DAFTAR PUSTAKA

1. Khellaf A, Khan DZ, Helmy A. Recent advances in traumatic brain injury. J Neurol. 2019;266(11):2878–89.
2. Singh R, Choudhri K, Sinha S, Mason S, Lecky F, Dawson J. Global outcome after traumatic brain injury in a prospective cohort. Clin Neurol Neurosurg. 2019;186:1–8.
3. Hanscom M, Loane DJ, Shea-Donohue T. Brain-gut axis dysfunction in the pathogenesis of traumatic brain injury. Journal of Clinical Investigation. 2021;131(12):1–15.
4. Pietro V Di, Yakoub K, Caruso G, Lazzarino G, Signoretti S. Antioxidant therapies in traumatic brain injury. Antioxidants. 2020;9(260):1–34.
5. Salim S. Oxidative stress and the central nervous system. Journal of Pharmacology and Experimental Therapeutics. 2017;360(1):201–5.
6. Forman HJ, Zhang H. Targeting oxidative stress in disease: promise and limitations of antioxidant therapy. Nat Rev Drug Discov. 2021;20(9):689–709.
7. Rahal A, Kumar A, Singh V, Yadav B, Tiwari R, Chakraborty S, et al. Oxidative stress, prooxidants, and antioxidants: The interplay. Biomed Res Int. 2014;2014:1–20.
8. Ayala A, Muñoz MF, Argüelles S. Lipid peroxidation: Production, metabolism, and signaling mechanisms of malondialdehyde and 4-hydroxy-2-nonenal. Oxid Med Cell Longev. 2014;2014:1–31.
9. Herrera E, Barbas C. Vitamin E: Action, metabolism and perspectives. J Physiol Biochem. 2001;57(1):43–56.
10. Medicine IO. Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids. Washington DC: The National Academies Press; 2000.
11. Gilgun-Sherki Y, Rosenbaum Z, Melamed E, Offen D. Antioxidant therapy in acute central nervous system injury: Current state. Pharmacol Rev. 2002;54(2):271–84.
12. Vonder Haar C, Peterson T, Martens K, Hoane M. Vitamins and Nutrients as Primary Treatments in Experimental Brain Injury: Clinical Implications for Nutraceutical Therapies. Brain Res. 2016;1640:114–29.
13. Hall ED, Vaishnav RA, Mustafa AG. Antioxidant Therapies for Traumatic Brain Injury. Neurotherapeutics. 2010;7(1):51–61.
14. Shen Q, Hiebert JB, Hartwell J, Thimmesch AR, Pierce JD. Systematic Review of Traumatic Brain Injury and the Impact of Antioxidant Therapy on Clinical Outcomes. Worldviews Evid Based Nurs. 2016;13(5):380–9.
15. Horn SD, Kinikini M, Moore LW, Hammond FM, Brandstater ME, Smout RJ, et al. Enteral nutrition for patients with traumatic brain injury in the rehabilitation setting: Associations with patient preinjury and injury characteristics and outcomes. Arch Phys Med Rehabil. 2015;96(8):S245–55.
16. Erdman J, Oria M, Pillsbury L. Nutrition and Traumatic Brain Injury: Improving Acute and Subacute Health Outcomes in Military Personnel. Institute of Medicine (US) Committee on Nutrition, Trauma, and the Brain; 2011.

17. Wu A, Ying Z, Gomez-Pinilla F. Vitamin e protects against oxidative damage and learning disability after mild traumatic brain injury in rats. Neurorehabil Neural Repair. 2010;24(3):290–8.
18. Inci S, Özcan OE, Kilinç K. Time-level relationship for lipid peroxidation and the protective effect of α-tocopherol in experimental mild and severe brain injury. Neurosurgery. 1998;43(2):330–6.
19. Conte V, Uryu K, Fujimoto S, Yao Y, Rokach J, Longhi L, et al. Vitamin E reduces amyloidosis and improves cognitive function in Tg2576 mice following repetitive concussive brain injury. J Neurochem. 2004;90(3):758–64.
20. Yang J, Han Y, Ye W, Liu F, Zhuang K, Wu G. Alpha tocopherol treatment reduces the expression of Nogo-A and NgR in rat brain after traumatic brain injury. Journal of Surgical Research. 2013;182(2):e69–77.
21. Ishaq GM, Saidu Y, Bilbis LS, Muhammad SA, Jinjir N, Shehu BB. Effects of α-tocopherol and ascorbic acid in the severity and management of traumatic brain injury in albino rats. J Neurosci Rural Pract. 2013;4(3):292–7.
22. Nathens AB, Neff MJ, Jurkovich GJ, Klotz P, Farver K, Ruzinski JT, et al. Randomized, prospective trial of antioxidant supplementation in critically III surgical patients. Ann Surg. 2002;236(6):814–22.
23. Berger MM, Soguel L, Shenkin A, Revelly JP, Pinget C, Baines M, et al. Influence of early antioxidant supplements on clinical evolution and organ function in critically ill cardiac surgery, major trauma, and subarachnoid hemorrhage patients. Crit Care. 2008;12(4):1–13.
24. Razmkon A, Sadidi A, Sherafat-Kazemzadeh E, Mehrafshan A, Jamali M, Malekpour B, et al. Administration of vitamin C and vitamin E in severe head injury: A randomized double-blind controlled trial. Clin Neurosurg. 2011;58:133–7.
25. Park GJ, Ro YS, Yoon H, Lee SGW, Jung E, Moon SB, et al. Serum vitamin E level and functional prognosis after traumatic brain injury with intracranial injury: A multicenter prospective study. Front Neurol. 2022;13:1–11.
26. Khalili H, Abdollahifard S, Niakan A, Aryaie M. The effect of Vitamin C and E on clinical outcomes of patients with severe traumatic brain injury: A propensity score matching study. Surg Neurol Int. 2022;2013(548):1–7.
27. Daia C, Scheau C, Spinu A, Andone I, Popescu C, Toader C, et al. Modulated neuroprotection in unresponsive wakefulness syndrome after severe traumatic brain injury. Brain Sci. 2021;11(8):1–13.
28. Riffel APK, Santos MCQ, De Souza JA, Scheid T, Horst A, Kolberg C, et al. Treatment with ascorbic acid and α-tocopherol modulates oxidative-stress markers in the spinal cord of rats with neuropathic pain. Brazilian Journal of Medical and Biological Research. 2018;51(4):1–11.
29. Bulama I, Nasiru S, Bilbis S, Abbas A, Nasiru J. Ascorbic acid treatment modulated traumatic brain injury-induced oxidative stress and neuropathic pain in rats. J Cell Neurosci Oxid Stress. 2020;12(1):922–36.
30. Sharma P, Halder S. Cognition, Quality Of Life And Mood State In Mild Traumatic Brain Injury: A Case Study. Indian Journal of Mental Health. 2020;8(1):112–6.

31. Dixon J, Comstock G, Whitfield J, Richards D, Burkholder TW, Leifer N, et al. Emergency department management of traumatic brain injuries: A resource tiered review. African Journal of Emergency Medicine. 2020;10(3):159–66.
32. Mckee AC, Daneshvar DH. The neuropathology of traumatic brain injury. 1st ed. Vol. 127, Handbook of Clinical Neurology. Elsevier B.V.; 2015. 45–66 p.
33. Blennow K, Brody DL, Kochanek PM, Levin H, Mckee A, Ribbers GM, et al. Traumatic brain injuries. Nature Publishing Group. 2016;2:1–19.
34. Galgano M, Toshkezi G, Qiu X, Russell T, Chin L, Zhao L. Traumatic Brain Injury: Current Treatment Strategies and Future Endeavors. Cell Transplant. 2017;26(7):1118–30.
35. Yamamoto S, DeWitt DS, Prough DS. Impact & blast traumatic brain injury: Implications for therapy. Molecules. 2018;23(2):1–11.
36. Kaur P, Sharma S. Recent Advances in Pathophysiology of Traumatic Brain Injury. Curr Neuropharmacol. 2018;16:1224–38.
37. Dixon KJ. Pathophysiology of Traumatic Brain Injury. Phys Med Rehabil Clin N Am. 2017;28(2):215–25.
38. Schmidt OI, Infanger M, Heyde CE, Ertel W, Stahel PF. The role of neuroinflammation in traumatic brain injury. European Journal of Trauma. 2004;30(3):135–49.
39. Ismail H, Shakkour Z, Tabet M, Abdelhady S, Kobaisi A, Abedi R, et al. Traumatic brain injury: Oxidative stress and novel anti-oxidants such as mitoquinone and edaravone. Antioxidants. 2020;9(10):1–18.
40. Bains M, Hall E. Antioxidant therapies in traumatic brain and spinal cord injury*. Biochim Biophys Acta. 2012;1822(5):675–84.
41. Poprac P, Jomova K, Simunkova M, Kollar V, Rhodes CJ, Valko M. Targeting Free Radicals in Oxidative Stress-Related Human Diseases. Trends Pharmacol Sci. 2017;38(7):592–607.
42. Azad MB, Chen Y, Gibson SB. Regulation of autophagy by reactive oxygen species (ROS): Implications for cancer progression and treatment. Vol. 11, Antioxidants and Redox Signaling. Mary Ann Liebert Inc.; 2009. p. 777–90.
43. Domijan AM, Ralić J, Radić Brkanac S, Rumora L, Žanić-Grubišić T. Quantification of malondialdehyde by HPLC-FL - application to various biological samples. Biomedical Chromatography. 2015;29(1):41–6.
44. Rahaman P, Del Bigio MR. Histology of Brain Trauma and Hypoxia-Ischemia. Vol. 8, Academic Forensic Pathology. SAGE Publications Inc.; 2018. p. 539–54.
45. Grafe MR, Woodworth KN, Noppens K, Regino Perez-Polo J. Long-term histological outcome after post-hypoxic treatment with 100% or 40% oxygen in a model of perinatal hypoxic-ischemic brain injury. Intl J of Devlp Neuroscience. 2008;26(1):119–24.
46. Watson BD. Evaluation of the concomitance of lipid peroxidation in experimental models of cerebral ischemia and stroke. Prog Brain Res. 1993;96:69–95.
47. Chan P. Role of Oxidants in Ischemic Brain Damage. Stroke. 1996;27(6):1124–9.

48. Bayir H, Kochanek PM, Kagan VE. Oxidative stress in immature brain after traumatic brain injury. Dev Neurosci. 2006;28(4–5):420–31.
49. Halliwell B. Reactive Oxygen Species and the Central Nervous System. J Neurochem. 1992;59(5):1609–23.
50. Bayir H, Kagan VE, Borisenko GG, Tyurina YY, Janesko KL, Vagni VA, et al. Enhanced oxidative stress in iNOS-deficient mice after traumatic brain injury: Support for a neuroprotective role of iNOS. Journal of Cerebral Blood Flow and Metabolism. 2005;25(6):673–84.
51. Kochanek PM, Dixon CE, Shellington DK, Shin SS, Bayir H, Jackson EK, et al. Screening of biochemical and molecular mechanisms of secondary injury and repair in the brain after experimental blast-induced traumatic brain injury in rats. J Neurotrauma. 2013;30(11):920–37.
52. Tavazzi B, Signoretti S, Lazzarino G, Amorini AM, Delfini R, Cimatti M, et al. Cerebral oxidative stress and depression of energy metabolism correlate with severity of diffuse brain injury in rats. Neurosurgery. 2005;56(3):582–8.
53. Ellis EM. Reactive carbonyls and oxidative stress: Potential for therapeutic intervention. Pharmacol Ther. 2007;115(1):13–24.
54. Al Nimer F, Ström M, Lindblom R, Aeinehband S, Bellander BM, Nyengaard JR, et al. Naturally occurring variation in the glutathione-s-transferase 4 gene determines neurodegeneration after traumatic brain injury. Antioxid Redox Signal. 2013;18(7):784–94.
55. Moss R. Free Radical: Albert Szent-Gyorgyi and the Battle over Vitamin C. J Hist Biol. 1989;22:180–1.
56. Ball G. Vitamins: Their Role in the Human Body. 1st ed. International Journal of Food Science & Technology. Oxford: Blackwell Publishing; 2004.
57. Davey MW, Van Montagu M, Inzé D, Sanmartin M, Kanellis A, Smirnoff N, et al. Plant L-ascorbic acid: Chemistry, function, metabolism, bioavailability and effects of processing. J Sci Food Agric. 2000;80(7):825–60.
58. Sotiriou S, Gispert S, Cheng J, Wang Y, Chen A, Hoogstraten-Miller S, et al. Ascorbic-acid transporter Slc23a1 is essential for vitamin C transport into the brain and for perinatal survival. Nat Med. 2002;8(5):514–7.
59. Harrison FE, Dawes SM, Meredith ME, Babaev VR, Li L, May JM. Low vitamin C and increased oxidative stress and cell death in mice that lack the sodium-dependent vitamin C transporter SVCT2. Free Radic Biol Med. 2010;49(5):821–9.
60. Hammarstrom L. Autoradiographic studies on the distribution of C14-labelled ascorbic acid and dehydroascorbic acid. Acta Physiol Scand. 2008;70(s289):1–83.
61. Lykkesfeldt J. Ascorbate and dehydroascorbic acid as reliable biomarkers of oxidative stress: Analytical reproducibility and long-term stability of plasma samples subjected to acidic deproteinization. Cancer Epidemiology Biomarkers and Prevention. 2007;16(11):2513–6.
62. Lykkesfeldt J, Loft S, Nielsen B, Poulsen E. Ascorbic acid and dehydroascorbic acid as biomarkers of oxidative stress caused by smoking. Am J Clin Nutr. 1997;65:959–63.

63. Kuo CH, Hata F, Yoshida H, Yamatodani A, Wada H. Effect of ascorbic acid on release of acetylcholine from synaptic vesicles prepared from different species of animals and release of noradrenaline from synaptic vesicles of rat brain. Life Sci. 1979;24(10):911–5.
64. Sies H, Stahl W. Vitamin E and C, beta-carotene, and other carotenoids as antioxidants. Am J Clin Nutr. 1995;62:1315S-21S.
65. Tsukaguchi H, Tokui T, Mackenzie B, Berger U, Chen X, Wang Y. A family of mammalian Na+-dependent L-ascorbic acid transporters. Nature. 1999;399:70–5.
66. Lindblad M, Tveden-Nyborg P, Lykkesfeldt J. Regulation of vitamin C homeostasis during deficiency. Nutrients. 2013;5(8):2860–79.
67. Wilson JX. Regulation of vitamin C transport. Annu Rev Nutr. 2005;25:105–25.
68. Hasselholt S, Tveden-Nyborg P, Lykkesfeldt J. Distribution of vitamin C is tissue specific with early saturation of the brain and adrenal glands following differential oral dose regimens in guinea pigs. British Journal of Nutrition. 2015;113(10):1539–49.
69. Nishikimi M, Yagi K. Molecular basis for the deficiency in humans of gulonolactone oxidase , a key enzyme for ascorbic acid biosynthesis. Am J Clin Nutr. 1991;54:1203S-1208S.
70. Gęgotek A, Skrzydlewska E. Antioxidative and Anti-Inflammatory Activity of Ascorbic Acid. Antioxidants. 2022;11(10):1–18.
71. Levine M, Conry-Cantilena C, Wang Y, Welch RW, Washko PW, Dhariwal KR, et al. Vitamin C pharmacokinetics in healthy volunteers: Evidence for a recommended dietary allowance. Proc Natl Acad Sci U S A. 1996;93(8):3704–9.
72. Pinnell SR, Yang H, Omar M, Riviere NM, DeBuys H V., Walker LC, et al. Topical L-ascorbic acid: Percutaneous absorption studies. Dermatologic Surgery. 2001;27(2):137–42.
73. Rose R. Transport of ascorhic acid and other water-soluble vitamhls.pdf. Biochimica et Biophvsico Acta. 1988;947:335–66.
74. MacDonald L, Thumser AE, Sharp P. Decreased expression of the vitamin C transporter SVCT1 by ascorbic acid in a human intestinal epithelial cell line. British Journal of Nutrition. 2002;87(2):97–100.
75. Levine M, Conry-Cantilenat C, Wang Y, Welch RW, Washko PW, Dhariwal KR, et al. 20892; §Division of Clinical Pharmacology, Uniformed Services University of the Health Sciences. Vol. 93, Medical SciencesMolecular and Clinical Nutrition Section, Building. Warren Grant Magnuson Clinical Center; 1996.
76. Levine M, Wang Y, Padayatty SJ, Morrow J. A new recommended dietary allowance of vitamin C for healthy young women [Internet]. Vol. 14, National Institutes of Health. 2001. Available from: www.pnas.orgcgidoi10.1073pnas.171318198
77. Nielsen TK, Højgaard M, Andersen JT, Poulsen HE, Lykkesfeldt J, Mikines KJ. Elimination of ascorbic acid after high-dose infusion in prostate cancer patients: A pharmacokinetic evaluation. Basic Clin Pharmacol Toxicol. 2015 Apr 1;116(4):343–8.

78. Davis JL, Paris HL, Beals JW, Binns SE, Giordano GR, Scalzo RL, et al. Liposomal-encapsulated ascorbic acid: Influence on vitamin C bioavailability and capacity to protect against ischemia-reperfusion injury. Nutr Metab Insights. 2016 Jun 20;9:25–30.
79. Stephenson CM, Levin RD, Spector T, Lis CG. Phase i clinical trial to evaluate the safety, tolerability, and pharmacokinetics of high-dose intravenous ascorbic acid in patients with advanced cancer. Cancer Chemother Pharmacol. 2013 Jul;72(1):139–46.
80. Kodentsova V. Gradation in the level of vitamin consumption: Possible risk of excessive consumption. Vopr Pitan. 2014;83:41–51.
81. Lamarche J, Nair R, Peguero A, Courville C. Vitamin C-Induced Oxalate Nephropathy. Int J Nephrol. 2011;2011:1–4.
82. Abdullah M, Jamil R, Attia F. Vitamin C (Ascorbic Acid) [Internet]. StatPearls. 2022 [cited 2023 Jan 25]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK499877/
83. Leichtle SW, Sarma AK, Strein M, Yajnik V, Rivet D, Sima A, et al. High-Dose Intravenous Ascorbic Acid: Ready for Prime Time in Traumatic Brain Injury? Neurocrit Care. 2020;32(1):333–9.
84. Reang J, Sharma PC, Thakur VK, Majeed J. Understanding the therapeutic potential of ascorbic acid in the battle to overcome cancer. Biomolecules. 2021;11(8):1–24.
85. Vissers MCM, Das AB. Potential mechanisms of action for vitamin C in cancer: Reviewing the evidence. Front Physiol. 2018;9:1–13.
86. Ngo B, Riper J, Cantley L, Yun J. Targeting cancer vulnerabilities with high-dose vitamin C. Nat Rev Cancer. 2019;19(5):271–82.
87. Bruno RS, Leonard SW, Atkinson J, Montine TJ, Ramakrishnan R, Bray TM, et al. Faster plasma vitamin E disappearance in smokers is normalized by vitamin C supplementation. Free Radic Biol Med. 2006;40(4):689–97.
88. Jacquens A, Needham EJ, Zanier ER, Degos V, Gressens P, Menon D. Neuro-Inflammation Modulation and Post-Traumatic Brain Injury Lesions: From Bench to Bed-Side. Int J Mol Sci. 2022;23(19):1–36.
89. Ranard KM, Erdman JW. Effects of dietary RRR α-tocopherol vs all-racemic α-tocopherol on health outcomes. Nutr Rev. 2018;76(3):141–53.
90. Pizzino G, Irrera N, Cucinotta M, Pallio G, Mannino F, Arcoraci V, et al. Oxidative Stress: Harms and Benefits for Human Health. Oxid Med Cell Longev. 2017;2017:1–14.
91. Medina J, Gupta V. Vitamin E. StatPearls. 2022.
92. Mohd Zaffarin AS, Ng SF, Ng MH, Hassan H, Alias E. Pharmacology and pharmacokinetics of Vitamin E: Nanoformulations to enhance bioavailability. Int J Nanomedicine. 2020;15:9961–74.
93. Miyazawa T, Burdeos GC, Itaya M, Nakagawa K, Miyazawa T. Vitamin E: Regulatory Redox Interactions. IUBMB Life. 2019;71(4):430–41.
94. Lee GY, Han SN. The role of vitamin E in immunity. Nutrients. 2018;10(11):1–18.
95. Lee P, Ulatowski LM. Vitamin E: Mechanism of transport and regulation in the CNS. IUBMB Life. 2019;71(4):424–9.
96. Wu JH, Croft KD. Vitamin E metabolism. Mol Aspects Med. 2007;28(5–6):437–52.

97. Traber M, Arai H. Molecular Mechanisms of Vitamin E Transport. Annu Rev Nutr1. 1999;19:343–55.
98. Bendich A, Machlin L. Safety of oral intake of vitamin E. Am J Clin Nutr. 1988;48:612–9.
99. Kappus H, Diplock AT. Tolerance and safety of vitamin E: A toxicological position report. Free Radic Biol Med. 1992;13(1):55–74.
100. Haghnejad Azar A, Oryan S, Bohlooli S, Panahpour H. Alpha-Tocopherol Reduces Brain Edema and Protects Blood-Brain Barrier Integrity following Focal Cerebral Ischemia in Rats. Medical Principles and Practice. 2017;26(1):17–22.
101. Kornbrust DJ, Mavis RD. Relative susceptibility of microsomes from lung, heart, liver, kidney, brain and testes to lipid peroxidation: Correlation with vitamin E content. Lipids. 1980;15(5):315–22.
102. Hennig B, Enoch C, Chow C. PROTECTION BY VITAMIN E AGAINST ENDOTHELIAL CELL INJURY BY LINOLEIC ACID HYDROPEROXIDES. Nutrition Research. 1987;7:1253–9.
103. Rana A, Singh S, Deshmukh R, Kumar A. Pharmacological potential of tocopherol and doxycycline against traumatic brain injury-induced cognitive/motor impairment in rats. Brain Inj. 2020;34(8):1039–50.
104. Wu A, Ying Z, Gomez-Pinilla F. Vitamin e protects against oxidative damage and learning disability after mild traumatic brain injury in rats. Neurorehabil Neural Repair. 2010;24(3):290–8.
105. Dobrovolny J, Smrcka M, Bienertova-Vasku J. Therapeutic potential of vitamin E and its derivatives in traumatic brain injury-associated dementia. Neurological Sciences. 2018;39(6):989–98.
106. McDonald BZ, Gee CC, Kievit FM. The Nanotheranostic Researcher's Guide for Use of Animal Models of Traumatic Brain Injury. Journal of Nanotheranostics. 2021 Dec 6;2(4):224–68.
107. Xiong Y, Mahmood A, Chopp M. Animal models of traumatic brain injury. Vol. 14, Nature Reviews Neuroscience. 2013. p. 128–42.
108. Xu L, Nguyen J V., Lehar M, Menon A, Rha E, Arena J, et al. Repetitive mild traumatic brain injury with impact acceleration in the mouse: Multifocal axonopathy, neuroinflammation, and neurodegeneration in the visual system. Exp Neurol. 2016 Jan 1;275:436–49.
109. Romine J, Gao X, Chen J. Controlled cortical impact model for traumatic brain injury. Journal of Visualized Experiments. 2014 Aug 5;90:1–5.
110. Cernak I, Vink R, Zapple DN, Cruz MI, Ahmed F, Chang T, et al. The pathobiology of moderate diffuse traumatic brain injury as identified using a new experimental model of injury in rats. Neurobiol Dis. 2004 Oct;17(1):29–43.
111. Chen YC, Mao H, Yang KH, Abel T, Meaney DF. A modified controlled cortical impact technique to model mild traumatic brain injury mechanics in mice. Front Neurol. 2014;5:1–14.
112. Namjoshi DR, Cheng WH ang, McInnes KA, Martens KM, Carr M, Wilkinson A, et al. Merging pathology with biomechanics using CHIMERA (Closed-Head Impact Model of Engineered Rotational Acceleration): a novel, surgery-free model of traumatic brain injury. Mol Neurodegener. 2014;9:1–18.

Lampiran 1 : Data Hasil Penelitian Inhibisi Radikal Anion Superoksida

KADAR INHIBISI RADIKAL ANION SUPEROKSIDA SERUM DARAH TIKUS U/L					
Waktu Pemeriksaan	**No, Sampel**	**Kelompok A (Ascorbic Acid) 60 mg/kg**	**Kelompok B (α -Tocopherol) 60 mg/kg**	**Kelompok C (Ascorbic Acid 60 mg/kg dan α -Tocopherol 60 mg/kg)**	**Kelompok D (Kontrol Positif) mg/kg**
0 Jam (pre-perlakuan)	1	2444,91	2640,17	2263,59	2938,81
	2	2643,45	2807,54	2734,52	2647,56
	3	2919,12	2874,47	2137,24	2766,52
	4	2531,05	2874,47	2738,63	2525,31
	5	2338,25	3172,64	2893,69	2631,15
	6	2291,49	2736,16	2220,93	2647,56
3 Jam pasca COT	1	2959,32	3993,24	2850,46	2677,22
	2	2599,97	2710,73	3347,28	2478,23
	3	2869,08	4102,31	2628,69	2572,45
	4	2739,73	4654,9	2798,52	2828,72
	5	2363,4	3684,55	2798,52	2743,43
	6	2466,24	4406,27	2526,13	2672,12
24 jam pasca COT (terminasi)	1	3906,27	3627,93	4270,41	2856,29
	2	3573,56	3212,96	3462,64	2628,71
	3	3839,71	3935,15	3526,46	2772,82
	4	3866,95	3841,31	4097,7	3074,47
	5	3892,39	3353,23	4108,43	2815,68
	6	3489	3977,88	3946,67	2735,63

Lampiran 2 : Data Hasil Penelitian Malondialdehyde

KADAR MDA SERUM DARAH TIKUS µmol/L					
Waktu Pemeriksaan	**No, Sampel**	**Kelompok A (Ascorbic Acid) 60 mg/kg**	**Kelompok B (α - Tocopherol) 60 mg/kg**	**Kelompok C (Ascorbic Acid 60 mg/kg dan α -Tocopherol 60 mg/kg)**	**Kelompok D (NaCl 0,9% - Kontrol Positif)**
0 Jam (pre-perlakuan)	1	1,72	2,18	1,66	1,53
	2	1,61	1,7	1,11	1,38
	3	0,96	1,65	1,12	1,12
	4	1,88	1,88	1,62	1,24
	5	1,5	1,45	1,73	1,84
	6	1,25	1,45	1,33	1,53
3 Jam pasca COT	1	1,61	1,08	1,18	1,43
	2	1,21	1,16	1,18	1,56
	3	0,87	1,05	1,21	1,39
	4	1,46	1,15	1,22	1,3
	5	0,99	1,18	1,08	1,95
	6	1	1,23	1,29	1,63
24 jam pasca COT (terminasi)	1	1,28	1,18	1,09	1
	2	1,18	1,18	0,56	1,45
	3	0,9	1,21	0,87	1,32
	4	1,04	1,22	1,19	1,56
	5	0,8	1,08	0,98	1,7
	6	1,12	1,29	0,98	1,73

Lampiran 3 : Data Hasil *Score* Gambaran Histopatologis

***Score* Gambaran Histopatologis**					
Waktu Pemeriksaan	**No. Sampel**	**Kelompok A (Ascorbic Acid) 60 mg/kg**	**Kelompok B (α - Tocopherol) 60 mg/kg**	**Kelompok C (Ascorbic Acid 60 mg/kg dan α -Tocopherol @ 60 mg/kg)**	**Kelompok D (NaCl 0.9% - Kontrol Positif)**
24 Jam (Terminasi)	1	**2**	**2**	2	3
	2	2	2	2	3
	3	1	2	1	3
	4	1	2	1	3
	5	3	2	1	3
	6	2	2	1	3

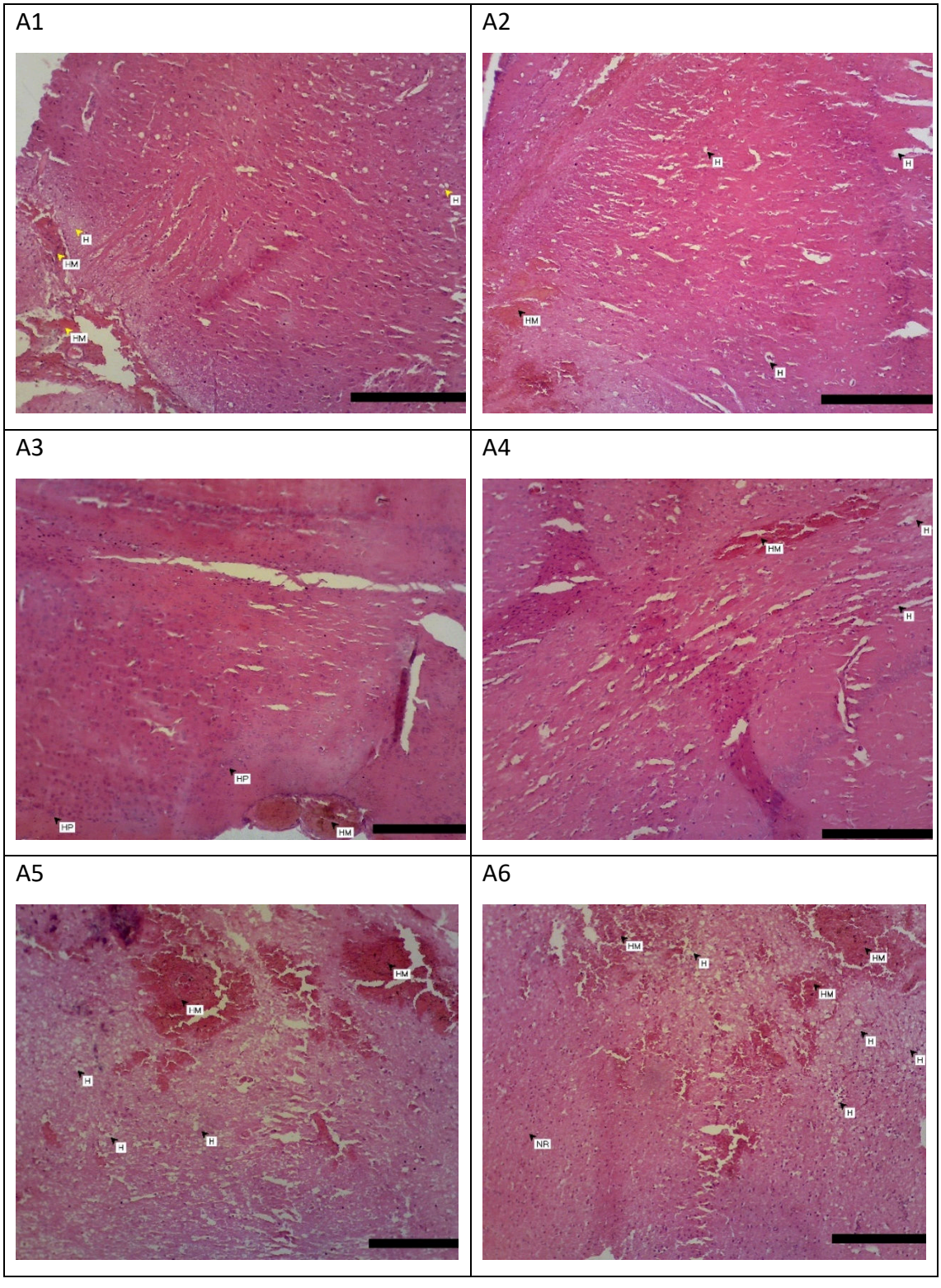
Kode sampel	*Score*	Interpretasi
A1	2	Adanya area (sel-sel) yang mengalami hipoksia (H), atrophy neuron (N), dan piknotik sel (PN). HE 400x. Disekitar area injury terdapat hemoragi (HM), area yang mengalami hipoksia (H), HE 100x
A2	2	Adanya area (sel-sel) yang mengalami hipoksia (H), Hemoragi pada area injury (HM). HE 400x Disekitar area injury terdapat hemoragi (HM), area yang mengalami hipoksia (H), HE 100x
A3	1	Adanya hemoragi pada area injury (HM), hyperplasia sel (HP). HE 100X
A4	1	Adanya area hemoragi (HM) dan area yang mengalami hipoksia (H). HE 100x

A5	3	Terdapat area yang mengalami hipoksia (H), dan hemoragi disekitar area injury (HM). HE 100x Terdapat gliosis (G) di jaringan, area hipoksia hampir diseluruh penampang jaringan.
A6	2	Terdapat area yang mengalami hipoksia (H), hemoragi (HM) disekitar area yang mengalami injury, dan area yang cenderung normal (NR). HE 100X Disekitar area injury terdapat hemoragi (HM), area yang mengalami hipoksia (H), HE 400x
B1	2	Terdapat area yang mengalami hipoksia (H), hemoragi (HM) disekitar area yang mengalami injury, dan area yang cenderung normal (NR). HE 100X Disekitar area injury terdapat hemoragi (HM), area yang mengalami hipoksia (H), HE 400x
B2	2	Terdapat area yang mengalami hipoksia (H), hemoragi (HM) disekitar area yang mengalami injury, he 100X
B3	2	Terdapat area yang mengalami hipoksia (H), disekitar area yang mengalami injury, he 100X
B4	2	Terdapat area yang mengalami hipoksia (H), hemoragi (HM) disekitar area yang mengalami injury, HE 100X
B5	2	Terdapat area yang mengalami hipoksia (H), hemoragi (HM) disekitar area yang mengalami injury, HE 100X
B6	2	Terdapat area yang mengalami hipoksia (H), hemoragi (HM) disekitar area yang mengalami injury, dan area yang cenderung normal HE 100X Disekitar area injury terdapat hemoragi (HM), area yang mengalami hipoksia (H), dan piknotik sel, HE 400x

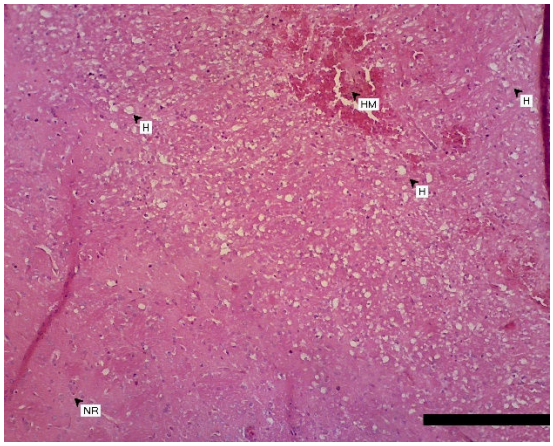
C1	2	Terdapat ar5a yang mengalami hipoksia (H), hemoragi (HM) disekitar area yang mengalami injury, he 100X
C2	2	Terdapat area yang mengalami hipoksia (H), hemoragi (HM) disekitar area yang mengalami injury, dan area yang cenderung normal HE 100X
C3	1	Terdapat area yang mengalami hipoksia (H), disekitar area yang mengalami injury, HE 100X
C4	1	Terdapat area yang mengalami hipoksia (H), hemoragi (HM) disekitar area yang mengalami injury, HE 100X
C5	1	Terdapat area yang mengalami hipoksia (H), hemoragi (HM) disekitar area yang mengalami injury, dan area yang cenderung normal HE 100X
C6	1	Terdapat area yang hemoragi (HM) disekitar area yang mengalami injury, dan area yang cenderung normal HE 100X
D1	2	Terdapat area yang mengalami hemoragi (HM), dan beberapa sel yang mengalami piknotik sel (P). HE 100x
D2	3	Terdapat area yang hemoragi (HM), area yang mengalami hipoksia (H) didaerah sekitar injury. HE 100x
D3	3	Terdapat area yang hemoragi (HM), dan terdapat area yang mengalami hipoksia (H) didaerah sekitar injury hampir diseluruh area. HE 100x
D4	3	Terdapat area yang hemoragi (HM), dan terdapat area yang mengalami hipoksia (H) didaerah sekitar injury hampir diseluruh area. HE 100x
D5	3	Terdapat area yang hemoragi (HM), dan terdapat area yang mengalami hipoksia (H) didaerah sekitar injury hampir diseluruh area, serta adanya gliosis (G). HE 100x

D6	3	Terdapat area yang hemoragi (HM), dan terdapat area yang mengalami hipoksia (H) didaerah sekitar injury hampir diseluruh area, serta adanya atrofi sel (A). HE 100X

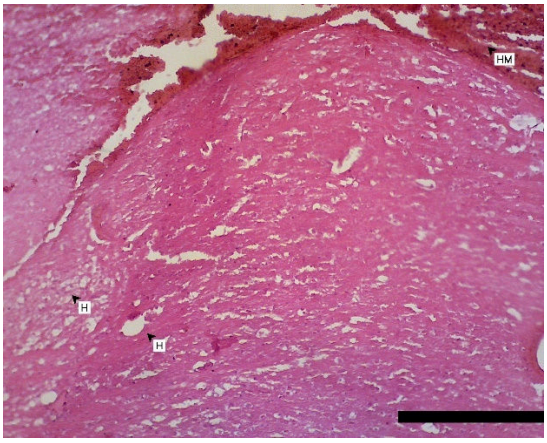
Lampiran 4 : Gambar Histopatologis Otak Tikus



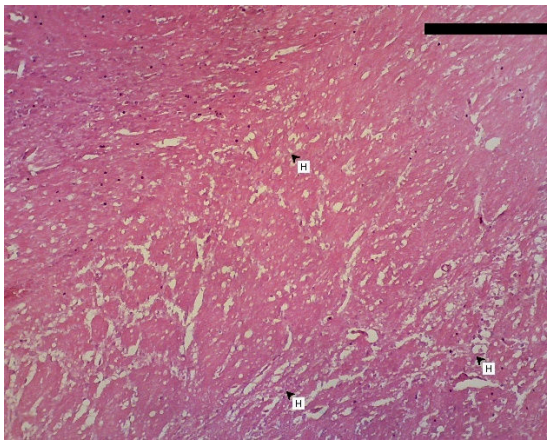
B1



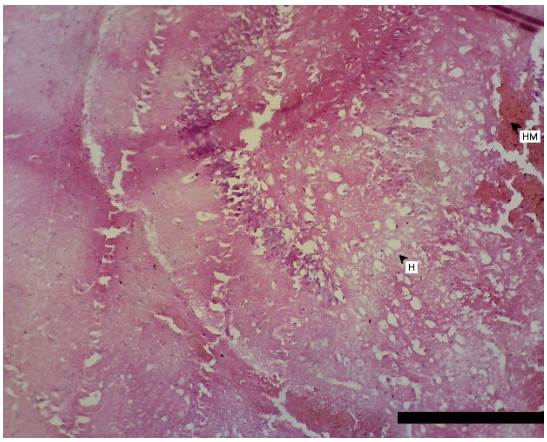
B2



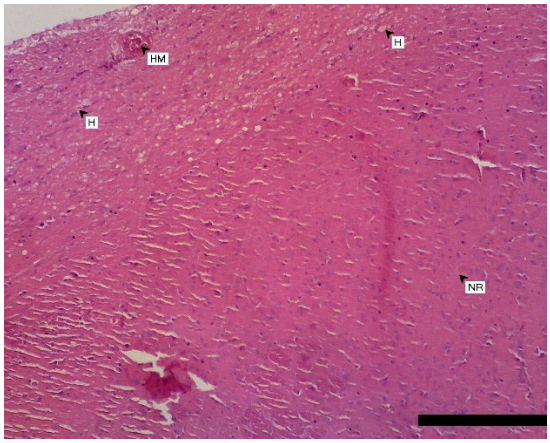
B3



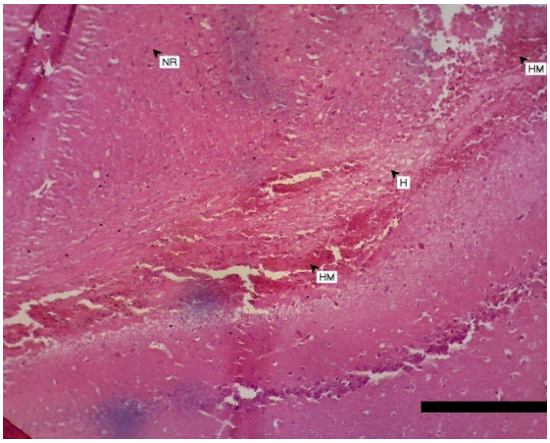
B4



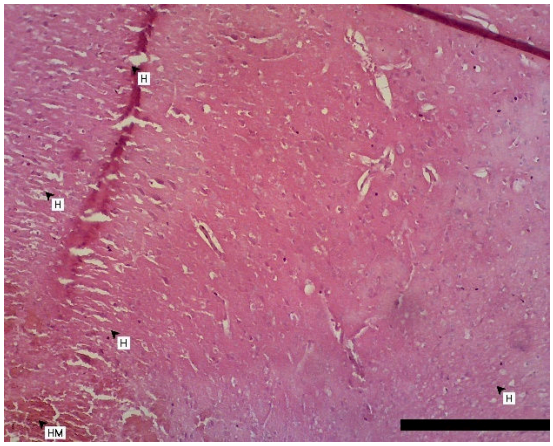
B5



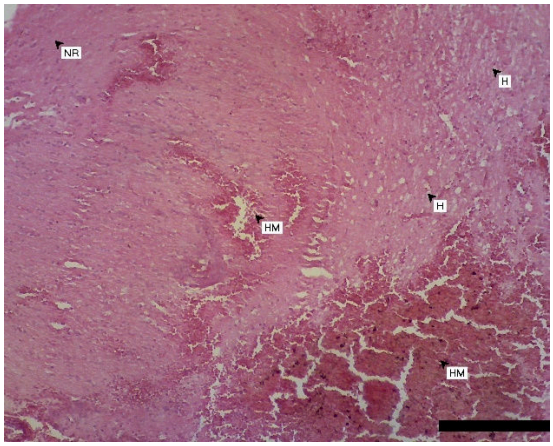
B6



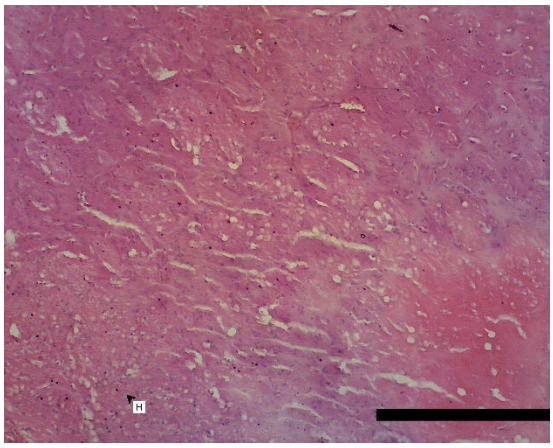
C1



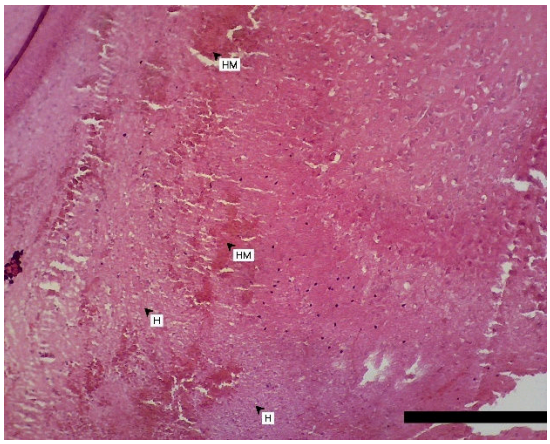
C2



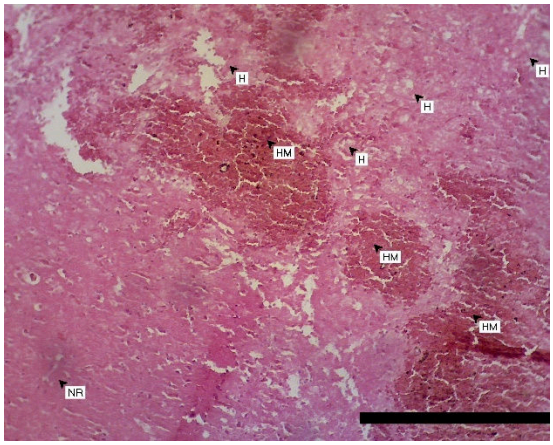
C3



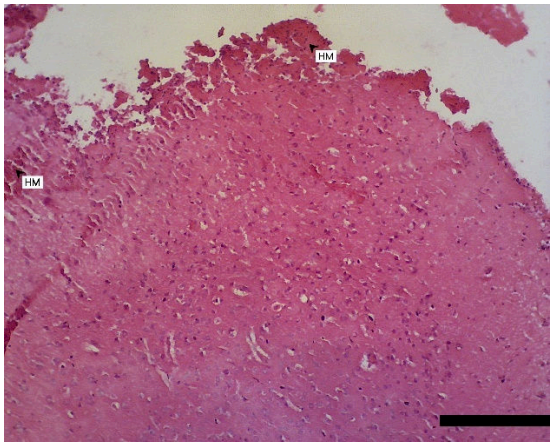
C4

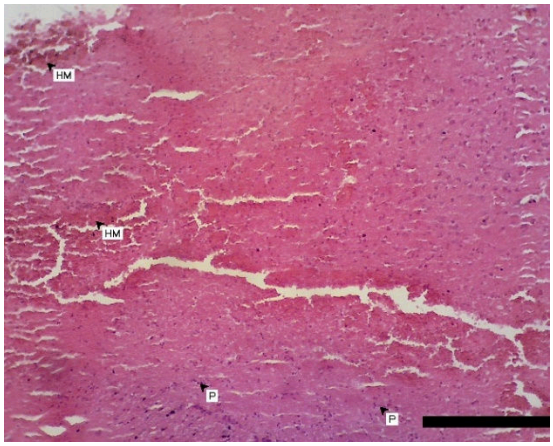
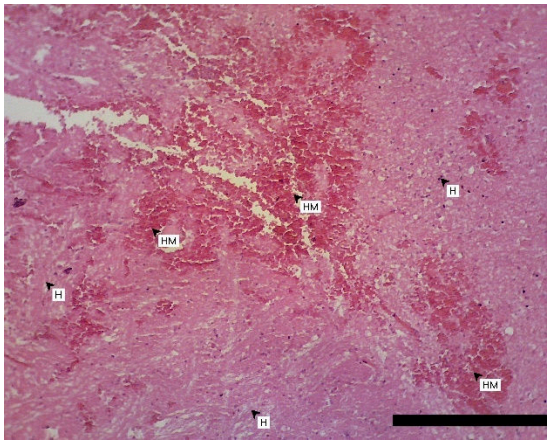
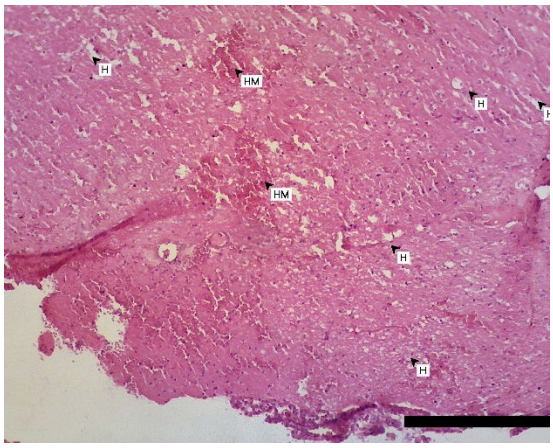
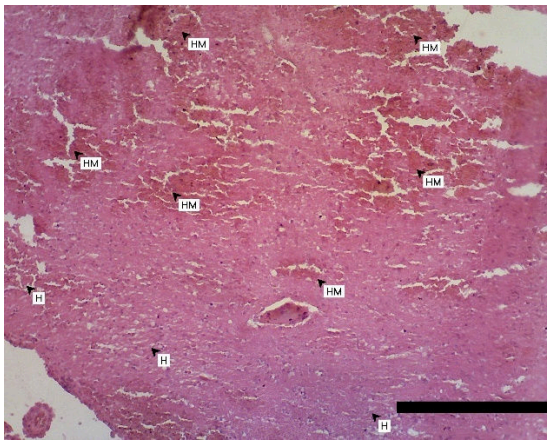
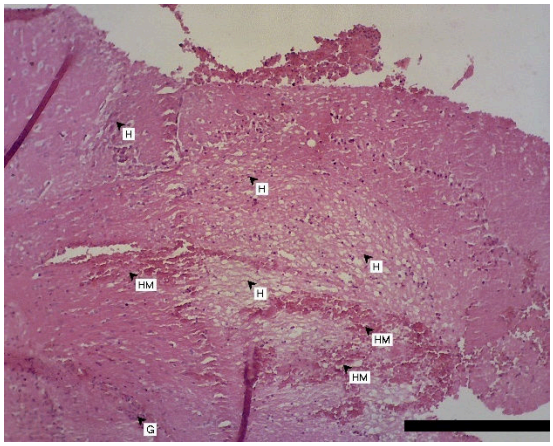


C5



C6



D1 	D2 
D3 	D4 
D5 	D6