ORIGINAL ARTICLE

Determination of trace element levels in patients with burst fractures

Shahab Ahmed Salih Gezh, M.D.,[1] Abdurrahman Aycan, M.D.,[2] Halit Demir, M.D.,[1] Cemal Bozlına, M.D.[3]

[1]Department of Chemistry, Yüzüncü Yıl University Faculty of Science and Literature, Van-*Turkey*
[2]Department of Neurosurgery, Yüzüncü Yıl University Faculty of Medicine, Van-*Turkey*
[3]Department of Neurosurgery, Van Training and Research Hospital, Van-*Turkey*

ABSTRACT

BACKGROUND: This study aimed to determine trace element levels (Zn, Fe, Mn, Mg, Cu, Cd, Co, and Pb) in patients with burst fractures in Van Province, Turkey.

METHODS: The study included a total of 44 participants with no additional pathologies, including 22 patients with burst fractures aged over 18 years who were admitted to the neurosurgery departments at two hospitals between June 15, 2015 and January 20, 2016 and 22 healthy volunteers. Serum samples were obtained from all participants to measure the serum levels of trace and heavy elements, including Mn, Cd, Cu, Pb, Fe, Co and Zn, using atomic absorbance spectrophotometry.

RESULTS: The trace element levels of Zn, Mn, Cu, Co, and Mg were significantly lower ($p<0.001$), whereas those of Fe, Cd, and Pb were significantly higher in the patient group than in the control group. In addition, the levels of Zn, Mn, Cu, Co, and Mg were lower and the levels of Fe, Cd, and Pb were higher in the patient group than in the control group.

CONCLUSION: The probability of burst fracture and its causes leading to any injury may be considered as an indicator balance for the concentration of trace elements between the patient group and control group and may also be a risk factor associated with the bone exposed to burst fracture Significant changes in serum levels of Zn, Cd, Mn, Mg, Pb, Fe, Cu and Zn elements can be observed in patients with burst fractures.

Keywords: Burst fracture; Cd; Co; Cu; Fe; Mg; Mn; Pb; Zn.

INTRODUCTION

Vertebral fractures predominantly occur in young people as a result of traumatic injuries and in elderly people as a result of osteoporosis.[1] Burst fractures account for 17% of all major vertebral fractures.[2,3] Of all spinal regions, burst fractures commonly occur in the thoracolumbar region due to its biomechanical properties and anatomical structure.[4,5]

The term "burst fracture" was first defined by Holdsworth.[6] The most frequent causes of burst fractures are motor vehicle accidents and falls from a height. The clinical criteria for spinal instability were first defined by Denise.[2] The anterior and middle vertebral columns are often damaged due to high-energy axial loading, leading to repositioning of the nucleus pulposus of the vertebral disc toward the corpus.[5] In some cases, bone fracture fragments can enter the spinal canal, causing neurological deficits as well as instability.[7–9] Neurological injury has been shown to occur in 30%–90% of patients with burst fracture due to damage in the proximal portion of the spinal cord, conus medullaris, or the filaments of the cauda equina.[10,11]

The treatment of burst fractures remains controversial. Some authors propose non-surgical methods, such as resting for a while, stability, or medical treatments, unless neurological

Cite this article as: Gezh SAS, Aycan A, Demir H, Bozlına C. Determination of trace element levels in patients with burst fractures. Ulus Travma Acil Cerrahi Derg 2018;24:244-248.

Address for correspondence: Abdurrahman Aycan, M.D.
Yüzüncü Yıl Üniversitesi Tıp Fakültesi, Nöroşirürji Anabilim Dalı, Van, Turkey
Tel: +90 432 - 444 50 65 E-mail: abdurrahmanaycan07@gmail.com

Ulus Travma Acil Cerrahi Derg 2018;24(3):244-248 DOI: 10.5505/tjtes.2017.08839 Submitted: 28.12.2016 Accepted: 07.09.2017 Online: 05.10.2017

deficits are presented.[12] On the other hand, the common view is that the stabilization of burst fractures should be achieved by emergency surgery, and compression on the neural tissue should be eliminated by enlarging the spinal canal if there are fractures inside the canal.[13,14] Moreover, surgery becomes unavoidable in the presence of neurological deficits.

Table 1. Demographic data, etiology and level of vertebral fracture

No	Age	Gender	Level	Etiology
1	36	Male	L1	γ
2	41	Male	T12	γ
3	39	Female	L3	∞
4	42	Female	L4	α
5	44	Female	L4	∞
6	40	Male	T7	α
7	38	Female	T9	∞
8	41	Male	L5	γ
9	41	Female	L1	∞
10	35	Female	L1	α
11	44	Female	T12	∞
12	38	Female	T12	γ
13	44	Male	T12	γ
14	37	Male	T11	α
15	40	Female	L3	α
16	40	Female	T5	γ
17	38	Female	L4	¥
18	43	Male	L1	γ
19	43	Female	L4	∞
20	40	Female	T12	γ
21	33	Female	T11	¥
22	41	Male	L1	γ

γ: Trauma; ∞: Falling from high; ¥: Spontaneous; α: Traffic accident.

MATERIALS AND METHODS

This study included a total of 44 participants with no additional pathologies, including 22 patients with burst fractures aged over 18 years who were admitted to the neurosurgery departments at Yuzuncu Yil University School of Medicine and Van Regional Training and Research Hospital between June 15, 2015 and January 20, 2016; 22 healthy volunteers were included as a control group (Table 1). The patient group included 8 (36.4%) women and 14 (63.6%) men with a mean age of 39.77±3.81 years, and the control group included 8 (36.4%) women and 14 (63.6%) men with a mean age of 40.5±0.473 years.

Biochemical Analysis

Blood samples (3 cm^3) were drawn from each participant in the morning after a 12-h fasting. Venous blood samples were placed in NF 800 tubes (Nüve-Bench Top Centrifuge, NF 800R, Ankara-Turkey) and were immediately stored in a freezer at 4°C. Blood samples were centrifuged at 5,000 rpm for 10 min to obtain serum. The serum samples to be used for the measurement of trace element levels were stored at −80°C until analysis. Serum samples were processed using atomic absorbance spectrophotometry at Central Research Laboratory of Yuzuncu Yil University.

Statistical Analysis

Table 2 presents the statistical evaluation of the concentrations of Zn, Fe, Mn, Mg, Cu, Cd, Co, and Pb using Error (Mean ± Standard Deviation) or (± SH) parameters. The serum levels of trace were compared in both groups.

RESULTS

The trace element levels of Zn, Mn, Cu, Co, and Mg were significantly lower (p<0.001), whereas those of Fe, Cd, and Pb were significantly higher in the patient group than in the control group. In addition, the levels of Zn, Mn, Cu, Co, and

Table 2. Trace element levels in the patient and control groups

	Control group (n=22)				Burst fracture patient group (n=22)				
	Mean	SD	Min.	Max.	Mean	SD	Min.	Max.	p
Zn (mg/L)	3.1849	0.1599	2.235	4.551	0.5699	0.0859	0.17	1.497	<0.0001
Fe (mg/L)	0.3026	0.0317	0.043	0.603	2.8893	0.3319	1.36	5.799	<0.0001
Mn (mg/L)	0.2932	0.0132	0.222	0.414	0.136	0.0124	0.04	0.2585	<0.0001
Mg (mg/L)	25.033	0.4345	22.162	30.435	7.6365	0.7432	3.24	14.2625	<0.0001
Cu (mg/L)	5.1564	0.2904	3.55	8.663	0.5552	0.0553	0.11	1.26	<0.0001
Cd (mg/L)	0.0067	0.0002	0.004	0.009	0.1408	0.0111	0.03	0.236	<0.0001
Co (mg/L)	0.4609	0.0338	0.28	0.93	0.1713	0.0274	0.03	0.4545	<0.0001
Pb (mg/L)	0.4048	0.0259	0.2123	0.6519	1.9719	0.0981	1.39	2.911	<0.0001

SD: Standard deviation; Min.: Minimum; Max.: Maximum.

Mg were lower and the levels of Fe, Cd, and Pb were higher in the patient group than in the control group.

DISCUSSION

Burst fractures constitute a type of traumatic spinal injury caused by severe compression of the vertebrae. These fractures typically result from severe traumas such as motor vehicle accidents, falls from a height, or assaults. Burst fractures can occur in any part of the vertebra. A vertically directed force on the spine may lead to vertebral fractures. Trace elements, in addition to being primary constituents of biological structures, have significant roles in various diseases. They also have a crucial role in the structure of bones. Numerous studies have investigated the concentrations of trace elements.[15,16] In the present study, the levels of certain trace elements (Zn, Fe, Mn, Mg, Cu, Cd, Co, and Pb) were measured in patients with burst fractures. The results revealed marked differences between the concentrations of these elements in patients with burst fractures and healthy volunteers.

The serum levels of all trace elements in patients with burst fractures are shown in Table 2. Zinc (Zn) is an essential nutritional microelement and plays a significant role in the growth of humans and animals.[17] As shown in Table 2, serum levels of Zn were lower in the patient group than in the healthy group. Although these findings were consistent with those reported by Cobanoglu et al. (2010),[11] Mazdak et al. (2010),[18] and Cebi et al. (2010),[10] they contradicted the findings reported for patients with malignant glioma by Arslan et al.[19] Although Zn has the ability to stimulate collagen synthesis in osteoblasts and activate alkaline phosphatase, these processes have a crucial role in the mineralization and calcification of bones.[20] Zn deficiency is commonly associated with numerous disorders such as retarded bone growth.[21] Intervertebral discs have the lowest Zn concentration because the peak concentrations start from the cartilage, bone, and posterior longitudinal ligaments.[22] Zn has also been shown to be effective in fracture healing. The primary role of Zn in fracture healing has been shown in diaphyseal tissues of rats that were removed 7 or 14 days after femoral diaphyseal fractures.[23]

The serum levels of Fe were higher in the patient group than in the control group. This finding was consistent with that reported by Cobanoglu et al. (2010)[11] and Arslan et al. (2011)[19] but contradicted the findings reported by Arslan et al. (2011).[24] Oxidative stress affects the mitochondrial DNA by impairing the balance.[25] Free Fe ions, which lead to the formation of reactive oxygen species (ROS), including superoxide and hydrogen peroxide, in mammalian cells, are released via the Fenton and Haber–Weiss reactions. Fe is a physiologically important trace element; however, it is biochemically hazardous. Fe is an important nutritional element for all living organisms, and low levels of Fe may have a role in the prevention of numerous diseases such as infection and cancer. Fe deficiency may also lead to various bone abnormalities in patients with thalassemia major.[26] The deposition of Fe in the bone marrow has been shown to cause delayed bone maturation and focal osteomalacia and to prevent local mineralization.[21]

In our study, the serum levels of Mn were lower in the patient group than in the healthy group. This finding was consistent with that reported by Onyeaghala et al.[16] and Asker et al.[24] but contradicted the findings reported by Cobanoglu et al.[11] and Arslan et al.[19] Mn is a trace mineral required for bone development, and it affects the activation of superoxide dismutase (SOD) and certain other enzymes that play a key role in antioxidant defense mechanisms.[27] Therefore, in patients with low serum levels of Mn, the antioxidant activity in the organs may be reduced and thus, these patients may become susceptible to various diseases. These findings were consistent with those reported by Mazdak et al.[18] Mn deficiency leads to numerous disorders in humans such as bone deficiency, joint pain, diabetes, and dysmnesia.

The serum levels of Mg were lower in the patient group than in the healthy group. This finding was consistent with that reported by Cobanoglu et al.[11] and Asker et al.[24] but contradicted the findings reported by Arslan et al.[19] More than 60% of Mg ions may accumulate in the bones and muscles and may also be present in hydroxyapatite surfaces. Because

Mg is diffused into the components of the body, the concentration of Mg in the bones is twice higher than that in the intervertebral disc (IVD).[28] Mg deficiency may result from reduced intestinal absorption and excessive urine retention, and it is also associated with osteoporosis, diabetes, hypertension, metabolic syndrome, nephropathies, and age-related diseases.[29] Moreover, Mg deficiency may also have a role in the progression of oxidative damage.

The serum levels of Cu were lower in the patient group than in the control group. This finding was consistent with that reported by Cobanoglu et al.,[11] Asker et al., and Onyeaghala et al. but contradicted the findings reported by Arslan et al.[5,17,19] Although free Cu+ is a potent oxidant, it may lead to certain diseases due to the production of ROS in the cells. Cu deficiency may result in osteoporosis and joint problems.[30] Although Cu deficiency may not affect the collagen content, it may lead to fragile bones in animals.[31]

The serum levels of Cd were lower in the patient group than in the control group. This finding was consistent with that reported by Kellen et al.[32] and Arslan et al.[19] The amount of toxic Cd in the bones and cartilage is three times higher than that in the IVD because the end-plate forms a barrier for the removal of Cd.[28] Cd has been shown to generate free radicals via the Fenton reaction. However, in other reaction models, Cd can activate oxidative stress and produce superoxide and hydrogen peroxide radicals.[33] Cd is a mutagen in mammals.[34]

The serum levels of Co were lower in the patient group than in the control group. This finding was consistent with that reported by Angelova et al. but contradicted the findings reported by Cobanoglu et al.[11] and Arslan et al.[19,35] The serum levels of Co have been assessed in the cell culture of experimental animal and human models.[36] Furthermore, the serum levels of Co have been investigated in various organs of experimental rat models, such as testicular edema and mitochondrial cardiac muscle, and significant findings have been reported.[37]

The serum levels of Pb were higher in the patient group than in the control group. This result was consistent with the reference values reported in the literature.[16,11,19,24] Excessive exposure to Pb leads to accumulation in bone tissues, thereby leading to toxic levels. Moreover, increasing concentration of Pb may pose a serious risk. Although it is a severe toxic element, Pb has been shown to yield an age-related positive correlation.[28]

Conclusion

This study determined the levels of trace elements in patients diagnosed with burst fractures. There was a relationship between the levels of trace elements and burst fractures. Therefore, we considered that the levels of Zn, Fe, Mn, Mg, Cu, Cd, Co, and Pb may be beneficial parameters for the clinical evaluation and diagnosis of burst fractures. Nevertheless, further prospective studies are needed to investigate the relationship between the changes in Mn and Zn levels and burst fractures.

Conflict of interest: None declared.

REFERENCES

1. Hu R, Mustard CA, Epidemiology of incident spinal fracture in a complete population. Burns C. Spine (Phila Pa 1976) 1996;21:492–9.
2. Denis F. The three column spine and its significance in the classification of acute thoracolumbar spinal injuries. Spine (Phila Pa 1976) 1983;8:817–31. [CrossRef]
3. Wilcox RK, Boerger TO, Allen DJ, Barton DC, Limb D, Dickson RA, et al. A dynamic study of thoracolumbar burst fractures. J Bone Joint Surg Am 2003;85-A:2184–9. [CrossRef]
4. Özer F, Zileli M. Torakolomber travmalar. In: Zileli MA, Özer F, editors. Omurilik ve Omurga Cerrahisi. 1st ed. Izmir: Başsaray Basımevi; 1997. p. 548–71.
5. Magerl F, Aebi M, Gertzbein SD, Harms J, Nazarian S. A comprehensive classification of thoracic and lumbar injuries. Eur Spine J 1994;3:184–201. [CrossRef]
6. Holdsworth F. Fractures, dislocations, and fracture-dislocations of the spine. J Bone Joint Surg Am 1970;52:1534–51. [CrossRef]
7. Daffner RH, Daffner SD. Vertebral injuries: detection and implications. Eur J Radiol 2002;42:100–16. [CrossRef]
8. Dai LY. Remodeling of the spinal canal after thoracolumbar burst fractures. Clin Orthop Relat Res 2001:119–23. [CrossRef]
9. Meves R, Avanzi O. Correlation between neurological deficit and spinal canal compromisein 198 patients with thoracolumbar and lumbar fractures. Spine (Phila Pa 1976) 2005;30:787–91. [CrossRef]
10. Cebi A, Kaya Y, Gungor H, Demir H, Yoruk IH, Soylemez N, et al. Trace elements, heavy metals and vitamin levels in patients with coronary artery disease. Int J Med Sci 2011;8:456–60. [CrossRef]
11. Cobanoglu U, Demir H, Sayir F, Duran M, Mergan D. Some mineral, trace element and heavy metal concentrations in lung cancer. Asian Pac J Cancer Prev 2010;11:1383–8.
12. Boerger TO, Limb D, Dickson RA. Does 'canal clearance' affect neurological outcome after thoracolumbar burst fractures? J Bone Joint Surg Br 2000;82:629–35. [CrossRef]
13. Akbarnia BA, Crandall DG, Burkus K, Matthews T. Use of long rods and a short arthrodesis for burst fractures of the thoracolumbar spine. A long-term follow-up study. J Bone Joint Surg Am 1994;76:1629–35.
14. Sasso RC, Cotler HB. Posterior instrumentation and fusion for unstable fractures and fracture-dislocations of the thoracic and lumbar spine. A comparative study of three fixation devices in 70 patients. Spine (Phila Pa 1976) 1993;18:450–60. [CrossRef]
15. Piccinini L, Borella P, Bargellini A, Medici CI, Zoboli A. A case-control study on selenium, zinc, and copper in plasma and hair of subjects affected by breast and lung cancer. Biol Trace Elem Res 1996;51:23–30.
16. Onyeaghala AA, Oduwole O, Ozoyiegbu R, Ebesunun M, Ogiogwa OTrace elements in patients with thyrotoxicosis. International Journal of Applied Biology and Pharmaceutical Technology 2012;3:313–9.
17. Burch RE, Hahn HK, Sullivan JF. Newer aspects of the roles of zinc, manganese, and copper in human nutrition. Clin Chem 1975;21:501–20.
18. Mazdak H, Yazdekhasti F, Movahedian A, Mirkheshti N, Shafieian M. The comparative study of serum iron, copper, and zinc levels between bladder cancer patients and a control group. Int Urol Nephrol 2010;42:89–93. [CrossRef]
19. Arslan M, Demir H, Arslan H, Gokalp AS, Demir C. Trace elements, heavy metals and other biochemical parameters in malignant glioma patients. Asian Pac J Cancer Prev 2011;12:447–51.
20. Yamaguchi M, Inamoto K, Suketa Y. Effect of essential trace metals on bone metabolism in weanling rats: comparison with zinc and other metals' actions. Res Exp Med (Berl) 1986;186:337–42. [CrossRef]
21. Terpos E, Voskaridou E. Treatment options for thalassemia patients with osteoporosis. Ann N Y Acad Sci 2010;1202:237–43. [CrossRef]
22. Killilea DW, Maier JA. A connection between magnesium deficiency and aging: new insightsfrom cellular studies. Magnes Res 2008;21:77–82.
23. Barrera R, Schattner M, Gabovich N, Zhang J, Saeed M, Genao A, et al. Bacteremic episodes and copper/zinc ratio in patients receiving homeparenteral nutrition. Nutr Clin Pract 2003;18:529–32. [CrossRef]
24. Asker S, Asker M, Yeltekin AC, Aslan M, Demir H. Serum levels of trace minerals and heavy metals in severe obstructive sleep apnea patients: correlates and clinical implications. Sleep Breath 2015;19:547–52.
25. Walter PB, Knutson MD, Paler-Martinez A, Lee S, Xu Y, Viteri FE, et al. Iron deficiency and iron excess damage mitochondria and mitochondrial DNA in rats. Proc Natl Acad Sci U S A 2002;99:2264–9. [CrossRef]
26. Valko M, Leibfritz D, Moncol J, Cronin MT, Mazur M, Telser J. Free radicals and antioxidants in normal physiological functions and human disease. Int J Biochem Cell Biol 2007;39:44–84. [CrossRef]
27. Aschner JL, Aschner M. Nutritional aspects of manganese homeostasis. Mol Aspects Med 2005;26:353–62. [CrossRef]
28. Kubaszewski Ł, Zioła-Frankowska A, Frankowski M, Nowakowski A, Czabak-Garbacz R, Kaczmarczyk J, et al. Atomic absorption spectrometry analysis of trace elements in degenerated intervertebral disc tissue. Med Sci Monit 2014;20:2157–64. [CrossRef]
29. de Baaij JH, Hoenderop JG, Bindels RJ. Regulation of magnesium balance: lessons learned from human genetic disease. Clin Kidney J

2012;5:i15–24. [CrossRef]

30. Osredkar J, Sustar N. Copper and Zinc, Biological Role and Significance of Copper/Zinc Imbalance. J Clinic Toxicol 2011:S3:1–18. [CrossRef]
31. Stern BR, Solioz M, Krewski D, Aggett P, Aw TC, Baker S, et al. Copper and human health: biochemistry, genetics, and strategies for modeling dose-response relationships. J Toxicol Environ Health B Crit Rev 2007;10:157–222. [CrossRef]
32. Kellen E, Zeegers MP, Hond ED, Buntinx F. Blood cadmium may be associated with bladder carcinogenesis: the Belgian case-control study on bladder cancer. Cancer Detect Prev 2007;31:77–82. [CrossRef]
33. Durham TR, Snow ET. Metal ions and carcinogenesis. EXS 2006:97–130. [CrossRef]
34. Hayes CW, Conway WF, Walsh JW, Coppage L, Gervin AS. Seat belt injuries: radiologic findings and clinical correlation. Radiographics 1991;11:23–36. [CrossRef]
35. Angelova MG, Petkova-Marinova TV, Pogorielov MV, Loboda AN, Nedkova-Kolarova VN, Bozhinova AN.mTrace Element Status (Iron, Zinc, Copper, Chromium, Cobalt, and Nickel) in Iron-Deficiency Anaemia of Children under 3 Years. Anemia 2014;2014:718089. [CrossRef]
36. Tandon VR, Sharma S, Mahajan A, Bardi GH. Oxidative stress: a novel strategy in cancer treatment. J K Science 2005;7:1–3.
37. Anderson MB, Pedigo NG, Katz RP, George WJ. Histopathology of testes from mice chronically treated with cobalt. Reprod Toxicol 1992;6:41–50. [CrossRef]

ORİJİNAL ÇALIŞMA - *ÖZET*

Burst kırığı olan hastalarda bazı eser element seviyelerinin incelenmesi

Dr. Shahab Ahmed Salih Gezh,[1] Dr. Abdurrahman Aycan,[2] Dr. Halit Demir,[1] Dr. Cemal Bozlına[3]

[1]Yüzüncü Yıl Üniversitesi Fen Fakültesi, Kimya Anabilim Dalı, Van
[2]Yüzüncü Yıl Üniversitesi Tıp Fakültesi, Nöroşirürji Anabilim Dalı, Van
[3]Van Eğitim ve Araştırma Hastanesi, Nöroşirürji Kliniği, Van

AMAÇ: Bu çalışmada Van ilinde burst kırığı olan hastalarda, bazı eser element seviyelerini (Zn, Fe, Mn, Mg, Cu, Cd, Co ve Pb) araştırmayı amaçladık.
GEREÇ VE YÖNTEM: Bu çalışma, 15 Haziran 2015 ile 20 Ocak 2016 tarihleri arasındaki dönemde iki hastaneye başvuran burst kırığı olan hastalardan alınan serum örnekleri üzerinde yürütüldü. Çalışma, ek patolojileri olmayan toplam 44 katılımcıyı kapsadı. Bunlara, 18 yaş üzeri patlama kırıkları olan ve nöroşirürji bölümüne kabul edilen 22 hasta ve 22 sağlıklı gönüllü dahildi. Mn, Cd, Cu, Pb, Fe, Co ve Zn dahil olmak üzere eser element ve ağır metallerin serum seviyeleri Atomik Emilim Spektrofotometrisi (AAS) ile analiz edildi.
BULGULAR: Sonuçlar, Zn, Mn, Cu, Co ve Mg iz düzeylerinin anlamlı olarak düşük ($p<0.001$) olduğunu ve Fe, Cd ve Pb düzeylerinin hasta grubunda kontrol grubuna göre anlamlı olarak daha yüksek olduğunu ortaya koymuştur. Zn, Mn, Cu, Co ve Mg seviyelerinin daha düşük olduğu ve Fe, Cd ve Pb düzeylerinin kontrol grubuna göre hasta grubunda daha yüksek olduğu bulundu.
TARTIŞMA: Patlamanın olasılığı ve herhangi bir yaralanmaya neden olan sebepleri, hasta grubu ile sağlıklı grup arasındaki eser element konsantrasyonu için bir gösterge dengesi olarak düşünülebilir ve patlama kırığına maruz kalan kemik ile ilişkili bir risk faktörü olabilir. Burst kırığı etiyolojisinde Zn, Cd, Mn, Mg, Pb, Fe, Cu ve Zn düzeyleri önemli rol oynayabilir.
Anahtar sözcükler: Burst kırığı; CD; Co; Cu; Fe; Mg; Mn; Pb; Zn.

Ulus Travma Acil Cerrahi Derg 2018;24(3):244-248 doi: 10.5505/tjtes.2017.08839