

Prognostic Value of ^{18}F -FDG PET in Monosegmental Stenosis and Myelopathy of the Cervical Spinal Cord

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MRI offers perfect visualization of spondylotic stenosis of the cervical spine, but morphologic imaging does not correlate with clinical symptoms and postoperative recovery after decompression surgery. In this prospective study, we investigated the role of ^{18}F -FDG PET in patients with degenerative stenosis of the cervical spinal cord in relation to postsurgical outcome. **Methods:** Twenty patients with monosegmental spondylotic stenosis of the middle cervical spine (C3/C4 or C4/C5) showing intramedullary hyperintensity on T2-weighted MRI and clinical symptoms of myelopathy (myelopathic patients) were investigated by ^{18}F -FDG PET. Maximum standardized uptake values (SUV_{max}) were measured at all levels of the cervical spine (C1–C7). Decompression surgery and anterior cervical fusion were performed on all patients, and clinical status (Japanese Orthopedic Association [JOA] score) was assessed before and 6 mo after surgery. The ^{18}F -FDG data of 10 individuals without cervical spine pathology were used as a reference (controls). **Results:** The myelopathic patients showed a significant decrease in ^{18}F -FDG uptake in the area of the lower cervical cord, compared with the control group (C7 SUV_{max} , 1.49 ± 0.18 vs. 1.71 ± 0.27 , $P = 0.01$). Ten myelopathic patients exhibited focally increased ^{18}F -FDG uptake at the level of the stenosis (SUV_{max} , 2.27 ± 0.41 vs. 1.75 ± 0.22 , $P = 0.002$). The remaining 10 patients showed inconspicuous ^{18}F -FDG uptake at the area of the stenosis. Postoperatively, the patients with focally increased ^{18}F -FDG accumulation at the level of stenosis showed good clinical recovery and a significant improvement in JOA scores (13.6 ± 2.3 vs. 9.5 ± 2.5 , $P = 0.001$), whereas no significant improvement was observed in the remaining patients (JOA score, 12.0 ± 2.4 vs. 11.6 ± 2.5 , not statistically significant). Multiple regression analysis identified the presence of focally increased ^{18}F -FDG uptake at the level of the stenosis as an independent predictor of postoperative outcome ($P = 0.002$). **Conclusion:** The results suggest that regional changes in ^{18}F -FDG uptake have prognostic significance in compression-induced cervical myelopathy that may be helpful in decisions on the timing of surgery.

Key Words: positron emission tomography; [^{18}F] fluorodeoxyglucose; [^{18}F]FDG PET; cervical myelopathy; degenerative spinal stenosis; spinal cord recovery

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Degenerative changes that include dislocated disk material, thickening of the intraspinal yellow ligaments, and slowly growing osteophytes of the vertebrae can lead to ongoing obstruction of the spinal canal (stenosis), with subsequent mechanic compression and dysfunction of the cervical spinal cord (myelopathy). The development of degenerative cervical myelopathy is usually slowly progressive and can finally lead to severe disturbances of all neurologic functions below the level of stenosis (1,2). Compression of the vulnerable spinal cord at the level of stenosis leads to a common pattern of structural tissue damage: Atrophy and neuronal loss in the gray matter and demyelination in the white matter have been demonstrated in biomechanical and autopsy studies (3,4).

Conventional MRI offers perfect visualization of the stenosis, with high spatial resolution, but neither the absolute diameter of the stenosis nor cord compression including the pathognomonic myelopathy sign (intramedullary hyperintensity on T2-weighted MRI scans) correlates with preoperative clinical activity or postoperative recovery after decompression (5–8).

Because morphologic imaging turned out to be of limited value for prediction of clinical activity and outcome in cervical myelopathy, PET using ^{18}F -FDG has been proposed for better functional and metabolic assessment of the damaged spinal cord tissue. The few ^{18}F -FDG PET studies on myelopathy patients reported either a global reduction or a global increase of ^{18}F -FDG uptake across the entire spinal cord independent of the level of stenosis (9–12). This finding is surprising, because myelopathy is morphologically and clinically a local phenomenon: the neurologic impairment such as sensory and motor deficits is strictly limited to the segments at and below the level of stenosis. Therefore, a local metabolic reaction of the damaged cervical cord at and below the level of stenosis should

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be expected instead of global changes in ^{18}F -FDG uptake in the entire cervical myelon.

In a preliminary evaluation of this ongoing study, we reported on ^{18}F -FDG uptake in a strictly selected group of patients with chronic compressive myelopathy and 10 control patients without known cervical abnormalities (13). In that report, the controls showed homogeneous ^{18}F -FDG uptake along the entire cervical cord whereas the patients with chronic compressive myelopathy had a significant decrease in ^{18}F -FDG uptake below their individual level of cord compression. In the current report, we present further results of the study, with the inclusion of patients with both chronic symptoms and acute symptoms. Furthermore, the postoperative clinical course after decompression of the stenosis, with special attention to the pattern of ^{18}F -FDG uptake in the cervical spine before surgery, was investigated.

MATERIALS AND METHODS

Patient Population

Within 2.1 y (June 2008–July 2010), 512 patients with degenerative cervical spine pathology underwent surgery at St. Vinzenz Hospital in Düsseldorf. MRI (1.5-T Sonata; Siemens) of the cervical spine with acquisition of T1- and T2-weighted images in axial and sagittal planes was performed on all patients for exact visualization of the level of cord compression and detection of intramedullary hyperintensity on T2-weighted scans as the radiologic sign of myelopathy. The functional status of the patients was assessed by the Japanese Orthopedic Association (JOA) scoring system, which reflects the clinical activity of the cervical myelopathy and has a maximum value of 17 in healthy individuals (14). The clinical dynamics were assessed by the change in JOA score during the last 3 mo before study entry as a measure of deterioration of cervical myelopathy (stable or progressive). The postoperative course and outcome after decompression surgery were assessed by the difference between the preoperative and 6-mo postoperative JOA score as a measure of recovery of cervical spinal cord function.

Of these patients, a group was selected according to criteria for pathology, symptoms, and vertebral level. Regarding pathology, typical features of degenerative spondylotic stenosis with local cord compression and the radiologic sign of myelopathy with intramedullary hyperintensity on T2-weighted MRI scans had to be present. Patients with other pathologies, such as tumor, trauma, ossification of the posterior longitudinal ligament, soft disk herniation, or infectious disease of the spine or without radiologic signs of myelopathy were excluded. Regarding symptoms, typical signs and symptoms of myelopathy with clinical impairment (JOA score, <16) had to be present at the time of study inclusion. Patients without or with only mild clinical activity of myelopathy (JOA score, 16–17) were excluded because evaluation of clinical recovery in these patients is not possible. Regarding vertebral level, monosegmental stenosis and local cord compression in the middle of the cervical spine (vertebral level C3/C4 or C4/C5) had to be present. Patients with bi- or multisegmental stenosis were excluded because of their diffuse pathology.

The limitation of the myelopathy location to the middle of the cervical spine reduced the potential number of study patients considerably because degenerative cervical stenosis typically affects the lower cervical spine at level C5/C6 or C6/C7. This study design, however, offers several advantages. First, this approach allows

the evaluation of a well-visible portion of the cervical myelon above and below the stenosis. In patients with upper cervical spine myelopathy (C1/C2), the myelon above the stenosis is influenced by high ^{18}F -FDG-uptake in the lower brain stem. In patients with stenosis of the lower cervical spine (C5/C6 and C6/C7), the evaluation of ^{18}F -FDG uptake in the thoracic spine is technically difficult.

Second, neurologic deficits in the arms and hands can be caused by myelopathy or radiculopathy due to stenosis of the neuroforamina with compression of the nerve roots, and the distinction can be difficult. Stenosis at the lower cervical spine (C5/C6 and C6/C7) often leads to both myelopathic and radiculopathic symptoms of the upper limbs, whereas stenosis at the middle cervical spine (C3/C4 and C4/C5) leads predominantly to myelopathic deficits.

During the study period, 20 of 512 patients (3.9%) with degenerative cervical spine pathology met the inclusion criteria and were admitted for the ^{18}F -FDG PET study. Most potential study patients had to be excluded because they had soft disk herniation, stenosis of the lower cervical spine at vertebral level C5/C6 and C6/C7, multisegmental stenosis, absence of radiologic myelopathy signs, or purely radicular symptoms. Furthermore, patients with diabetes mellitus and peripheral polyneuropathy were excluded.

The actual patient group of 20 myelopathic patients consisted of 7 patients who were already included in our earlier study (patients 21–27) and 13 newly recruited patients (patients 11–20 and 28–30) (supplemental Table 1; supplemental materials are available online only at <http://jnm.snmjournals.org>). Three patients from the earlier study were excluded—2 because they showed C1/C2 myelopathy and 1 because postoperative follow-up was not available.

All 20 study patients gave written informed consent to participate in the study. There were 3 women and 17 men; mean age was 64.9 ± 12.6 y (range, 45–83 y) and mean duration of symptoms was 10.0 ± 8.6 mo (range, 1–30 mo), with an average preoperative JOA score of 10.5 ± 2.6 (range, 6–15). Twelve patients had stenosis and myelopathy at vertebral level C3/C4, and 8 patients at level C4/C5.

As a control group, we used 10 patients who were admitted for whole-body ^{18}F -FDG PET at the state of initial diagnosis for various peripheral tumors between October 2008 and November 2010. The group included 1 woman and 9 men; mean age was 61 ± 11 y (range, 41–75 y). None of the control patients had neck or arm pain or symptoms of myelopathy (JOA score, 17). Four patients had cancer of unknown primary site, 3 had gastrointestinal tumors, 1 had a testicular tumor, 1 had non-Hodgkin lymphoma, and 1 had small cell lung cancer. ^{18}F -FDG PET of the neck showed no abnormalities in these patients. Detailed information on the biologic and clinical data of the myelopathic and control patients is given in supplemental Table 1.

^{18}F -FDG PET

The technique for the PET scans was identical in myelopathic patients and controls. All subjects fasted at least 12 h before the PET study. The scans were performed 1 h after intravenous injection of 370 MBq of ^{18}F -FDG. In the myelopathic patients, the scans were limited to the neck region, whereas the 10 tumor patients of the control group had whole-body scans that included the neck region. Blood glucose levels were checked in all subjects before ^{18}F -FDG injection to ensure normoglycemia. All were euglycemic, and no one received insulin to reduce blood glucose to normal levels.

The measurements were performed on an ECAT EXACT HR+ scanner in 3-dimensional mode (Siemens; 32 rings; axial field of view, 15.5 cm).

For attenuation correction, transmission scans with three $^{68}\text{Ge}/^{68}\text{Ga}$ rotating line sources were used. After correction for random and scattered coincidences and dead time, image data were obtained by iterative reconstruction; the reconstructed image resolution was about 5.5 mm.

Data Analysis

The ^{18}F -FDG data were processed using dedicated software (MPI tool, version 3.28; ATV) and were visualized in axial, coronal, and sagittal sections. The data were coregistered with the corresponding MRI scans in patients with cervical myelopathy. To guarantee comparability of the data with those of previous publications, the regional evaluation of ^{18}F -FDG uptake in the spinal cord was done in the same way as described previously (9–13). In short, circular regions of interest with a diameter of 10 mm were placed onto the spinal cord in transaxial slices using sagittal images as an online reference. The maximum uptake value in the region of interest was used as representative for tissue radioactivity to eliminate the partial-volume effect caused by the slightly larger size of the region of interest than of the diameter of the spinal cord. ^{18}F -FDG uptake was measured in 21 transaxial slices covering the cervical spinal cord from C1 to C7 (3 slices for each vertebral level). The 3 values for every vertebral level were averaged to 1 uptake value for each vertebral level from C1 to C7. The maximum standardized uptake value (SUV_{max}) of ^{18}F -FDG was calculated by dividing the maximum radioactivity (kBq/mL) of the region of interest by the radioactivity injected per gram of body weight (13). For the statistical analysis, the data of vertebral level C1 were discarded because there was spillover from the lower brainstem in most patients.

Statistical Analysis

All values are expressed as mean \pm SD. Differences between groups were tested by the Student *t* test for independent samples, the Wilcoxon rank sum test, or 1-way ANOVA. In patients with focally increased ^{18}F -FDG uptake at the level of the stenosis, the individual peak values were compared with the average value of C3–C5 in healthy controls.

The individual course of ^{18}F -FDG uptake in the cervical spine was estimated by a regression analysis of SUV_{max} from C2 to C7, and the differences of the regression coefficients between the groups were tested by the Student *t* test for independent samples.

Changes in clinical parameters within the subgroups of myelopathic patients after surgery were tested using the Student paired *t* test. To determine the influence of the age, duration of symptoms, deterioration of JOA score, and presence of focally increased ^{18}F -FDG uptake at the level of the stenosis on clinical outcome, a correlation analysis was performed using the Pearson product moment and Spearman rank correlation coefficient. The independent influence of the different variables on clinical outcome was tested by multiple regression analysis. The computations were done using Sigma plot software (version 11.0; Systat Software Inc.). Probability values of less than 0.05 were considered significant. No correction for multiple statistical comparisons was done.

RESULTS

The patients with cervical myelopathy showed a significant decrease in ^{18}F -FDG uptake in the lower cervical cord,

compared with the control group (C7 SUV_{max} , 1.49 ± 0.18 vs. 1.71 ± 0.27 , $P = 0.01$). Further visual analysis of the PET scans revealed 2 markedly different patterns of uptake in the 20 myelopathic patients: a pattern of increased uptake at the level of spinal stenosis ($n = 10$) and a pattern of inconspicuous uptake at the level of stenosis ($n = 10$). For further evaluation, patients with an uptake peak at the level of stenosis were classified as having myelopathy type 1, and those with inconspicuous uptake at the level of stenosis were classified as having myelopathy type 2. Control patients without cervical abnormalities showed an almost homogeneous glucose utilization along the entire cervical cord. There was no significant difference in the mean SUV_{max} of the cervical spine from C1 to C7 (control SUV_{max} , 1.79 ± 0.26 ; myelopathy type 1 SUV_{max} , 1.93 ± 0.30 ; myelopathy type 2 SUV_{max} , 1.78 ± 0.15) and no significant difference in age between the different groups (controls, 61 ± 11 y; myelopathy type 1, 71 ± 8 y; myelopathy type 2, 59 ± 14 y). Correlation analysis revealed no significant correlation between age and mean SUV_{max} in the cervical spine or between age and decrease in ^{18}F -FDG uptake from C2 to C7 in either the myelopathic or control subjects. The data on SUV_{max} at the different levels of the cervical spine for myelopathic patients and controls are given in detail in supplemental Table 1.

Myelopathy Type 1

In patients classified as having myelopathy type 1, increased ^{18}F -FDG uptake was noted at the individual level of stenosis and cord compression. SUV_{max} was significantly higher at the level of stenosis than at the C3–C5 level in controls (SUV_{max} , 2.27 ± 0.41 vs. 1.75 ± 0.22 , $P = 0.002$). An example is shown in Figure 1. Figure 2 illustrates ^{18}F -FDG uptake along the cervical spinal cord of all patients with myelopathy type 1, compared with controls, and demonstrates the significant increase of ^{18}F -FDG uptake at the level of cord compression in the entire group. Furthermore, ^{18}F -FDG uptake in the lower cervical spine was lower in myelopathic patients than in controls (SUV_{max} at C7, 1.51 ± 0.18 vs. 1.71 ± 0.27 , $P = 0.05$). This finding is supported by the regression analysis of SUV_{max} in the spine from C2 to C7, showing a negative slope in patients with myelopathy type 1 that was significantly different from that of controls (-0.70 ± 0.20 vs. 0.25 ± 0.47 , $P = 0.001$).

Myelopathy Type 2

In patients classified as having myelopathy type 2, inconspicuous ^{18}F -FDG uptake was noted at the individual level of stenosis and cord compression. An example is shown in Figure 3. Figure 4 illustrates ^{18}F -FDG uptake along the cervical spinal cord of patients with myelopathy type 2, compared with controls. Similar to patients with myelopathy type 1, patients with myelopathy type 2 had less ^{18}F -FDG uptake in the lower cervical spine than did controls (SUV_{max} at C7, 1.47 ± 0.19 vs. 1.71 ± 0.27 , $P = 0.03$). Again, regression analysis of SUV_{max} in the spine from C2 to C7

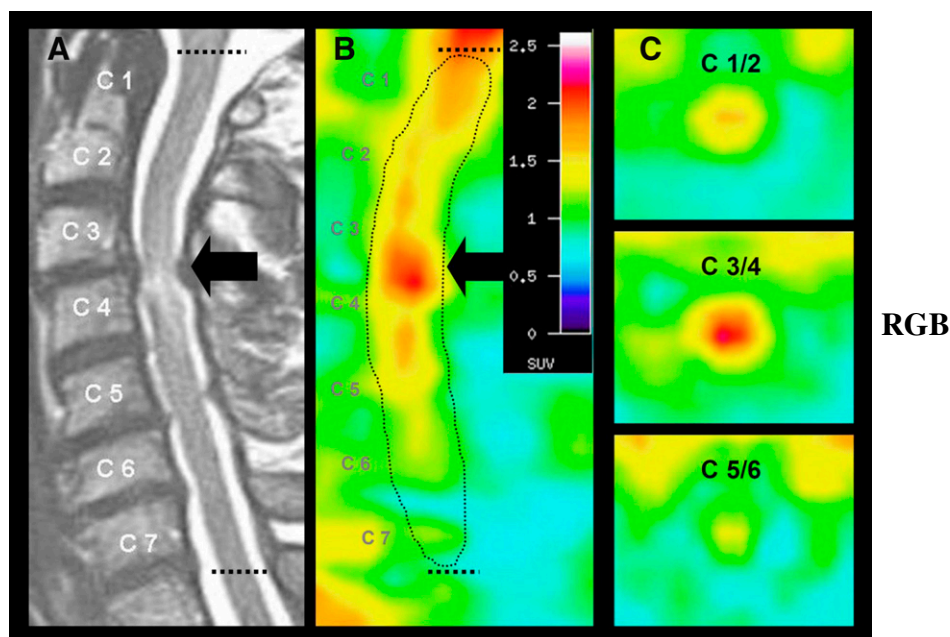


FIGURE 1. Patient 13, with myelopathy type 1. (A) Sagittal T2-weighted MRI scan shows stenosis with compression of cervical cord and intramedullary hyperintensity at level C3/C4 (arrow). (B and C) Corresponding sagittal (B) and transaxial (C) ^{18}F -FDG PET slices demonstrate focally increased uptake at level of stenosis. Patient had good clinical recovery 6 mo after decompression of stenosis.

confirmed this finding and showed a negative slope that was significantly different from that of controls (-0.76 ± 0.23 vs. 0.25 ± 0.47 , $P = 0.001$).

Clinical Status and Postoperative Outcome

Symptoms were of significantly shorter duration in patients with myelopathy type 1 than in patients with myelopathy type 2 (5.5 ± 4.7 vs. 14.5 ± 9.5 mo, $P = 0.014$). Moreover, the deterioration of the JOA score during

the last 3 mo was significantly higher in type 1 than in type 2 (4.1 ± 2.5 vs. 0.7 ± 0.5 , $P < 0.001$). These findings indicate that type 1 represents myelopathy in its acute and often rapidly progressive phase whereas type 2 represents the chronic and stable phase.

There were no obvious differences in intraoperative findings between patients with type 1 and patients with type 2. Concerning postoperative outcome, we observed differences in recovery of clinical symptoms 6 mo after decompression surgery and anterior cervical fusion of the stenotic level. Type 1 myelopathic patients showed a significant improvement in JOA score (13.6 ± 2.3 vs. 9.5 ± 2.5 , $P < 0.001$), whereas there was no significant change in type 2 myelopathic patients (12.0 ± 2.4 vs. 11.6 ± 2.5 , not statistically significant) (Fig. 5). A significant correlation was found between postoperative outcome and duration of symptoms ($r = -0.44$, $P = 0.05$), preoperative JOA score ($r = -0.52$, $P = 0.02$), preoperative deterioration ($r = 0.75$, $P < 0.001$), and the presence of a peak on ^{18}F -FDG PET ($r = 0.85$, $P < 0.00001$). Multiple regression analysis identified the following parameters as independent predictors of postoperative outcome: age ($P = 0.01$), preoperative JOA score ($P = 0.04$), and the presence of a peak on ^{18}F -FDG PET ($P = 0.002$).

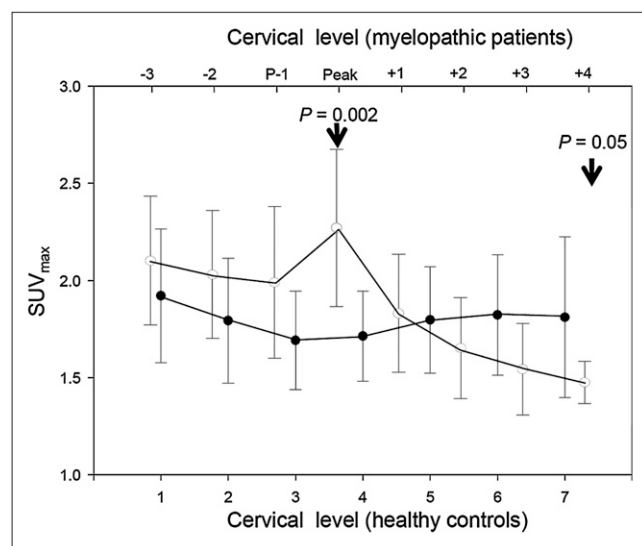


FIGURE 2. Comparison of SUV_{max} of ^{18}F -FDG uptake along cervical spine (C1–C7) in patients with myelopathy type 1 (○) and patients without abnormalities of cervical spine (●). Curve in patients with myelopathy is adjusted to peak ^{18}F -FDG uptake (upper scale). ^{18}F -FDG uptake at individual level of stenosis (C3–C5) is significantly increased, compared with control group. Furthermore, ^{18}F -FDG uptake in myelopathic patients is decreased in lower cervical spine (C7).

DISCUSSION

^{18}F -FDG PET has been used for more than 25 y in lesions of the spinal cord to differentiate between malignant neoplastic diseases (15–22) and benign inflammatory lesions (23–26). Myelopathy caused by degenerative compression, however, is a much more frequent clinical problem than inflammatory or neoplastic disorders of the cervical spine. Because conventional imaging with CT and MRI does not reliably correlate with functional defi-

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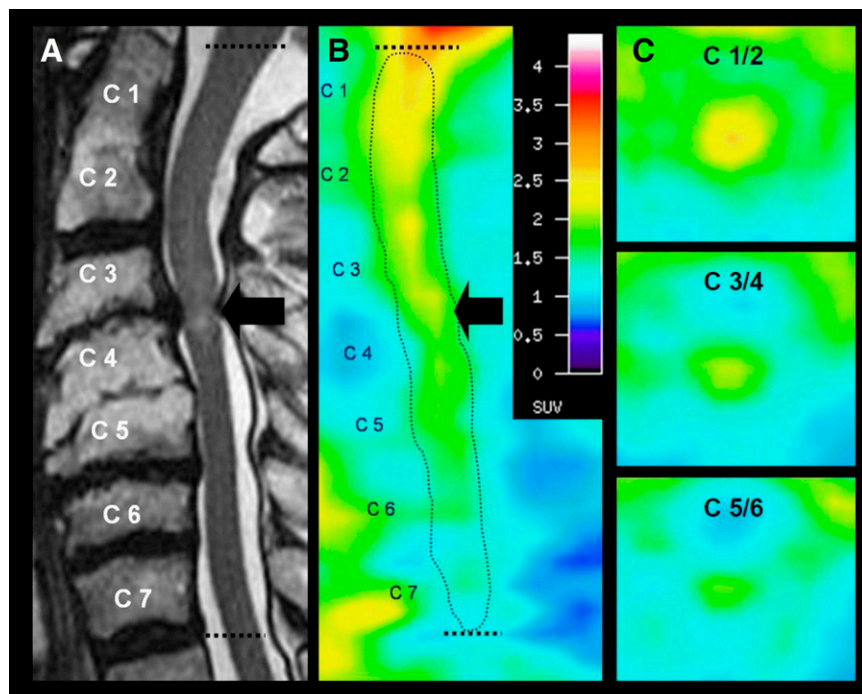


FIGURE 3. Patient 28, with myelopathy type 2. (A) Sagittal T2-weighted MRI scan shows stenosis with compression of cervical cord and intramedullary hyperintensity at level C3/C4 (arrow). (B and C) Corresponding sagittal (B) and transaxial (C) ^{18}F -FDG PET slices demonstrate inconspicuous ^{18}F -FDG uptake at level of stenosis. This patient showed no clinical improvement 6 mo after decompression of stenosis.

cits or the clinical course of myelopathy (5–8), there is a need to include alternative diagnostic methods such as PET for better evaluation of functional impairment of the neuronal tissue and prognostication of surgical outcome.

Anatomic visualization and glucose utilization patterns within the healthy, noncompressed cervical spinal cord of adults is well documented (12,22,27), and normal ^{18}F -FDG uptake along the unaffected cervical myelon is relatively constant. The reported SUV_{max} varies between 1.7 ± 0.6

(22), 1.84 ± 0.23 (13), 1.95 ± 0.30 (12), and 2.12 ± 0.44 , which are in the same range as values reported for controls in the present study.

So far, only a few studies have investigated ^{18}F -FDG uptake in the cervical spine of patients with compressive myelopathy. In 1 study, 2 of 7 patients showed increased and 5 patients decreased uptake in the entire cervical spine, and there was an increase in glucose utilization after surgery in all but 1 patient (11). Another study, including 23 patients, reported increased SUV_{max} in the cervical spinal cord (2.14 ± 0.41) and a significant correlation with the pre- and postoperative JOA score (10). The latest study ($n = 24$) reported a decrease of the elevated SUV_{max} in the cervical cord after decompressive surgery (9). Thus, the results of previous studies give a mixed picture, and none of the studies observed a local change in ^{18}F -FDG uptake in relation to the individual level of cervical stenosis. It is possible that these inconsistent findings are caused by the inclusion of patients with a variety of mono- or multisegmental lesions, as well as hard and soft disk pathologies, and of patients with or without signs of myelopathy on MRI.

Therefore, in this prospective study, strict inclusion criteria were met and only patients with monosegmental compressive hard disk stenosis in the middle cervical spine and typical signs of myelopathy on MRI were included. A preliminary analysis of 10 patients with chronic myelopathy indicated that the disturbance of glucose utilization in the cervical myelon is not a global but a local phenomenon: we observed a progressive decrease in ^{18}F -FDG uptake below the individual level of stenosis (13).

In the present study, data on 20 patients with monosegmental stenosis in the middle cervical spine were evaluated,

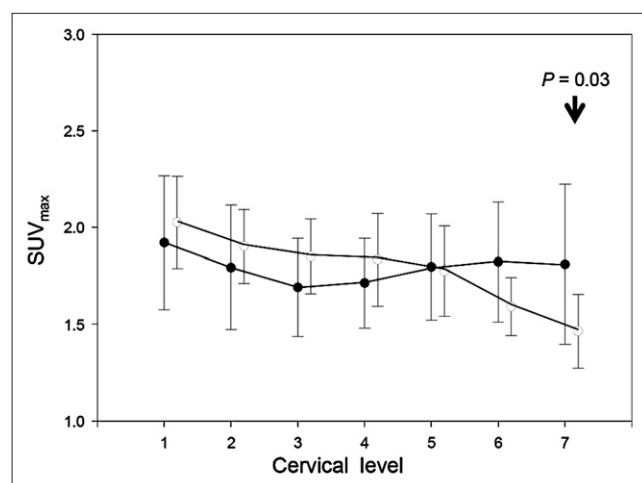
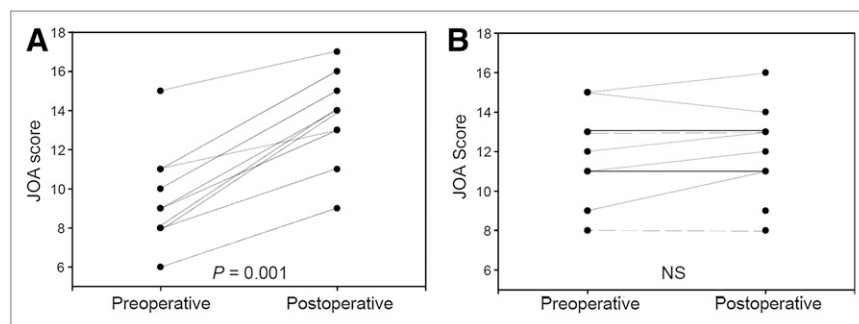


FIGURE 4. Comparison of SUV_{max} of ^{18}F -FDG uptake along cervical spine (C1–C7) in patients with myelopathy type 2 (○) and patients without abnormalities of cervical spine (●). ^{18}F -FDG uptake at level of stenosis (C3–C5) in type 2 myelopathic patients is similar to that in control patients. Significantly decreased ^{18}F -FDG uptake, however, is present in myelopathic patients in lower cervical spine (C7).

FIGURE 5. Comparison of clinical status (JOA score) before and after decompressive surgery of cervical spine: results of patients with peak ^{18}F -FDG uptake at level of stenosis (myelopathy type 1) (A) and patients with inconspicuous ^{18}F -FDG uptake at level of stenosis (myelopathy type 2) (B). Patients with myelopathy type 1 showed significant improvement of clinical status after surgery, whereas patients with myelopathy type 2 showed no improvement.



including patients with acute myelopathy, chronic myelopathy with stable disease, or chronic myelopathy with recent progression of symptoms. A major finding was the 2 different patterns of ^{18}F -FDG uptake: patients with increased ^{18}F -FDG uptake at the level of spinal stenosis were assigned to myelopathy type 1, and patients with inconspicuous ^{18}F -FDG uptake at the level of spinal stenosis were assigned to myelopathy type 2. Patients with type 1 had a significantly shorter duration of symptoms and a significantly greater clinical deterioration during the last 3 mo than did patients with type 2. These findings indicate that ^{18}F -FDG PET is helpful to identify patients with myelopathy in the acute and rapid progressive stage and patients with myelopathy in the chronic stage.

Another important finding of this study is that patients with myelopathy type 1 showed a significant improvement in clinical symptoms after surgery whereas the JOA score of patients with myelopathy type 2 remained unchanged. Thus, increased ^{18}F -FDG uptake at the level of spinal stenosis is associated with good clinical outcome after decompression surgery. Moreover, multivariate regression analysis identified the presence of an ^{18}F -FDG peak in the cervical spine as an independent factor to predict the success of surgical intervention.

On the basis of these results, it is tempting to speculate that the different patterns of ^{18}F -FDG uptake at the site of the compressed cervical myelon reflect different stages of the pathophysiologic course of myelopathy. Animal experiments have shown that early stages of cord compression are associated with increased immunoreactivity of brain-derived neurotrophic factor and neurotrophin-3 in neurons and astroglial cells, as may explain the increased glucose metabolism (28). At that stage, the process is still reversible, as is supported by the good clinical outcome after surgical intervention. Chronic compression, however, leads to atrophy and necrosis of anterior gray horn cells and the loss of glucose-consuming neurons, resulting in decreased ^{18}F -FDG uptake in the spinal cord (29,30).

Similarly, experiences in patients with radiation myelopathy support this hypothesis. After initially increased ^{18}F -FDG uptake ($\text{SUV}_{\text{max}} \leq 2.7$) within the area of demyelination, a slow normalization of glucose utilization can be observed. The decline of the increased glucose metabolism is accompanied by a good clinical recovery under treatment

with glucocorticoids (31–34). These authors suggest that the regionally increased ^{18}F -FDG uptake is caused by reparative processes in radiation-induced damage to the spinal cord. In line with our observations, an increased glucose metabolism in radiogenic myelopathy seems to be associated with good recovery of the patient during the further course of the myelopathy.

Clinical parameters such as rapid clinical deterioration or short duration of disease are important factors in predicting surgical outcome and may be sufficient for deciding on the timing of surgery. However, the clinical situation is unclear in many patients, and a meaningful method of imaging would be of great value for decision making. Patients with acute disease can elect immediate surgery to try to improve clinical symptoms, whereas in chronic disease an improvement is not to be expected. In those cases, surgery may be delayed to prevent further progression of disease.

The MRI examinations in our study were performed without application of a contrast medium, as it is not necessary for this indication and therefore unusual. Contrast enhancement in the area of cervical myelopathy is observed relatively rarely. In a recent prospective multicenter trial on 683 patients with cervical myelopathy, contrast enhancement was observed in 7.3% of the patients (35). The intramedullary contrast enhancement did not correlate with the severity of preoperative clinical symptoms but was, however, a sign of a poor prognosis. As in our study, a focal accumulation of ^{18}F -FDG was associated with a better prognosis; the 2 diagnostic phenomena seem to have different causes.

A limitation of our study is the small number of patients and the relatively short time of postoperative follow-up. Therefore, confirmation of the results in more patients and with a longer follow-up is required. Nevertheless, in our opinion the results are still impressive and could open a new clinical application for ^{18}F -FDG PET.

CONCLUSION

^{18}F -FDG PET of the cervical spinal cord in patients with spondylotic myelopathy exhibits local disturbances of glucose utilization depending on the level of mechanic compression. Locally increased ^{18}F -FDG uptake at the level of the stenosis is associated with acute or recently progressive symptoms. The short-term outcome of these type 1 myelopathic patients after decompression surgery is favorable.

Patients with inconspicuous ^{18}F -FDG uptake at the level of the stenosis usually have chronic symptoms without recent progression. The recovery 6 mo after decompression in this type 2 myelopathy is poor.

DISCLOSURE STATEMENT

The costs of publication of this article were defrayed in part by the payment of page charges. Therefore, and solely to indicate this fact, this article is hereby marked “advertisement” in accordance with 18 USC section 1734.

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