesenterial lymph vessels.

In the forestomach of control female dolphin we found 4 large European pilchards (*Sardina pilchardus*) that were partially digested, and numerous big chitinous teeth of squid. Chyle was visible in mesenterial lymph vessels.

The forestomach of control male contained an undigested conger eel (*Conger conger*) and many remains of partly-digested fish and numerous fish bones, but no chitinous teeth of squid. Chyle was visible in mesenterial lymph vessels.

The liver of diseased female dolphin contained 469.4 ppm of total mercury, 0.12 ppm of lead and 0.181 ppm of cadmium.

The liver of control male contained 198.1 ppm of total mercury, 0.11 ppm of lead and 0.332 ppm of cadmium.

None of the control animals showed pathoanatomical signs of disease, although we did find nematodes and slight pneumonia that was probably caused by parasites. Livers of both control animals were distinctly red-

brown, almost black in colour and their consistency were compact (Fig. 1, above). Serous cavities (peritoneal, pericardial and pleural cavities) contained no liquid, but their walls were profusely moist. In the body trunk, blubber was found attached to dark muscles, with the line between blubber and muscles being obvious and well-defined, without any sign of subcutaneous edema (Fig. 2, left).

During the autopsy of diseased female dolphin we found extremely profound subcutaneous edema (between blubber and musculature) that extended along dorsolateral side of the entire body, 2.8 cm thick and composed of clear light-yellowish liquid. Larger amounts of clear light-yellowish liquid were also found in peritoneal, pleural and pericardial cavities. We also noted slight brain edema. The most pronounced change was found in the liver, which was distinctly yellow (Fig. 1, below), which contrasted sharply with the livers of healthy dolphins (Fig. 1, above). The liver was not enlarged but its consistency was fragile - it disintegrated without any haemorrhage when we touched it with forceps after removing the capsule. Macroscopic examination failed to reveal any other significant changes that would suggest the cause of death of the diseased female dolphin.

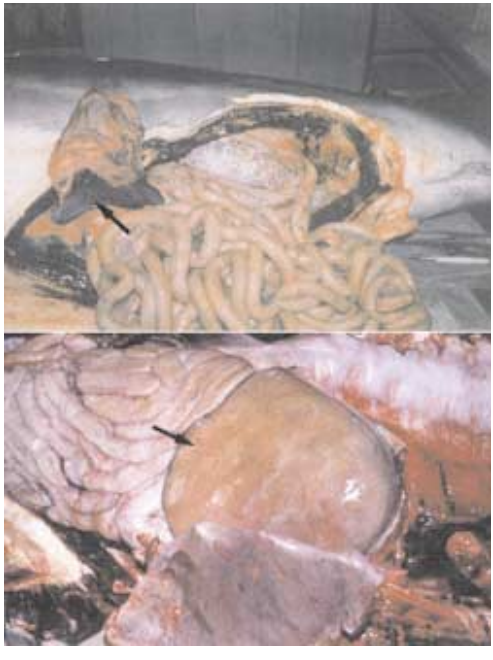

Fig. 1. The liver (arrow) of healthy dolphin is dark red-black in colour (above) and the liver (arrow) of the diseased female dolphin is yellow in colour (below)

Fig. 2. The blubber (subcutaneous adipose tissue) in healthy dolphin (left) is in direct contact with musculature, but in the diseased female dolphin (right) between blubber and musculature is 2.8 cm thick, clearly visible edema (arrow)

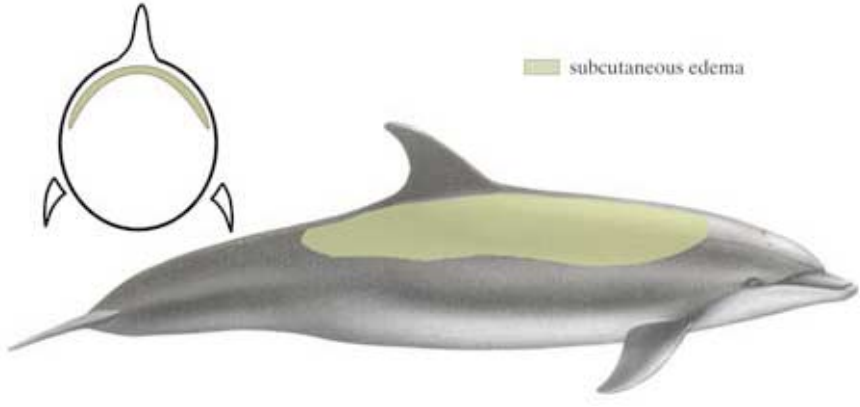

Fig. 3. Position of subcutaneous edema in diseased dolphin body

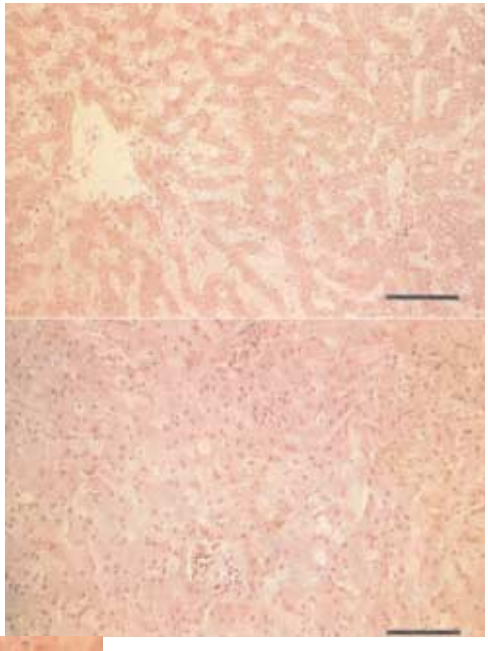

Fig. 4. Liver microscopical structure of healthy dolphin (above) with recognisable plates of hepatocytes and separated from each other by wide sinusoids filled by erythrocytes. Liver of the diseased female dolphin (below) with destroyed liver structure and without any normal hepatocytes. H&E; 20×2.5; scale bar 100 m.

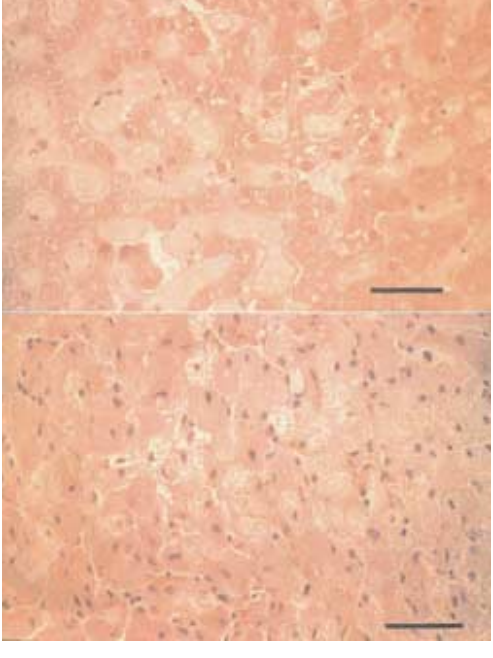

Fig. 5. Detail of Fig. 4. In the diseased female dolphin (below) numerous hepatocytes are foamy, and all nuclei are pyknotic. Sinusoids of healthy dolphin (above) are broad and filled with blood, but sinusoids of diseased female dolphin (below) are not visible. H&E; 40×2.5; scale bar 50 m.

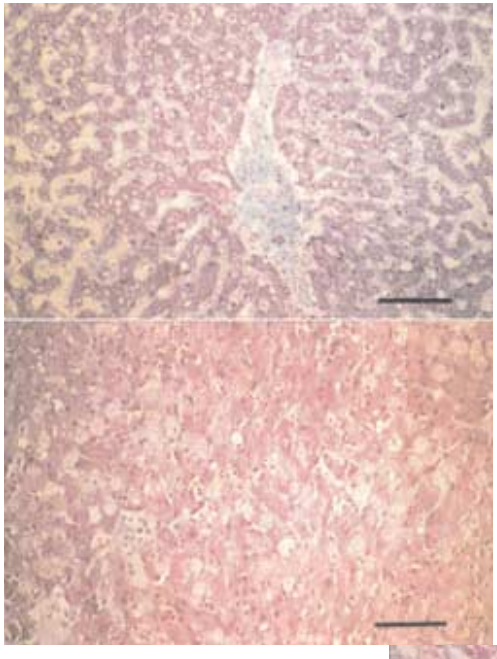

Fig. 6. Microscopic structure of the liver of healthy dolphin (above) with plates of hepatocytes and wide sinusoids filled with erythrocytes. In the diseased female dolphin (below) the liver structure is destroyed (nearly unrecognizable hepatocytes are markedly foamy). Small and larger vacuoles are seen. The amount of connective tissue is not increased in the diseased female dolphin and it is unperceivable in both livers. Mallory stain; 4×5; scale bar 200 m.

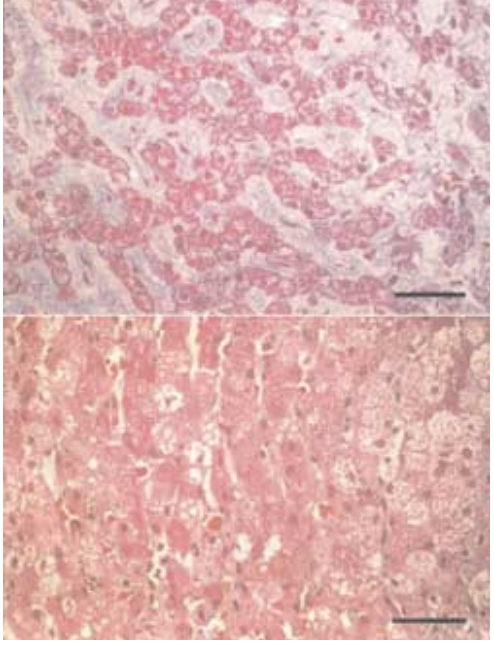

Fig. 7. Details of Fig. 6. In healthy dolphin (above) there are plates of normally structured hepatocytes; between plates there are sinusoids filled by erythrocytes. In the diseased female dolphin (below) there are destroyed foamy hepatocytes and only few erythrocytes in very narrow sinusoids. Mallory stain; 40×2.5; scale bar 50 m.

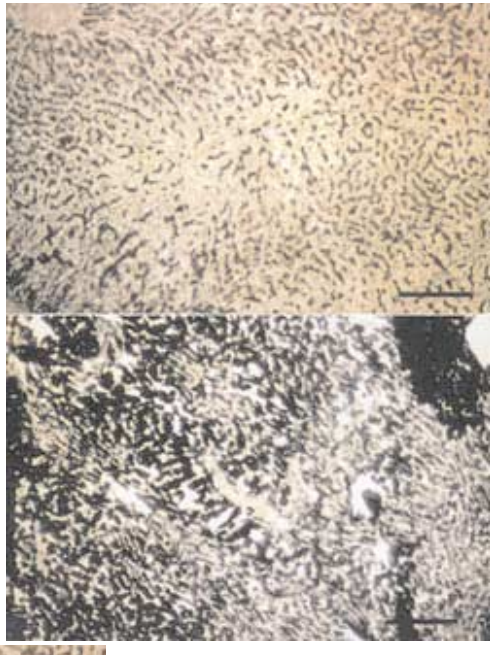

Fig. 8. Fatty substances in the liver of healthy dolphin (above) and in the diseased female dolphin (below). In hepatocellular cytoplasm of healthy dolphin there is a small quantity of fatty substances stained black, but in the diseased female dolphin the fatty substances are increased significantly. Sudan black B; 4×5; scale bar 200 m.

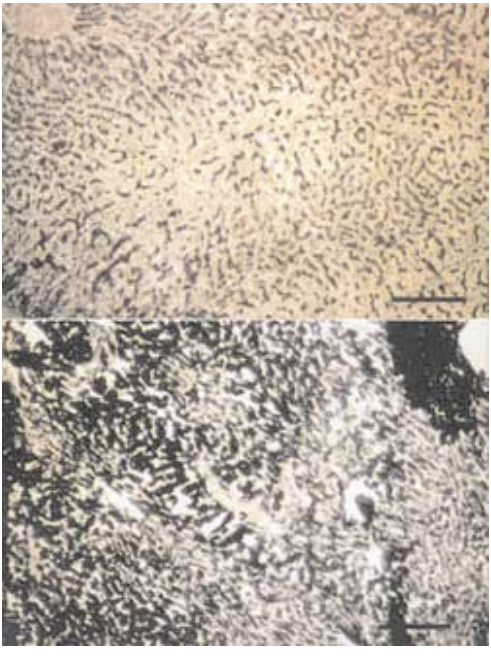

Fig. 9. Detail of Fig. 8. In some hepatocytes of healthy dolphin (above) there are few fatty droplets stained black, while in the diseased female dolphin (below) the number of fatty droplets are significantly higher and hepatocytes are nearly black stained due to accumulation of fatty droplets. Sudan black B; 40×5; scale bar 25 m.

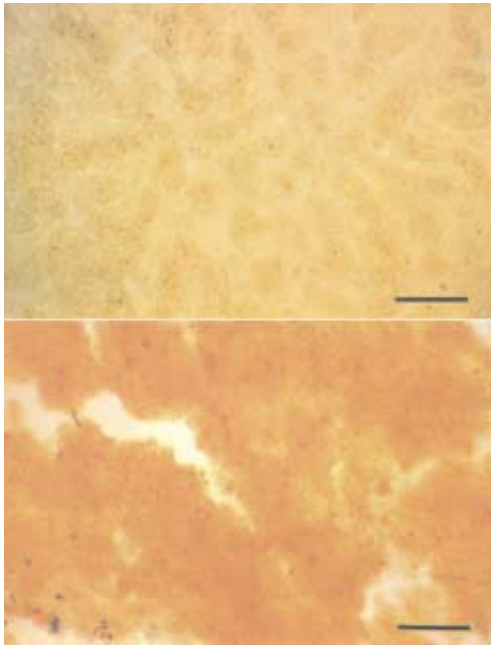

Fig. 10. Fatty substances in the liver of healthy dolphin (above) and in the diseased female dolphin (below). In hepatocellular cytoplasm of healthy dolphin there is a small quantity of fatty substances stained red, but in the diseased female dolphin the fatty substances are increased significantly and hepatocytes are nearly red stained owing to accumulation of fatty droplets. Sudan III.; 4×2.5; scale bar 50 m.

Light microscopic examination revealed complete disintegration of liver structure, an almost complete absence of blood and pronounced fat degeneration of parenchyma (Figs. 4, 5, 6, 7, 8, 9, 10 - below). No enlargement of connective tissue was found. In all specimens, both healthy control animals and the diseased female animal, glycogen content was very small.

Electron microscopic analysis revealed that all hepatocytes have undergone some change. In all of them, their cellular membranes had completely disappeared. We were able to find only remains of individual cell organelles, mostly swollen mitochondria and dilatated endoplasmic reticulum (Figs. 11, 12). Cell nuclei were the best preserved, although they often showed chromatin condensation and initial signs of karyolysis and dilatation of perinuclear spaces, as well as breakage of nuclear envelope (Figs. 17, 18). There were small and somewhat larger fat droplets in whole liver parenchyma that was disintegrated (Figs. 13, 14, 15, 16). We were unable to find any erythrocytes in liver tissue. Sinusoids, endothelium and Disse's spaces were completely decomposed (Figs. 19, 20). Hence, we concluded that all examined liver specimens were completely degenerate.

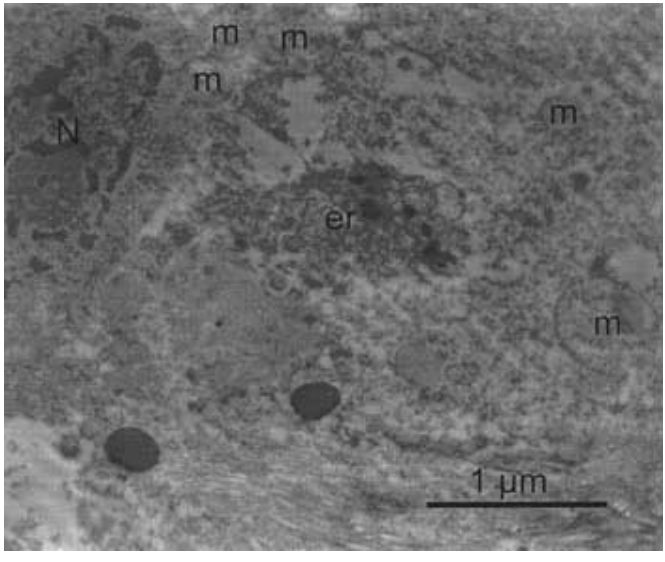

Fig. 11. Complete disintegration of liver parenchyma of the diseased dolphin with remains of swollen mitochondria (m), enlarged endoplasmic reticulum (er) and the beginning of karyolysis (N). 15,000×

Fig. 12. Disintegrated liver parenchyma of the diseased dolphin with three nuclei (N) in which can be seen the beginning of chromatin condensation and karyolysis (arrow). 4,000×

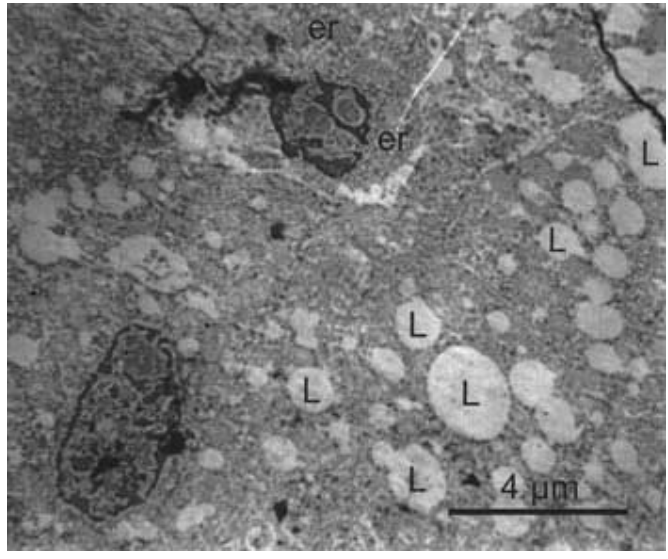

Fig. 13. Marked accumulation of fatty substances (L) in disintegrated liver parenchyma of the diseased dolphin, with vesiculation and dilatation of endoplasmic reticulum (er). 4,000×

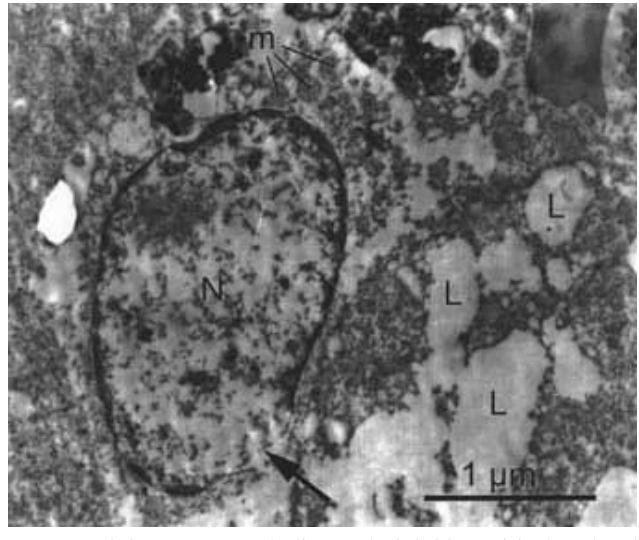

Fig. 14. Damaged hepatocyte of diseased dolphin, with fat droplets (L), a few mitochondria (m) and nucleus (N) that is almost unchanged, apart from partial discontinuity of karyolema and enlargement of perinuclear spaces (arrow). 15,000×

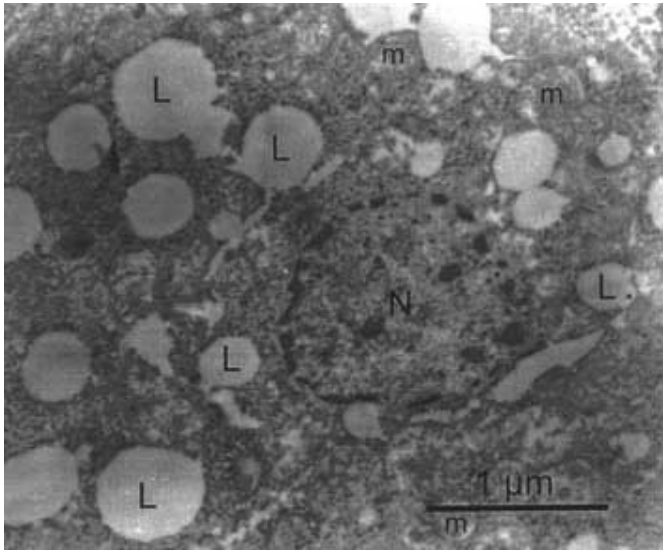

Fig. 15. Numerous fat droplets (L) in disintegrated hepatocyte of the diseased dolphin, with the nucleus (N) and individual mitochondria (m) still preserved. 15,000×

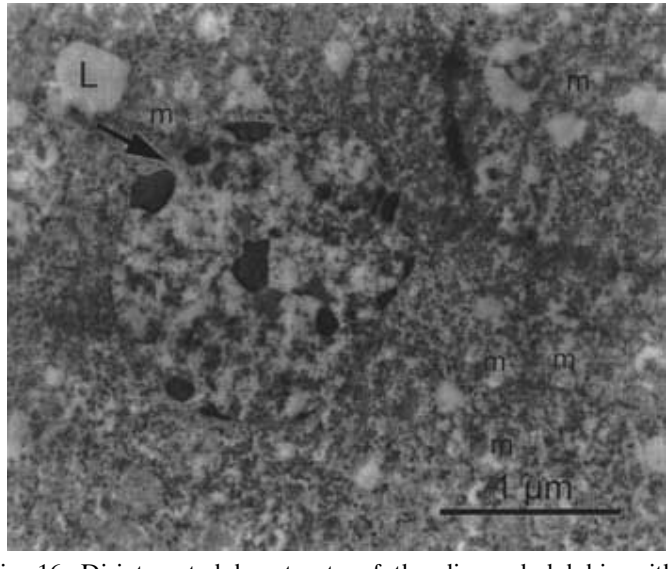

Fig. 16. Disintegrated hepatocyte of the diseased dolphin with mitochondria remains (m), fat droplets (L), nucleus with chromatin condensation and partial karyolemolysis (arrow). 15,000×

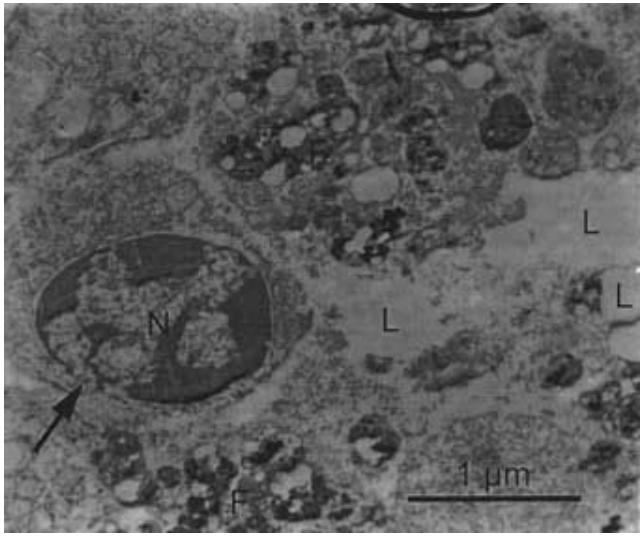

Fig. 17. Disintegrated liver parenchyma of the diseased dolphin with numerous fragments of decomposed cells (F), fused fat droplets (L) and a nucleus (N) that is almost unchanged, with intact nuclear envelope (arrow) and condensed chromatin. 15,000×.

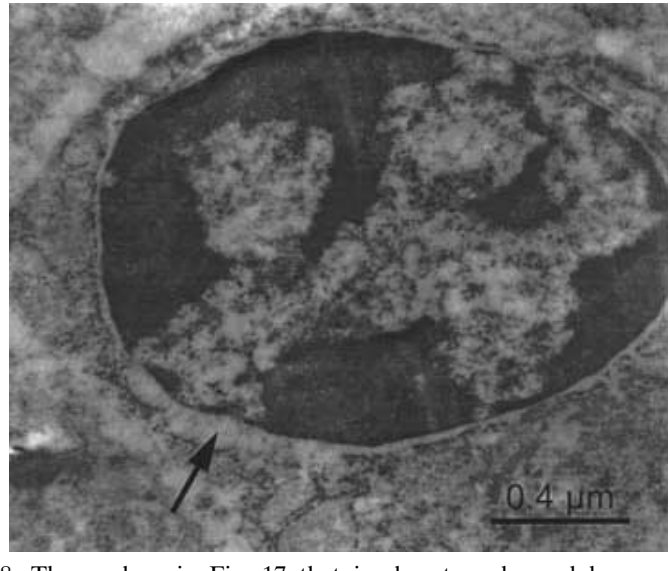

Fig. 18. The nucleus in Fig. 17 that is almost unchanged has condensed chromatin and some enlarged perinuclear space (arrow). 30,000×

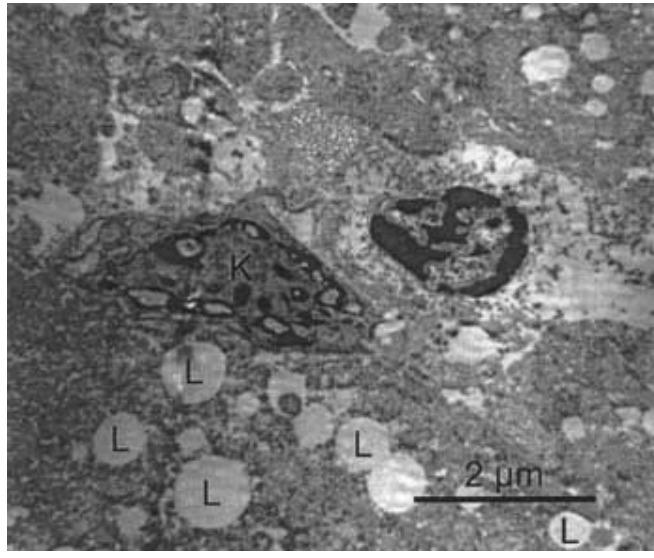

Fig. 19. Kupffer's cell (K) beside destroyed Disse's space and completely disintegrated liver parenchyma with fat droplets (L) from the diseased dolphin. 7,500×.

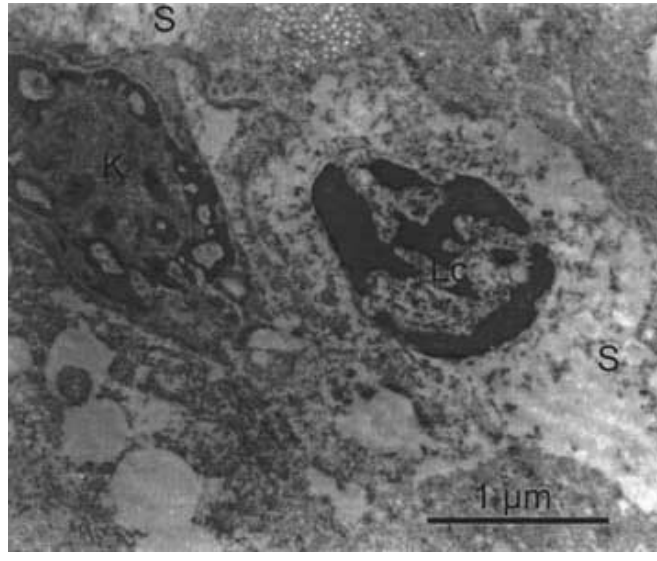

Fig. 20. Kupffer's cell (K) in Fig. 19 along with sinusoids (S) filled with cell detritus and remains of a lymphocyte (Lc). 15,000×.

Discussion

Based on autopsy analysis, light microscopic and electron microscopic investigation of the liver tissue, we concluded that the dead diseased female dolphin lost its liver functions rather rapidly due to irreparable changes of parenchymal and non-parenchymal liver cells that manifested in fat degeneration and ultimately in necrosis of hepatocytes. The consequence of those drastic ultramicroscopic and microscopic changes of liver cells is surely subcutaneous edema, hydrops of serous cavities and slight brain edema, as well as rapid intoxication of the animal which eventually led to death of the diseased female bottlenose dolphin. Most probably the liver damage was acute, due to the fact that we found no increase in the amount of connective tissue, i.e., signs of cirrhosis. It is supposed that the animal was affected by disease only for a brief period of time. That was also suggested by the animal's good condition, without any signs of cachexia.

The foregoing is in agreement with the statements of CULLEN and RUEBNER (1991) and FORENBACHER (1993) who claim that continuous or repetitive liver injury causes chronic liver injury associated with fibrosis and, if continued long enough, may lead to cirrhosis. If the injury is relatively mild, it may regress; if severe, it may progress to necrosis and become irreversible (CULLEN and RUEBNER, 1991).

The observed neural symptoms of the diseased dolphin (hepatoencephalic syndrome) were most probably due to acute liver disorder. The same hepatargic crisis is known in domestic animals with severe liver parenchymal damage (such as diffuse necrosis, extensive steatosis, acute hepatitis and cirrhosis) where there are signs of encephalic symptoms due to liver dysfunction (FORENBACHER, 1993).

Necrotic liver damage is usually accompanied by inflammatory reaction of blood vessels and mesenchymal cells. The exception is the case in which the animal dies rapidly, when reactions do not have the time to develop (FORENBACHER, 1993), which was the case with the investigated diseased female dolphin.

In the diseased female dolphin we found increased amounts of clear yellowish intercellular fluid in subcutaneous tissue, in serous cavities (pleural, pericardial and peritoneal cavities) and in the brain. This was not caused by blood stasis due to stenosis of branches of liver portal veins, but by dysfunctioning of the liver and probably of consequent hypoproteinaemia, which resulted in edema and fluid accumulation in serous cavities. If the cause of those changes had been stasis of portal blood flow in the liver, then we would have found more pronounced

ascites and less pronounced hydrops of pleural and pericardial cavities, and edema of subcutis and brain. However, the amount of fluid in peritoneal cavity was not greater than that in pleural and pericardial cavities. The defective liver function presumably caused hepatargy, which in turn caused hepatoencephalic syndrome, as mentioned above.

The increased level of total mercury in liver of diseased female dolphin (469.4 ppm), in comparison with control male dolphin (198.1 ppm), was not the likely cause of changes we found in the liver, as there are no data in the literature that would claim that the increased accumulation of mercury and other heavy metals in the organism cause such changes in liver. Rather, it could be the consequence of the difference in age and sex - that is to say, the diseased dolphin was much older (21 years old, compared to the control male who was 9) and the female that had been gravid and in lactation many times, so that during her lifetime she consumed larger quantities of feed than control male animal. This is why she had accumulated more than double the quantity of total mercury in her liver.

Hepatic edema caused by dysfunction of the liver in terrestrial animals is most often placed subcutaneously in ventral parts of the body, due to gravity. In the diseased female dolphin we found subcutaneous (between blubber and musculature) edema in the dorsal parts of dolphin trunk (Figs. 2, 3). This unusual dorsal position of subcutaneous edema in dolphin is a result of gravity, hydrostatic and hydrodynamic pressure, which cause aggregation of intercellular fluid in the dorsal part of body where pressure on the dolphin's surface is lowest.

The cause of changes in the liver described in this paper can only be speculative. There is an enormous number of hepatotoxic agents (biological, chemical or physical) that could provoke such changes. Until now, such spontaneous changes have not been described in dolphins, and certainly not in the dolphins from the Adriatic Sea.

Acknowledgement

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References

BANKS, W. J. (1981): Applied Veterinary Histology. Williams and Wilkins. Baltimore and London.

BIRKUN, A. (1996): Complexities of by-catch diagnosis in Black Sea cetaceans. In: Diagnosis of By-Catch in Cetaceans: Proceedings of the second European Cetacean Society workshop on cetacean pathology, Montpellier, France, 2 March 1994 (Kuiken, T., Ed.). European Cetacean Society. Saskatoon, Saskatchewan, Canada. pp. 12-15.

BOYER, K. W. (1984): Metals and other elements in food. In: Official Methods of Analysis. 14th ed. (Williams, S., Ed.). AOAC Inc. Arlington, USA. pp. 444-478.

CAPAR, S. G. (1977): Atomic absorption spectrophotometric determination of Pb, Cd, Zn and Cu in clams and oysters. Collaboration study. J. A. O. A. C. 60, 1400-1407

CULLEN, J. M., B. H. RUEBNER (1991): A histopathologic classification of chemical-induced injury of the liver. In: Hepatotoxicology (Meeks, R. G., S. D. Harrison, R. J. Bull, Eds.). CRC Press. Boca Raton, Florida, USA. pp. 67-92.

CULLING, C. F. A. (1974): Handbook of Histopathological and Histochemical Techniques (Including Museum Techniques). 3rd ed. Butterworths. Trowbridge and Esher.

DELLMANN, H-D., E. M. BROWN (1976): Textbook of Veterinary Histology. Lea and Febiger. Philadelphia.

FORENBACHER, S. (1993): Klinička patologija probave i mijene tvari domaćih životinja, Svezak II., Jetra. Hrvatska akademija znanosti i umjetnosti, Školska knjiga. Zagreb.

GOMERČIĆ, H., Đ. HUBER, V. GOMERČIĆ, D. ŠKRTIĆ, A. GOMERČIĆ, S. VUKOVIĆ (1998): Dolphin morbilliviral infection from the Mediterranean Sea did not spread into the Adriatic Sea. Acta vet. Hungarica 46, 127-134.

HATCH, W. R., W. L. OTT (1968): Determination of submicrograms quantities of mercury by atomic absorption spectrophotometry. Anal. Chem. 40, 2085-2087.

JUNQUEIRA, L. C., J. CARNEIRO, R. O. KELLEY (1995): Osnove histologije. Školska knjiga. Zagreb.

KUIKEN, T., M. O'LEARY, J. BAKER, J. KIRKWOOD (1996): Pathology of harbour porpoises (*Phocoena phocoena*) from the coast of England, suspected of by-catch. In: Diagnosis of By-Catch in Cetaceans: Proceedings of the second European Cetacean Society workshop on cetacean pathology, Montpellier, France, 2 March 1994 (Kuiken, T., Ed.). European Cetacean Society. Saskatoon, Saskatchewan, Canada. pp. 31-34.

LIPSCOMB, T. P. (1996): Pathologic findings in dolphins known to have died from underwater entrapment. In: Diagnosis of By-Catch in Cetaceans: Proceedings of the second European Cetacean Society workshop on cetacean pathology, Montpellier, France, 2 March 1994 (Kuiken, T., Ed.). European Cetacean Society. Saskatoon, Saskatchewan, Canada. pp. 1-3.

MARZELLA, L., B. F. TRUPM (1991): Pathology of the liver: functional and structural alterations of hepatocyte organells induced by cell injury. In: Hepatotoxicology (Meeks, R. G., S. D. Harrison, R. J. Bull, Eds.). CRC Press. Boca Raton, Florida, USA. pp. 93-138.

MIYAI, K. (1991): Structural organization of the liver. In: Hepatotoxicology (Meeks, R. G., S. D. Harrison, R. J. Bull, Eds.). CRC Press. Boca Raton, Florida, USA. pp. 1-65.

MOSIMANN, W., T. KOHLER (1990): Verdauungsorgane. In: Zytologie, Histologie und mikroskopische Anatomie der Haussäugetiere (Mosimann, W., T. Kohler, Eds.). Verlag Paul Parey. Berlin and Hamburg.

PERRIN, W. (1975): Variations of Spotted and Spiner Porpoise (Genus Stenella) in the Eastern and Tropical Pacific and Hawaii. University of California Press. Berkeley, Los Angeles, London.

SIEBERT, U., H. BENKE, K. FRESE, F. PIRRO, R. LICK (1996): Postmortem examination of by-catches from German fisheries and of suspected by-catches found on the coast of Germany. In: Diagnosis of By-Catch in Cetaceans: Proceedings of the second European Cetacean Society workshop on cetacean pathology, Montpellier, France, 2 March 1994 (Kuiken, T., Ed.). European Cetacean Society. Saskatoon, Saskatchewan, Canada. pp. 27-30.

SIMPSON, J. G., M. B. GARDNER (1972): Comparative microscopic anatomy of selected marine mammals. In: Mammals of the Sea. Biology and Medicine (Ridgway, S. H., Ed.). Charles C. Thomas. Springfield, Illinois, U.S.A. pp. 298-418.

SLOOTEN, E. (1991): Age, growth, and reproduction in Hector's dolphins. Can. J. Zool. 69, 1689-1700.

STANLEY, P. I., G. R. ELLIOT (1976): An assessment based on residues in owls of environmental contamination arising from the use of mercury compounds in British agriculture. Agro-ecosystems 2, 223-234.

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GOMERČIĆ, H., Đ. HUBER, V. GOMERČIĆ, S. VUKOVIĆ, D. ŠKRTIĆ, T. GOMERČIĆ, V. DOBRANIĆ, H. LUCIĆ, M. ĐURAS, S. ĆURKOVIĆ, A. GOMERČIĆ, LJ. KARDOŠ: Masna jetra i potkožni edem slobodno živućeg dobrog dupina (*Tursiops truncatus*, Montagu 1821) iz Jadranskog mora; svjetlosno i elektronsko mikroskopsko istraživanje. Vet. arhiv 70, 259-277, 2000.

SAŽETAK

Istražena je jedna ženka dobrog dupina (*Tursiops truncatus*, Montagu 1821) koja je rađala više puta, a živjela je slobodno u Jadranskom moru te je uginula 8. listopada 1997. u blizini Šibenika. U času smrti bila je stara 21 godinu, mase 214 kg i dužine 288 cm. Neposredno prije smrti životinja je pokazivala neke poremetnje funkcije središnjeg živčanog sustava. Za kontrolu i usporedbu korištena su trupla i tkiva dva dobra dupina za koje se može pretpostaviti da su bili zdravi. Razudbom trupla uginule ženke dupina nađen je na dorzalnoj strani tijela životinje prostrani potkožni edem između potkožnog masnog omotača i mišićja. Edem je činila bistra svijetložučkasta međustanična tekućina. Nešto povećana količina iste takve tekućine je nađena i u seroznim šupljinama tijela ove oboljele i uginule ženke dupina, kao i lagani edem mozga. Najizrazitija promjena nađena je u građi jetre bolesne ženke dupina, koja je bila izrazito žute boje i prhka na dodir, ali normalne veličine. Struktura jetre bila je potpuno razorena, bez i najmanjih tragova krvi u jetri, te s jako izraženom masnom degeneracijom jetrenog parenhima. Svi hepatociti su bili razoreni s ostatcima nabubrenih mitohondrija i proširenog endoplazmatskog retikuluma. Ova sveobuhvatna degeneracija jetre bila je vjerojatno posljedica akutnog djelovanja nekog neutvrđenog biološkog ili kemijskog agensa, a izazvala je hepatargičnu krizu i smrt ove ženke dupina koja je živjela slobodno u Jadranskom moru.

Ključne riječi: dupin, dobri dupin, *Tursiops truncatus*, masna jetra, potkožni edem, svjetlosna mikroskopija, elektronska mikroskopija, histokemija, Jadransko more