Relation between pelvic floor neurophysiological abnormalities and erectile dysfunction in patients with obstructed defecation

Mervat Sheta Elsawy and Emmanuel Kamal Aziz Saba*

Abstract

**Background:** Obstructed defecation is a common pelvic floor medical problem among adult population. Pelvic floor disorders were reported to be associated with sexual dysfunction including erectile dysfunction among male patients. The aim was to determine the relation between pelvic floor neurophysiological abnormalities and erectile dysfunction in male patients with obstructed defecation.

**Methods:** This cross-sectional study included 65 married male patients with obstructed defecation and a control group consisted of 15 apparently healthy married males. Assessment of obstructed defecation severity was done by using modified obstructed defecation score, time of toileting and Patient Assessment of Constipation-Quality of Life questionnaire. Assessment of erectile functions was done using erectile function domain of International Index of Erectile Function questionnaire and Erectile Dysfunction-Effect on Quality of Life Questionnaire. Anal manometry and dynamic pelvis magnetic resonance imaging were done. Electrophysiological studies included pudendal nerve motor conduction study and needle electromyography of external anal sphincter, puborectalis and bulbocavernosus muscles.

**Results:** There were 32 patients (49.2%) who had erectile dysfunction. The maximum straining anal pressure was significantly higher among patients with erectile dysfunction. Pudendal nerve terminal motor latency was significantly delayed and the percentage of bilateral pudendal neuropathy was significantly higher among patients with erectile dysfunction. The percentage of electromyography evidence of denervation with chronic reinnervation in the external anal sphincter and bulbocavernosus muscles were significantly higher among patients with erectile dysfunction. Regression analysis detected three co-variables to be associated with significantly increasing the likelihood of development of erectile dysfunction. These were maximum straining anal pressure (odd ratio = 1.122), right pudendal nerve terminal motor latency (odd ratio = 3.755) and left pudendal nerve terminal motor latency (odd ratio = 3.770).

**Conclusions:** Erectile dysfunction is prevalent among patients with obstructed defecation. It is associated with characteristic pelvic floor electrophysiological abnormalities. Pelvic floor neurophysiological changes vary from minimal to severe neuromuscular abnormalities that usually accompanying erectile dysfunction. Pudendal neuropathy and increased maximum straining anal pressure are essential risk factors for increasing the likelihood of development of erectile dysfunction in patients with obstructed defecation.
1 Background

Obstructed defecation (OD) is a prevalent problem. Its prevalence is about 7% of the adult population [1, 2]. It has two different pathophysiological mechanisms. Functional OD is anismus which is due to a functional abnormality. Mechanical OD is due to anatomical lesions as rectocele and rectal intussusception [3, 4]. OD affects and impairs health-related quality of life to a great extent [5, 6].

Sexual health is an essential part in the health-related quality of life [7–10]. Pelvic floor disorders including OD were reported to be associated with sexual dysfunction [11]. Male patients with OD may suffer from sexual dysfunctions and sexual life impairments. Sexual dysfunction as erectile dysfunction (ED) is present among patients suffering from OD, and questionnaires are useful in assessment of these patients [9, 12].

However, among OD patients, the evaluation of the anorectal function and the anatomical defects often receive more attention than the sexual function [11]. OD is usually associated with electrophysiological changes in the pelvic floor muscles and nerves. These include pudendal neuropathy and neuropathic abnormalities in the external anal sphincter (EAS) and puborectalis (PR) muscles, as well as other pelvic floor muscles as bulbocavernosus (BC) muscle. These changes vary in severity among different patients according to the severity of OD [13, 14]. These neurological changes could have a role in the impairment of the sexual functions among male patients with OD [15, 16]. ED could be as a result of neurological abnormalities in the pelvic floor nerves and muscles [7, 9, 12, 17].

To the best of the authors’ knowledge, there is no previous research that discussed the relationship between different pelvic floor electrophysiological changes and ED among patients with OD. The knowledge of this could help in the explanation of the improvement of the erectile sexual dysfunctions after pelvic floor rehabilitation with biofeedback therapy [18]. The aim of the work was to determine the relation between pelvic floor neurophysiological abnormalities and ED in male patients with OD.

2 Methods

Sixty-five married male patients with OD were included in this cross-sectional study. All of them were recruited randomly from those who attended the Pelvic Floor Rehabilitation clinic. It included a control group of 15 apparently healthy married male volunteers.

Figure 1 illustrates the inclusion criteria which is the clinical diagnosis of OD (Rome III criteria for OD) [3, 19]. The two different forms of OD were included in the study whether functional OD as anismus or mechanical OD due to anatomical lesions as rectocele, as well as the combination of both conditions together (mixed form of OD) [3, 4]. The patients were divided into two groups depending on the presence of ED. It is defined as the inability of achievement and maintenance of adequate penile erection which is sufficient to allow a satisfactory sexual performance. Patients were classified as having ED when they complained of ED and the score of erectile function domain of International Index of Erectile Function (IIEF) questionnaire was less than 26 [20]. Figure 1 illustrates the exclusion criteria of the study [6, 14, 21]. The researchers explained the work to the participants who gave an informed consent. Institutional Ethics Committee accepted the study.

All patients were assessed as the following: demographic parameters collection, history taking and measurements of body mass index (BMI) [22]. Assessment of OD severity was done by using modified OD score (MODS) and time of toileting [6, 23]. MODS represents the sum of individual scores for 7 symptoms and one score for life style alteration. Its total score ranges between zero (best) to 24 (severest form) [23]. The patient’s quality of life was graded using the Patient Assessment of Constipation-Quality of Life questionnaire (PAC-QoL) [23]. It assesses physical discomfort, psychological discomfort, worries and concerns, and dissatisfaction. PAC-QoL score consists of 28 questions scored from zero to four with a total score ranges between zero (best) to 112 (worst) [23].

Assessment of the erectile functions of the patient was done using erectile function domain of IIEF questionnaire (Fig. 2) [20]. Assessment of the effect of ED on the patient’s QoL was done using the Erectile Dysfunction-Effect on Quality of Life (ED-EQoL) questionnaire (Fig. 2) [10].

Clinical evaluation included perineal, anorectal and neurological examination [24]. Pelvic floor muscle strength was graded using Modified Oxford Scale (MOS) [25, 26]. The muscle strength was quantified from zero (absence of muscle contraction) to five (a completely normal muscle strength) [26].

Anal manometry assessment was performed using the manometric biofeedback device (Myomed 632-equipment, Enraf Nonius, The Netherlands). It included...
The clinical diagnosis of obstructed defecation was based on the presence of two or more symptoms in ≥25% of defecation attempts over a period of two years’ duration: (i) Straining. (ii) Lumpy or hard stools. (iii) Sensation of incomplete evacuation. (iv) Sensation of anorectal obstruction/blockage. (v) Manual maneuvers to promote defecation. This was supported by the presence of evidence of impaired evacuation on imaging and inappropriate contraction of the external anal sphincter and/or puborectalis muscles by imaging or electromyography and/or less than 20% relaxation of the basal resting anal pressure by anal manometry on straining.

(i) Systemic disorders as endocrine disorders and metabolic disorders. (ii) Slow transit syndrome. (iii) Inflammatory bowel disorders. (iv) Irritable bowel disorder. (v) Tumors in the anorectal region and/or genitourinary region. (vi) Previous abdominal and/or pelvic surgeries. (vii) Neurogenic bowel disorders. (viii) Neurological disorders as peripheral neuropathy and lumbosacral plexopathy. (ix) Erectile dysfunction due to vascular etiology, hormonal etiology, drug induced as due to antidepressants. (x) Congenital anomalies in the male genital organs. (xi) Patients not willing to participate in the study.

3 Results
There were 65 male patients with OD included in the study. Their mean age was 42.93 ± 10.33 years (range 22 to 65 years). The control group consisted of 15 apparently healthy males, and their mean age was 42.80 ± 13.83 years (range 21 to 63 years). No significant difference was found between both groups regarding age (Z = −0.056, P = 0.956).
The patients were divided according to the presence of ED into two groups depending on the results of patients’ complaint and erectile function domain of IIEF score. Patients with ED group consisted of 32 (49.2%) patients, and patients without ED group consisted of 33 (50.8%) patients. The clinical characteristics of the two patient groups and control group are tabulated in Table 1. There were no statistically significant differences between the two patient groups regarding the duration of OD symptoms, MOS, etiology and type of OD.

The results of anal manometric assessment are tabulated in Table 2. All patients had manometric evidence of failure of relaxation of the pelvic floor muscles during attempts at defecation. There were statistically significant differences between the two patient groups versus the control group regarding the maximum squeezing anal pressure and the maximum straining anal pressure (Table 2). The maximum straining anal pressure was statistically significantly higher among the OD with ED group in comparison to the OD without ED group (Table 2).

Results of electrophysiological assessment are tabulated in Table 3. The PNTML was significantly delayed in the OD with ED group in comparison to the control group, as well as OD without ED group (Table 3). The percentage of bilateral pudendal neuropathy was significantly higher among OD with ED group versus the other patient group (Table 3). The percentage of EMG evidence of denervation with chronic reinnervation (i.e., neuropathic abnormalities) in the EAS and BC muscles were significantly higher among OD with ED group (Table 3).

Correlation between erectile function domain of IIEF questionnaire and ED-EQoL questionnaire with different clinical, anal manometric and electrophysiological
### Table 1  Clinical characteristics of the two patient groups and control group

| Clinical characteristics | OD with ED group (n = 32 patients) mean ± SD | OD without ED group (n = 33 patients) mean ± SD | Control group (n = 15 subjects) mean ± SD | Test of significance | P |
|--------------------------|---------------------------------------------|-----------------------------------------------|-------------------------------------------|----------------------|---|
| **Age (years)**          | 40.96 ± 8.94                               | 44.84 ± 11.33                                | 42.80 ± 13.83                             | K = 2.076            | 0.354 |
| **Anthropometric measurements** |                                             |                                              |                                           |                      |    |
| Weight (kg)              | 72.43 ± 22.60                               | 77.18 ± 13.92                                | 79.10 ± 15.65                             | K = 2.491            | 0.288 |
| Height (cm)              | 162.37 ± 6.26                               | 162.12 ± 8.06                                | 164.00 ± 6.76                             | K = 0.476            | 0.788 |
| BMI (kg/m²)              | 27.36 ± 8.18                                | 29.39 ± 5.15                                 | 26.40 ± 6.60                              | K = 4.449            | 0.108 |
| **BMI category**         |                                             |                                              |                                           |                      |    |
| Underweight†             | 4 (12.5)                                    | 1 (3.0)                                      | 2 (13.3)                                  | X² = 14.506          | 0.069 |
| Normal weight†           | 11 (34.3)                                   | 5 (15.2)                                     | 5 (33.3)                                  |                       |    |
| Overweight†              | 7 (21.9)                                    | 12 (36.3)                                    | 4 (26.7)                                  |                       |    |
| Obesity†                 | 6 (18.8)                                    | 15 (45.5)                                    | 3 (20.0)                                  |                       |    |
| Morbid obesity†          | 4 (12.5)                                    | 0 (0.0)                                      | 1 (6.7)                                   |                       |    |
| **Duration of symptoms (years)** | 5.84 ± 4.23                   | 3.84 ± 1.85                                   | NA                                        | Z = −1.748           | 0.081 |
| MODS                     | 13.06 ± 5.16                                | 12.96 ± 5.03                                 | NA                                        | Z = −0.158           | 0.875 |
| **Time of toileting (minutes)** | 24.37 ± 14.71              | 20.75 ± 9.27                                 | NA                                        | Z = −0.756           | 0.450 |
| PAC-QoL                  | 42.34 ± 14.64                               | 42.39 ± 14.41                                | NA                                        | Z = −0.013           | 0.990 |
| **Erectile function domain of IIEF** | 18.71 ± 3.32                | 28.36 ± 1.47                                 | NA                                        | Z = −6.956           | ≤ 0.0001* |
| Erectile function domain of IIEF interpretation |                       |                                              |                                           |                      |    |
| No ED†                   | 0 (0)                                       | 33 (100)                                     | NA                                        | X² = 65.000          | ≤ 0.0001* |
| Mild ED†                 | 13 (40.6)                                   | 0 (0)                                        | NA                                        |                       |    |
| Mild to moderate ED†     | 11 (34.4)                                   | 0 (0)                                        | NA                                        |                       |    |
| Moderate ED†             | 0 (0)                                       | 0 (0)                                        | NA                                        |                       |    |
| **ED-EQoL**              | 44.25 ± 10.09                               | NA                                           | NA                                        | NA                   |    |
| **Clinical and MRI findings** |                       |                                              |                                           |                      |    |
| MOS‡                     | 4 (3–4)†‡                                   | 4 (3–5)†                                     | 5 (5–0)†                                   | K = 42.160           | ≤ 0.0001* |
| Rectocele†               | 14 (43.8)                                   | 13 (39.4)                                    | NA                                        | X² = 0.127           | 0.804 |
| Rectal intussusception†  | 7 (21.9)                                    | 4 (12.1)                                     | NA                                        | X² = 1.099           | 0.237 |
| Increased perineal descent† | 18 (56.3)                              | 14 (42.4)                                    | NA                                        | X² = 1.242           | 0.325 |
| Anismus†                 | 28 (87.5)                                   | 25 (75.8)                                    | NA                                        | X² = 1.488           | 0.339 |
| **Type of OD**           |                                             |                                              |                                           |                      |    |
| Functional OD†           | 13 (40.6)                                   | 18 (54.5)                                    | NA                                        | X² = 1.262           | 0.324 |
| Functional with mechanical OD† | 19 (59.4)                                | 15 (45.5)                                    | NA                                        |                       |    |
| Hypoesthesia on the sensory territory of pudendal nerve† | 22 (68.8)                              | 3 (9.1)                                      | NA                                        | X² = 24.430          | ≤ 0.0001* |

Kg, kilogram; cm, centimeter; BMI, body mass index; kg/m², kilogram per meter square; MODS, modified obstructed defecation score; PAC-QoL, Patient assessment of Constipation-Quality of life questionnaire; IIEF, International Index of Erectile Function questionnaire; ED-EQoL, Erectile Dysfunction-Effect on Quality of Life questionnaire; MRI, dynamic pelvis magnetic resonance imaging; MOS, Modified Oxford Scale; OD, obstructed defecation; ED, erectile dysfunction; n, number of subjects; SD, standard deviation; K, value of Kruskal-Wallis test; X², value of Chi-square test; Z, value of Mann Whitney test; NA, not applicable

*P is significant at < 0.05
† Data are reported as number (percentage)
‡ Data are reported as median (range)
§ Value of Fisher’s Exact test

| Statistical significance difference between OD with ED group versus control group (Z = −6.137, P ≤ 0.0001) |
|                                                                                                         |
| Statistical significance difference between OD without ED group versus control group (Z = −5.405, P ≤ 0.0001) |
parameters among OD with ED group of patients are tabulated in Table 4. There were statistical significant negative correlations between erectile function domain of IIEF questionnaire and time of toileting and PAC-QoL (Table 4). There was a statistically significant positive correlation between erectile function domain of IIEF questionnaire and maximum squeezing anal pressure (Table 4). There was a statistically significant negative correlation between ED-EQoL questionnaire and maximum squeezing anal pressure (Table 4).

Some co-variables were selected to be tested to assess their relation for the development of ED (Table 5). After adjusting the selected co-variables, the maximum straining anal pressure (OR = 1.122), right PNTML (OR = 3.755) and left PNTML (OR = 3.770) were associated with significantly increasing the likelihood of
Table 4 Correlation between erectile function domain of International Index of Erectile Function questionnaire and Erectile Dysfunction-Effect on Quality of Life questionnaire with different assessed parameters among patient group with erectile dysfunction (n = 32 patients)

| Different clinical, anal manometric and electrophysiological parameters | Erectile function domain of IIEF questionnaire | ED-EQoL questionnaire |
|---|---|---|
| | rs | P | rs | P |
| Duration of symptoms (years) | 0.039 | 0.833 | 0.058 | 0.753 |
| MODS | −0.344 | 0.054 | 0.103 | 0.576 |
| Time of toileting (minutes) | −0.377 | 0.033* | 0.138 | 0.452 |
| PAC-QoL | −0.398 | 0.024* | 0.275 | 0.127 |
| Anal manometric parameters | | | | |
| Maximum squeezing anal pressure (hPa) | 0.546 | 0.001* | −0.489 | 0.005* |
| Maximum straining anal pressure (hPa) | 0.076 | 0.678 | 0.119 | 0.516 |
| Pelvic floor electrophysiological parameters | | | | |
| Rt PNTML (ms) | −0.372 | 0.051 | 0.303 | 0.118 |
| Lt PNTML (ms) | −0.202 | 0.303 | 0.241 | 0.217 |

MODS, modified obstructed defecation score; PAC-QoL, Patient assessment of Constipation-Quality of life questionnaire; hPa, hectopascal (it is the unit of pressure and it is equal to 100 Pascals); Rt, right side; PNTML, pudendal nerve terminal motor latency; ms, millisecond; Lt, left side; IIEF, International Index of Erectile Function questionnaire; ED-EQoL, Erectile Dysfunction-Effect on Quality of Life questionnaire; rs, Spearman correlation coefficient

*P is significant at < 0.05

Table 5 The results of logistic regression analysis predicting the development of erectile dysfunction from some selected assessed parameters among obstructed defecation patients (n = 65 patients)

| Selected co-variables | β | Wald (X²) | Odd ratio | 95% CI | P |
|---|---|---|---|---|---|
| | | | Lower | Upper | |
| Age (years) | −0.043 | 1.128 | 0.958 | 0.884 | 1.037 |
| MODS | 0.017 | 0.026 | 1.017 | 0.827 | 1.252 |
| Time of toileting (minutes) | 0.123 | 2.697 | 1.131 | 0.976 | 1.311 |
| PAC-QoL | −0.146 | 3.770 | 0.864 | 0.746 | 1.001 |
| Maximum squeezing anal pressure (hPa) | 0.017 | 0.485 | 1.017 | 0.970 | 1.065 |
| Maximum straining anal pressure (hPa) | 0.115 | 5.917 | 1.122 | 1.023 | 1.231 |
| Rt PNTML (ms) | 1.323 | 4.459 | 3.755 | 1.100 | 12.822 |
| Lt PNTML (ms) | 1.327 | 3.964 | 3.770 | 1.021 | 13.926 |
| X²| 11.563 | | | | |
| P* | 0.172 | | | | |

MODS, modified obstructed defecation score; PAC-QoL, Patient assessment of Constipation-Quality of life questionnaire; hPa, hectopascal (it is the unit of pressure and it is equal to 100 Pascals); Rt, right side; PNTML, pudendal nerve terminal motor latency; ms, millisecond; Lt, left side; β, logistic regression standardized coefficients; Wald (X²), Wald Chi-square test tests the unique contribution of each co-variable in relation to all other examined co-variables, through eliminating any overlap effect between them (i.e. when examining a co-variable, it holds all other co-variable constant); CI, confidence interval

*P is significant at < 0.05

† Value of Chi-square Hosmer and Lemeshow test

4 Discussion

Erection is a physiological process. It depends on many vascular, hormonal, intra-corporal structure factors [7]. ED is the inability to attain and maintain an erection adequate and sufficient to allow satisfactory sexual performance [32]. It is a prevalent medical problem. It is present in about 52% of males between the 4th and 7th decade of life [33].

The pelvic floor muscles include the deep pelvic floor muscles and the superficial pelvic floor muscles. These superficial pelvic floor muscles include ischiocavernosus (IC) and BC muscles which have a role in erection [7]. The contraction of IC and BC muscles leads to penile erection by temporary increase in the corporeal body pressure [7]. Contraction of these two pairs of muscles performs compressive action necessary to increase intra-cavernous pressure. IC muscle contraction facilitates erection. BC muscle contraction slows the venous development of ED (Table 5). Goodness of fit using the Hosmer and Lemeshow test was 11.563 with P = 0.172.
drainage from the corpora cavernosa through producing pressure on the deep dorsal vein of the penis [34]. These affect penile rigidity. Also, rhythmic BC muscles contraction propels the semen down the urethra producing ejaculation [35, 36].

These muscles are supplied by the perineal branch of pudendal nerve. Pudendal nerve is a branch arise from the sacral plexus. It leaves the pelvis through the inferior part of the greater sciatic foramen. It passes posterior to the ischial spine to reenter the pelvis through the superior part of the lesser sciatic foramen to enter the lateral wall of the ischio-rectal fossa. At the anterior end of the pudendal canal, it branches into the perineal nerve and the dorsal nerve of the penis. The perineal nerve supplies the IC and BC muscles [37].

There were 49.2% of the participated patients with OD had ED. This indicated that ED was a significant problem among OD patients. This should be taken into consideration when dealing with male OD patients. To the best of the authors’ knowledge, this has not been previously reported in the literature.

The study was in accordance with previous studies regarding the presence of pudendal neuropathy among OD patients [13, 14, 38, 39]. Pudendal neuropathy is a stretch neuropathy due to the long-standing stretching of the pudendal nerve with straining during defecation. Long-term straining during defecation results in subluxation and sagging of the levator ani muscles [16]. This leads to excessive perineal downward descent which stretches the pudendal nerve from the point of its fixation at the ischial spine [13, 14]. Because the pudendal nerve winds around the ischial spine, the pudendal nerve is fixed at the ischial spine point and makes the distal part liable to stretch. It affects the distal part of the nerve that extends from the ischial spine to the pelvic floor muscles. Long-term stretching leads to neurapraxia that could be advanced to axonotmesis if stretch increased in severity [13]. This affects the function of all pudendal nerve supplied pelvic floor muscles including IC and BC muscles.

The percentage of patients with bilateral pudendal neuropathy was significantly higher among OD patients with ED. Pudendal neuropathy results in neurological changes in all pudendal nerve supplied muscles including EAS, IC and BC muscles [13, 14, 16, 17]. This explained the statistically significant difference between patients with ED versus those without ED regarding the percentage of patients with evidence of neuropathic abnormalities in the form of denervation and chronic reinnervation in the EAS and BC muscles. These neuropathic changes indicated that pudendal neuropathy had an essential role in the development of ED among OD patients. Regression analysis for some co-variables (Table 5) found that right PNTML (OR = 3.755) and left PNTML (OR = 3.770) were associated with significantly increasing the likelihood of development of ED. The OR of PNTML (whether the right side or the left side) indicated that, when fixing all other selected co-variables constant, one-point increase in the PNTML is associated with the odds of developing ED by a multiplicative factor of 3.7. These data were not previously assessed in the literature. In the situation that pudendal neuropathy led to ED, this could be considered a form of neurogenic impotence [16, 17, 40].

Subsequently, OD patients with ED had a significantly lower MOS and maximum squeezing anal pressure. This could be due to the presence of stretch pudendal neuropathy which weakens the EAS muscle, as well as weakens other pudendal nerve innervated muscles including IC and BC muscles. BC muscle is considered an integral part of the EAS muscle [41, 42]. It was reported that squeezing of EAS muscle is associated with BC muscle contraction [43]. Both BC and IC muscles share their contractile activity with the EAS [43–45]. The pelvic floor muscles are considered to form a single functional unit [7, 34]. Also, there is a concept that pathological changes in the EAS muscle (as the neuropathic changes presented in the present study) affect the erectile function with the development of ED. This concept is known as anocavernal erectile dysfunction syndrome [43, 45].

The patients with ED had a significantly higher maximum straining anal pressure in comparison to those patients without ED. This indicated that the paradoxical contraction of the anal sphincter muscles in OD was more severe among those patients with ED. This led to more straining during defecation with more stretch pudendal neuropathy with subsequent the development of ED [13, 16]. Subsequently, regression analysis for some co-variables (Table 5) found that maximum straining anal pressure (OR = 1.122) was associated with significantly increasing the likelihood of development of ED. The OR of maximum straining anal pressure indicated that, when fixing all other selected co-variables constant, one-point increase in the maximum straining anal pressure is associated with the odds of developing ED by a multiplicative factor of 1.122. This was not mentioned previously in the literature.

There was no statistically significant difference between patients with ED versus those without regarding the presence of neuropathic changes in the PR muscle. These neuropathic changes were due to the stretch neuropathy of the PR motor nerve supply which arises directly from the sacral plexus. This stretch could be due to the long-standing straining during defecation [46]. PR muscle has no role in the process of erection [7].

Anismus was present in EAS and PR muscles in a high percentage of patients. Anismus is the functional form of OD. This could be present as the only pathology in OD
(i.e., functional OD) or associated with other anatomical structural abnormalities as rectocele. These were in accordance with previous studies [13, 29].

The statistically significant negative correlation between erectile function domain of IIEF questionnaire and the time of toileting could be an indication that the more the OD severity measured as the duration of time of toileting with straining during defecation associated with more pudendal neuropathy and subsequently poorer ED. This was not assessed previously in the literature.

The statistically significant negative correlation between erectile function domain of IIEF questionnaire and the PAC-QoL could be due to the impact of ED severity on the patient’s QoL. Sexuality is considered a necessity for human well-being and abnormality in it affects the patient’s overall QoL [47]. It was reported that ED affects the individual well-being and QoL [48, 49].

The statistically significant positive correlation between erectile function domain of IIEF questionnaire and the maximum squeezing anal pressure could be because the better pelvic floor muscles strength associated with better erectile function measured by IIEF. Also, the statistically significant negative correlation between ED-EQoL questionnaire and the maximum squeezing anal pressure could be because the better muscle strength of pelvic floor muscles associated with better erectile function with consequently the better the erectile function related QoL. The EAS muscle is the main responsible muscle for the generation of the maximum squeezing anal pressure. It forms a functional unit with other pelvic floor muscles including IC and BC muscles [7]. So, the poor function of the EAS muscle was associated with poor BC and IC muscles function manifested as ED. The BC and IC muscles assumed to be involved in different pathologies affecting the EAS muscle as in case of OD associated with pudendal neuropathy [43, 44, 50–52].

Pudendal neuropathy and maximum straining anal pressure were essential risk factors for the development of ED detected by regression analysis. In spite that these variables were not correlated significantly with erectile function domain of IIEF questionnaire, they were essential likelihood factors for the development of ED. These data were not reported previously in the literature.

The explanation that some patients with OD had no ED while others developed ED could be due to the following: (I) Excessive straining results in stretch of the inferior rectal branch of the pudendal nerve. This results in neuropathic changes in the EAS muscle. However, in severe and long-standing OD, excessive straining results in more severe pudendal neuropathy. This could affect all branches of the pudendal nerve including the perineal branch that supplies the IC and BC muscles with subsequently ED development. (II) From the anatomical and physiological point of view, the IC and BC muscles share their contractile activity with the EAS muscle. Consequently, the IC and BC muscles assumed to be involved in different pathologies affecting the EAS muscle [43, 44]. So, the more involvement of the EAS muscle was associated with more involvement in the IC and BC muscles. (III) The consequences of OD are a wide spectrum of neuromuscular abnormalities that extends from mild condition to very severe condition in the form of severe bilateral pudendal neuropathy that results in ED and even fecal incontinence as a late complication [13, 14, 53].

5 Limitations of the work

(i) Assessment of erectile function using the nocturnal penile tumescence assessment was not done [54]. This was in agreement with other researches [47]. Assessment of ED was done directly by subjective assessment questionnaire in the form of erectile function domain of IIEF questionnaire and indirectly by objective assessment of the maximum squeezing anal pressure which reflect the strength of contraction of EAS, as well as other pelvic floor muscles including IC and BC muscles [18, 20, 47].

(ii) The research did not evaluate pudendal somatosensory evoked potential. It is essential for diagnosis of patients with neurological disorders as central lesion (i.e., upper motor neuron lesion) and cauda equine lesions. However, patients with obvious neurological disorders were excluded from the study [55, 56].

(iii) BC reflex was not assessed. It was reported that the latency of the pelvic floor electrophysiological reflexes lacks sensitivity in detecting minimal to moderate grades of pudendal neuropathy. Also, they are not sensitive to detect incomplete nerve lesions (whether demyelinating or axonal neuropathy). This is because their assessment is based on conduction and not on compound muscle action potential amplitude [57]. The pelvic floor electrophysiological reflexes have a wide physiological range of latencies. When there is a minimal delay in their latency, it could still be within the accepted limit [53]. Consequently, when there is pelvic floor electrophysiological reflex within the normal range of latency, this does not exclude a lesion [14, 30, 55–57].

(iv) The limited number of included patients could be due to the wide range of exclusion criteria in the present study. Further studies on a larger scale of patients are recommended.
This study is considered the first study that assessed the presence of ED among OD patients. Also, this study is considered the first study that assessed the relation between pelvic floor electrophysiological changes and ED among male OD patients. Early diagnosis of OD is essential for prevention of OD progression to more severe and more advanced stage that usually associated with more pudendal neuropathy with all its consequences as ED. Any physician dealing with a male patient with OD should be aware of the risk of development of ED. Enquiring about this issue is essential for proper diagnosis and treatment of OD and its consequences. The presence of severe and bilateral pudendal neuropathy associated with increased maximum straining anal pressure in a patient with OD should alert the treating physician for the risk of the presence of ED. This will encourage the role of pelvic floor rehabilitation for OD patients for early prevention, as well as for improvement of ED. This clinical problem could be treated by pelvic floor muscles exercises, as well as pelvic floor biofeedback therapy for improvement of the erectile function in association with improvement of OD [18, 47, 58].

The pelvic floor is considered a complex multifunctional unit which is essential for proper fecal continence and defecation; urinary continence and urination; and reproductive functions [7]. Pelvic floor muscle training helps in improving the strength of the pelvic floor muscles including the IC and BC muscles [58, 59]. It is recommended that pelvic floor rehabilitation should start as early as possible for treatment of OD among male patients. It should be started early enough before the development of ED.

6 Conclusions
In conclusion, ED is prevalent among OD patients. It is associated with characteristic pelvic floor electrophysiological abnormalities in the form of bilateral pudendal neuropathy with neuropathic abnormalities in the EAS and BC muscles. Pelvic floor electrophysiological changes in OD vary from minimal to severe neuromuscular abnormalities that usually accompanying ED. Pudendal neuropathy and increased maximum straining anal pressure are essential risk factors for increasing the likelihood of development of ED in patients with OD.

Abbreviations
BC: Bulbocavernosus; EAS: External anal sphincter; ED: Erectile dysfunction; ED-EQoL: Erectile Dysfunction-Effect on Quality of Life Questionnaire; EMG: Electromyography; IC: Ischiocavernosus; IIEF: International Index of Erectile Function; MODS: Modified Obstructed Defecation Score; MRI: Magnetic resonance imaging; QoL: Quality of life; PAC-QoL: Patient Assessment of Constipation-Quality of Life Questionnaire; PNTML: Pudendal nerve terminal motor latency; PR: Puborectalis; OD: Obstructed defecation; OR: Odd ratio; SD: Standard deviation.

Acknowledgements
The authors are grateful to Mariam Kamal Aziz Saba for her assistance in the statistical analysis. The authors are grateful to Maria Kamal Aziz Saba for her assistance in the preparation of the figures.

Authors’ contributions (MSE) contributed in the concepts, design, definition of intellectual content, clinical studies, data acquisition and manuscript revision. (EKAS) contributed in the concepts, design and definition of intellectual content, and did literature search, clinical studies, data acquisition and analysis, manuscript preparation, editing and revision. All the authors read and approved the manuscript.

Funding
The authors received no specific funding for this work. The authors declare that no financial or material support was provided by any parties and that there are no equity interests, patent rights or corporate affiliations for this work. This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. There were no sponsors or funders (other than the named author) played any role in study design, data collection and analysis, decision to publish and preparation of the manuscript. All research facilities are available in our department for with no restrictions.

Availability of data and materials
The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations
Ethics approval and consent to participate
The local Ethics Committee of Faculty of Medicine, Alexandria University, Egypt (IRB NO.00012098-FWA NO.00018699) approved the study. Date of approval: 15/7/2019. Serial number: 0304356. A written informed consent was given by each.

Consent for publication
Not applicable

Competing interests
The authors declare that they have no competing interests.

Received: 10 June 2021   Accepted: 6 August 2021
Published online: 22 September 2021

References
1. Pucciani F, Ringressi M (2012) Obstructed defecation: the role of anorectal manometry. Tech Coloproctol 16:67–72
2. Hoore A, Penninckx F (2003) Obstructed defecation. Colorectal Dis 5:280–287
3. Longstreth GF, Thompson WG, Chey WD, Houghton LA, Mearin F, Spiller RC (2006) Functional bowel disorders Gastroenterol 130:1480–1491
4. Spiller R, Thompson W (2010) Bowel disorders. Am J Gastroenterol 105:775–785
5. Andromanolakis N, Skandalakis P, Troupi T, Filippou D (2006) Constipation of anorectal outlet obstruction: pathophysiology, evaluation and management. J Gastro Hepatol 21:638–646
6. Madbouly KM, Abbas KS, Saba EK (2017) Bilateral posterior tibial nerve stimulation in the treatment of rectal evacuation disorder: a preliminary report. Dis Colon Rectum 60:311–317
7. Pischedda A, Fusco F, Curreli A, Grimoldi G, Farina FP (2013) Pelvic floor and sexual male dysfunction. Arch Ital Urol Androl 85 (1):1–7
8. Kandeel FR, Koussa VK, Sverdloff RS (2001) Male sexual function and its disorders: physiology, pathophysiology, clinical investigation, and treatment. Endocr Rev 22:342–388
9. Cohen D, Gonzalez J, Goldstein I (2016) The role of pelvic floor muscles in male sexual function and pelvic pain. Sex Med Rev 4:53–62
10. MacDonagh R, Ewings P, Porter T (2002) The effect of erectile dysfunction on quality of life: psychometric testing of a new quality of life measure for patients with erectile dysfunction. J Urol 167 (1):212–217
11. Mestre M, Lleberia J, Pubill J, Espuña-Pons M (2015) Questionnaires in the assessment of sexual function in women with urinary incontinence and pelvic organ prolapse. Actas Urol Esp 39 (3):175–182
12. Caretta N, Foresta C (2007) Clinical diagnostic approach to erectile dysfunction. Minerva Ginecol 59:51–61
13. Saba EKA, El-Tantawi GAY, Zahran MH, Ibrahim KI, Shehata MA, Sultan HA et al (2015) Pelvic floor electrophysiology patterns associated with obstructed defecation (abstract). Int J Med Health Sci 2 (7):118
14. Saba EKA, Elsawy A (2019) Pelvic floor electrophysiological changes associated with female pelvic organ prolapse. World J Med Sci 16 (2):79–85
15. Antuna VC, Gomez JMF, Escaf S, Fernandez-Gonzalez F (2008) Neurogenic etiology in patients with erectile dysfunction. Arch Esp Urol 61:403–411
16. Shafik A (1994) Pudendal canal decompression in the treatment of erectile dysfunction. Arch Androl 32 (2):141–149
17. Naouar S, Braiek S, El Kamel R (2017) Erectile dysfunction secondary to pudendal nerve injury complicating orthopedic surgery: practical recommendations. J Curr Surg 7 (1–2):1–3
18. Al-Helou MR, Abdul-Hady H, Fathalla MA, Zakiy A, Hussein O, El Gahndour T (2014) The role of biofeedback in the rehabilitation of veno-occlusive erectile dysfunction. Egypt Rheumatol Rehabil 41:179–186
19. Drost J, Harris L (2006) Diagnosis and management of chronic constipation. J Am Acad Phys Assistants 19:24–30
20. Rosen RC, Cappelleri JC, Gendrano N (2002) The International Index of Erectile Function (IIEF): a state-of-the-science review. Int J Impot Res 14 (4):226–244
21. Hamdan FB, Al-Matubsi HY (2009) Assessment of erectile dysfunction in diabetic patients. Int J Androl 32:176–185
22. Agu AU, Esom EE, Anyaeri PS, Nzekwe KC, Chimie SC, Ikele II et al (2019) Obesity indices and academic performance of medical students of Igbo extraction at College of Medicine, University of Nigeria. World J Med Sci 16 (4):191–195
23. Sharma S, Agarwal BB (2012) Scoring systems in evaluation of constipation and obstructed defecation syndrome (ODS). J Int Med Sci Acad 25 (1):57–59
24. Bassotti G, Villanacci V (2013) A practical approach to diagnosis and management of functional constipation in adults. Intern Emerg Med 8 (4):275–282
25. Saba EKA, El-Tantawi GAY, Zahran MH, Ibrahim IK, Shehata MA, Sultan HA et al (2015) Evaluation of digital assessment of anal sphincter muscle strength (abstract). Int J Med Health Sci 2 (9):122
26. Lang J, Brown H, Crombie E (2007) Assessment of the anal sphincter muscle: comparison of a digital and a manometric technique. Physiotherapy 93:121–128
27. Gadel Hak N, El-hemaly M, Hamdy E (2011) Pelvic floor dyssynergia: efficacy of biofeedback training. Arab J Gastroenterol 12:15–19
28. Parry AH, Wani AH (2020) Evaluation of obstructed defecation syndrome (ODS) using magnetic resonance defecography (MRD). Egyptian Journal of Radiology and Nuclear Medicine 51:78 (https://doi.org/10.1186/s43055-020-00197-z)
29. El-Shazly W, El-Nekady A, Hassan H (2010) Role of dynamic magnetic imaging in management of obstructed defecation case series. Int J Surg 8:274–282
30. Roberts M (2008) Neurophysiology in neurourology. Muscle Nerve 38:815–836
57. Fowler C (1995) Pelvic floor neurophysiology. In: Binnie C, Cooper R, Fowler C, Mauguiere F, Prior P (eds) Clinical neurophysiology: electromyography, nerve conduction and evoked potentials. Butterworth-Heinemann, Oxford, pp 233–252

58. Van Kampen M, De Weerdt W, Claes H, Feys H, De Maeyer M, Van Poppel H (2003) Treatment of erectile dysfunction by perineal exercise, electromyographic biofeedback, and electrical stimulation. Phys Ther 83 (6):536–543

59. Claes H, Baert L (1993) Pelvic floor exercise versus surgery in the treatment of impotence. Br J Urol 71:52–57

**Publisher's Note**
Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.