INTRODUCTION
Parkinson's disease (PD) is a neurodegenerative disorder characterized by bradykinesia, tremor, rigidity, and postural instability which progress through the loss of dopaminergic neurons (1). Progressive loss of dopaminergic neurons at the substantia nigra pars compacta is responsible for the pathophysiology (2). Postmortem studies have shown the essential role of oxidative stress in neuronal degeneration of the dopaminergic nigral neurons (3).

Recent reports describe nesfatin-1 and glucagon-like peptide-1 (GLP-1) as molecules with neuroprotective property that relieve oxidative stress. Here we aimed to determine the blood levels of nesfatin-1 and GLP-1 in PD and their effect on oxidative processes.

MATERIALS AND METHOD
In this study, 40 patients with Parkinson, followed-up at the Department of Neurology of Mugla Sityki Kocman University Training and Research Hospital were enrolled, as well as 40 age- and sex-matched cases as a control group. For the purpose of clinical evaluation of PD, Unified Parkinson’s Disease Rating Scale (UPDRS) and Hoehn-Yahr staging were applied while Addenbrooke’s Cognitive Examination was used to conduct a cognitive assessment. Blood samples collected from patient and control groups were tested to measure nesfatin-1, GLP-1, TAS (total antioxidant status), and TOS (total oxidant status) levels. TAS and TOS levels were detected by a spectrophotometric measurement and ELISA method was used to determine nesfatin-1 and GLP-1 levels.

RESULTS
Mean GLP-1 and nesfatin-1 values of Parkinson patients were lower than those of the control group (p<0.001, Welch t-test), whereas their mean TOS values were higher (p<0.001, Welch t-test). Mean TAS measurements, on the other hand, did not reveal any statistically significant difference between the patient group and the control group (p=0.51, Welch t-test) (Table 1).

DISCUSSION
Parkinson's disease is a progressive neurodegenerative disorder characterized by tremor, rigidity, bradykinesia, and postural instability during which dopaminergic neurons at the substantia nigra are lost (4-5). Postmortem studies have shown the essential role of oxidative stress in neuronal degeneration. Studies also have demonstrated that substantia nigra of Parkinson patients has an increased oxidative damage of lipids, proteins, and DNA (3). Likewise, in our study, TOS values of Parkinson patients found to be higher than that of the control group which is in parallel to the hypothesis of increased oxidative process in PD. Similarly, in the study of Kirbas et al. oxidative stress index (OSI) and TOS levels of Parkinson patients were higher than those of the control group (6).

Nesfatin-1 and GLP-1 have been reported as molecules with neuroprotective property that relieve oxidative stress. Nesfatin-1 is an 82 amino acid peptide, first identified in rat hypothalamus. Experimental studies on nesfatin-1 identified its antiinflammatory, antiapoptotic and antioxidative property (7-9). In our study, Parkinson patients were found to have lower nesfatin-1, but higher TOS levels than those of the control group. These findings suggest that oxidative process might be enhanced by the decrease in nesfatin-1, which is a well-known antioxidant.

GLP-1 peptide expressed by enteroendocrine cells located in the small intestine acts in regulation of blood glucose and is also recognized as a growth factor (10). In neurotoxin-producing PD models, GLP-1 receptor stimulation has been reported to exert benefits in dopaminergic cell survival and functionality, and in the resolution of abnormal behaviour in addition to its relieving function in oxidative stress which plays a role in neurodegenerative disorders (11,12). In our study, GLP-1 levels of Parkinson patients found to be lower than those of the control group. This finding suggest a possible protective role of GLP-1 in dopaminergic neurons of Parkinson patients.

CONCLUSION
Our study revealed lower nesfatin-1 and GLP-1 levels, in Parkinson patients compared to those of control group. TAS values, on the other hand, were similar. Our findings suggest that the neuroprotective effects of nesfatin-1 and GLP-1 molecules might be related to the oxidative processes. In this study, we have investigated nesfatin-1 and GLP-1 levels. We believe our study will contribute to further studies on above-named molecules to investigate their use as treatment options and the likelihood that they may prevent or slow down the disease progression.

REFERENCES

Table 1. Mean nesfatin-1, GLP-1, TAS, and TOS values of the Parkinson patients in comparison to those of the control group.

<table>
<thead>
<tr>
<th></th>
<th>Parkinson patients</th>
<th>Control group</th>
<th>p-value</th>
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</thead>
<tbody>
<tr>
<td>Nesfatin-1 (pg/mL)</td>
<td>27.29 ± 11.07</td>
<td>100.49 ± 24.07</td>
<td>&lt; 0.001</td>
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<tr>
<td>GLP-1 (ng/mL)</td>
<td>0.72 ± 0.32</td>
<td>1.76 ± 0.70</td>
<td>&lt; 0.001</td>
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<tr>
<td>TAS (mmol Trolox equiv./L)</td>
<td>1.15 ± 0.27</td>
<td>1.20 ± 0.30</td>
<td>0.51</td>
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<tr>
<td>TOS (mmol H2O2 equiv./L)</td>
<td>9.05 ± 1.43</td>
<td>5.86 ± 1.13</td>
<td>&lt; 0.001</td>
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