Antepartum pudendal neuropathy and the effect of vaginal delivery in women with type 1 diabetes mellitus in pregnancy

Rhona Mahony¹, Conor O’ Brien¹, Brendan Kinsley², Richard Firth², Mary Coffey³, Ronan O’Connell⁴
Colm O’Herlihy¹

Department of Obstetrics and Gynaecology, University College Dublin, National Maternity Hospital, Dublin.¹Department of Medicine, University College Dublin,Mater Misericordiae Hospital, Dublin.² National Maternity Hospital³, Department of Surgery, University College Dublin,Mater Misericordiae Hospital,Dublin⁴

Abstract

Vaginal delivery is associated with a significant risk of pelvic floor neuropathy and anal sphincter dysfunction. Because insulin-dependent diabetes mellitus (IDDM) predisposes to neuropathy, our aim was to determine the incidence of pudendal nerve dysfunction in a cohort of pregnant insulin-dependent diabetic patients and to examine the effect of vaginal delivery on pudendal nerve and anal sphincter function. Materials and Methods: The pudendal nerve was evaluated using electromyography of the anal sphincter (EMG) and clitoral anal reflex (CAR) assessment, during the third trimester in 16 pregnant insulin-dependent diabetic women. Upper limb neurological assessment included median motor, ulnar f-wave, median and ulnar sensory nerve and median transpalmar nerve conduction studies, while the lower limbs were assessed using tibial motor and f-wave studies. Anal manometry and endoanal ultrasound studies were also performed. Thirteen women underwent full reassessment at three months postpartum. Results: An increased sensory threshold (>9mA) on CAR was demonstrable antenatally in 5 women on the right and 8 on the left. Seven women showed prolonged distal latency (>42ms) on the right and in 8 on the left. Five women showed prolongation of median transpalmar nerve conduction studies (>2.2ms), which was associated with diabetes of greater than 12 years duration (p=0.029). The median squeeze pressure was 129mmHg and median resting pressure was 62mmHg. There were no significant changes in nerve conduction or anal manometric pressures postnatally. Discussion: Pregnant women with type 1 IDDM exhibit subtle changes of axonal neuropathy and demyelination, but we found no increased susceptibility to pudendal nerve injury during vaginal childbirth. (Int J Diabetes Metab 14: 82-86, 2006)

Key Words: Childbirth, Insulin dependent diabetes, pudendal nerve.

Introduction

In the general population, faecal incontinence occurs with a female to male preponderance of 8:1, consistent with pelvic floor injury following childbirth as the primary causative factor.¹²³ Up to 25% of women experience some alteration in faecal continence postpartum and up to one third have ultrasound evidence of anal sphincter trauma following apparently uncomplicated vaginal delivery.¹² The aetiology of faecal incontinence following vaginal delivery reflects either direct anal sphincter disruption or pudendal nerve injury or a combination of both. The anatomy of the pudendal nerve renders it particularly susceptible to traction and compression injuries within the pelvis.⁴⁻⁸

Diabetes mellitus is the most common cause of polyneuropathy in the developed countries,⁹ often manifesting as a distal symmetric sensory polyneuropathy, although almost any form of neuropathy can occur. Pregnant women not uncommonly experience neuropathies which may be focal or multiple and specific to gestation, the primary example of which is carpal tunnel syndrome secondary to median nerve compression.⁸⁻¹⁰

Because diabetes is associated with increased susceptibility to both focal and polyneuropathy, it was our hypothesis that the pudendal nerve might be more susceptible to injury in pregnant diabetic women during vaginal childbirth. The aim of this study was to determine the susceptibility of the pudendal nerve to injury during vaginal childbirth in a cohort of pregnant insulin-dependent women.

Materials and Methods

Sixteen pregnant women with type 1 IDDM (9 primigravid and 7 multigravid) were recruited from the dedicated diabetic antenatal outpatient clinic of the National Maternity Hospital and were assessed during the third trimester of pregnancy. Women with a history of anorectal disease, previous third degree tear or anorectal surgery and irritable bowel syndrome were excluded from the study. Antenatal continence assessment consisted of a structured bowel function questionnaire modified from Jorge and Wexner.¹¹ This score was based on the presence of flatal, liquid and solid faecal incontinence and included faecal urgency as this is a common and debilitating symptom. A score of 0 implies complete continence while a score of 20 implies complete incontinence. Anal sphincter function was assessed using anal manometry and endoanal ultrasound. Neurophysiological screening for polyneuropathy consisted of upper and lower limb motor and sensory nerve conduction studies. The pudendal nerve was assessed using clitoral anal reflex (CAR) and surface electromyography (EMG) of the anal sphincter.¹² Postnatally patients were assessed at 3 months postpartum, at which time the antenatal neurophysiological and anal physiological investigations were repeated and compared to antenatal values.
### Table 1: Antenatal and postnatal anal physiology results in a cohort of insulin dependent diabetic women who underwent vaginal delivery (N=11)

<table>
<thead>
<tr>
<th>Anal Physiology Results</th>
<th>Antenatal N=16</th>
<th>Postnatal N=11</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median Continence Score</td>
<td>0 (0-3)</td>
<td>0 (0-3)</td>
<td>0.86</td>
</tr>
<tr>
<td>Anal Manometry</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median Squeeze Pressure/mMg</td>
<td>119 (75-200)</td>
<td>112 (50-155)</td>
<td>0.107</td>
</tr>
<tr>
<td>Median Resting Pressure/mMg</td>
<td>59 (37-100)</td>
<td>65 (37-94)</td>
<td>0.325</td>
</tr>
<tr>
<td>Median Squeeze Increment/mMg</td>
<td>49 (14-146)</td>
<td>44 (3-87)</td>
<td>0.042</td>
</tr>
<tr>
<td>Endoanal Ultrasound</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal IAS</td>
<td>13</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>&lt;1 Quadrant Defect</td>
<td>1</td>
<td>1</td>
<td>0.436</td>
</tr>
<tr>
<td>Normal EAS</td>
<td>13</td>
<td>9</td>
<td>0.102</td>
</tr>
<tr>
<td>&gt;1 Quadrant Defect</td>
<td>2</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

IAS = Internal anal Sphincter  
EAS = External Anal Sphincter  
P-value derived from Wilcoxon Signed Ranks Test

### Table 2: Antenatal and postnatal pudendal and median nerve conduction studies in a cohort of insulin dependent women who underwent vaginal delivery

<table>
<thead>
<tr>
<th>Pudendal and median nerve conduction studies</th>
<th>Antenatal N=16</th>
<th>Postnatal N=11</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median CAR ST/mA</td>
<td>7.7</td>
<td>7.0</td>
<td>R 0.07</td>
</tr>
<tr>
<td>Prolonged &gt;9mA</td>
<td>N=3</td>
<td>N=4</td>
<td>L 0.12</td>
</tr>
<tr>
<td>Median CAR Latency/ms</td>
<td>40.8</td>
<td>38.7</td>
<td>R 0.899</td>
</tr>
<tr>
<td>Prolonged &gt;42ms</td>
<td>N=5</td>
<td>N=3</td>
<td>L 0.48</td>
</tr>
<tr>
<td>Median Transpalmer Latency</td>
<td>2.1ms</td>
<td>2.1ms</td>
<td>0.62</td>
</tr>
<tr>
<td>Prolonged &gt;2.2ms</td>
<td>N=1</td>
<td>N=2</td>
<td></td>
</tr>
</tbody>
</table>

P-value derived from Wilcoxon Signed Ranks Test

### Table 3: Antenatal and postnatal peripheral nerve conduction studies in a cohort of insulin dependent diabetic women

<table>
<thead>
<tr>
<th>Peripheral nerve conduction studies</th>
<th>Antenatal N=16</th>
<th>Postnatal N=13</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median Nerve/ms</td>
<td>3.2</td>
<td>3.1</td>
<td>0.88</td>
</tr>
<tr>
<td>Ulnar Nerve/ms</td>
<td>2.9</td>
<td>2.7</td>
<td>0.83</td>
</tr>
<tr>
<td>Ulnar-f wave/ms</td>
<td>27.7</td>
<td>26.7</td>
<td>0.92</td>
</tr>
<tr>
<td>Tibial-f-wave/ms</td>
<td>49.9</td>
<td>49.05</td>
<td>0.25</td>
</tr>
</tbody>
</table>

P-value derived from Wilcoxon Signed Ranks Test

Anal endosonography was performed using a Bruel and Kjaer 10 MHz rotating transanal endoprobe (Naerum, Denmark). Endosonographic injury was graded for both the internal and external anal sphincters according to whether the injury was full or partial thickness and the number of quadrants of muscle involved.  
Anorectal manometry was performed using a Synectics PC Polygraf Lower GI system (Synectics, Stokholm, Sweden)
Peripheral nerve conduction studies were performed using standard AAEM protocols on a Nicolet Viking Quest System (Nicolet/Biomedical/IBM/USA) with standard settings and filters for both motor and sensory nerves. Surface electrodes were placed over appropriate anatomical sites while the supplying nerve was stimulated by a surface electrical stimulator. The conduction velocity, the amplitude of response and distal latency were recorded in each case. Late responses (f-wave) were also recorded to assess conduction along the entire nerve. The median, ulnar and transpalmar median nerve (TPM) distal latency were examined.

Pudendal nerve conduction studies using the clitoral anal reflex (CAR) were performed using a Nicolet Viking Quest System (Nicolet/Biomedical/IBM/USA). The CAR was performed by stimulating the paracloacal area using a prong electrode firstly on the left side and then on the right while recording at both left and right sphincter areas. Both the conduction latency and the sensory threshold (ST) were recorded. The current intensity used was below the individual tolerance threshold (20-30mv) using a pulse duration of 0.2ms/s; sweep speed of 10ms/cm, amplifier gain set at 200uv/cm and standard sensory filter settings.

All data were stored on an IBM compatible database. Statistical analysis was performed using the Chi Square test and Wilcoxon Signed Rank Test as appropriate on an SPSS™ for windows statistical software package version 11 (SPSS Inc., Chicago, IL, USA).

The study was approved by the Ethical Committees of the National Maternity Hospital, Dublin. All patients gave written informed consent.

**Results**

The median age was 30 (range 23-42). In the 16 women studied, Median parity was 1 (range = 0-4) and median duration of diabetes mellitus was 11.5 years (range 3-22 years). Three women (19%) had evidence of diabetic retinopathy, while none had nephropathy or microalbuminuria. Median booking glycosylated haemoglobin was 7.25% ( range 5.0-9.9%) and median number of antenatal hospital admissions for stabilization of diabetic control was 1 ( range 0-5).

The median antenatal continence score was 0/20 (range 0-3). Median resting pressure was 56mmHg (range 37-100 mmHg), median squeeze pressure was 117.5 mmHg (range 75-200 mmHg), and median squeeze increment was 48 mmHg (range 14-146 mmHg).

Thirteen women (81%) had normal endoanal ultrasound findings, while three multiparous patients had an abnormal scan (Table 1).

Antenatally, the median sensory threshold of the right pudendal nerve was 8.3mA (range 4.4-23.1mA) and of the left was 8.8mA (range =3.4-21.6mA). The CAR sensory threshold was increased (>9mA) in five patients on the right and eight on the left pudendal nerve. Median CAR distal latency was 40.8ms (range 21.6-59.5ms) on the right side and was 40.5 (range 30.7-52.2ms) on the left. The CAR distal latency was prolonged (>42ms) in 7 patients (43.7%) on the right side and in 8 (50%) on the left (Table 2).

The results of the peripheral nerve conduction studies are given in table 3. The median TPM distal latency was at the upper limit of normal and correlated with the duration of disease.

Labour was induced in 10 women. The median duration of labour was 194.5 mins (range 3-699 mins). Ten women received epidural anaesthesia during labour for pain relief. Eight women (50%) underwent spontaneous vaginal deliveries and five women (31%) were delivered instrumentally. Three women were delivered by caesarean sections of which two were prelabour procedures and one an emergency intrapartum caesarean section at 7cm dilatation for dystocia. Median birth weight was 4,000g (mean 3847, range 2660g-4270g); of the 13 women who delivered vaginally, four women had an intact perineum following delivery, five underwent episiotomy and 4 women sustained second degree lacerations. No woman in the study sustained a third degree perineal tear.

Thirteen women returned for full postnatal assessment, including 11 of the 13 women who delivered vaginally. Following vaginal delivery the median postnatal continence score was unchanged at 0 (range 0-4). There was no significant change in either anal manometric median squeeze pressure (p=0.107) or median resting pressure (p=0.325) postnatally although the squeeze pressure increment was significantly lower following vaginal delivery (table 2). There were no significant differences between CAR sensory thresholds on the right (p=0.069) and left pudendal nerves (p=0.123) nor in the distal latencies on the right (p=0.889) and left (p=0.484). (Table 2).

There was no significant change in either neurophysiological or manometric findings in the two women who returned for postnatal assessment following caesarean section. There was no change in the peripheral nerve conduction studies postnatally in the thirteen women who returned for assessment (Table 3).

**Discussion**

This study was designed to answer the clinically important question of whether type 1 IDDM is associated with pudendal nerve neuropathy and whether the presence of diabetes mellitus predisposes the pudendal nerve to injury during parturition. The results are reassuring in both respects. Almost half the patients exhibited a subtle mixed motor and sensory polyneuropathy on antenatal investigation, consistent with findings common in diabetes mellitus but pregnancy and parturition was generally not associated with significant progression of neuropathy. A reduction in median anal canal squeeze pressure increment was observed postnatally, a recognized finding in the non diabetic population. There was no significant reduction in either median anal canal resting or squeeze pressures and these values remained well within normal limits and were not associated with any symptomatic deterioration. Most
importantly, there was no significant alteration in faecal continence scores, anal manometry pressures and endoanal ultrasound results after vaginal delivery, although the numbers included in the study are relatively small.

The pudendal nerve is the predominant nerve supply to the pelvic floor and is derived from the S2-4 spinal cord nerve roots. It follows a tortuous course running in the posterior hollow of the pelvis along the piriformis muscle before exiting the pelvis by crossing the ischial spine and entering the ischiorectal fossa via the lesser sciatic foramen. It runs forwards in the fibrous tunnel of Alcock's canal, on the medial surface of the obturator internus muscle and enters the perineum. The pudendal nerve or its branches may be injured during vaginal delivery anywhere along its path. It is particularly vulnerable as it crosses bony prominences such as the ischial spine. Nerve injury probably results from a combination of direct trauma secondary to compression or traction injury to the pudendal nerve during descent of the fetal head in the second stage of labour. Traction injury can be further exacerbated by abnormal perineal descent, which can persist for several months following vaginal delivery.21

Pudendal nerve trauma can result in both demyelinating and axonal types of injury.6 Prolongation of second stage of labour and fetal macrosomia are particularly significant aetiological factors and injury may be cumulative with successive vaginal deliveries.4,22

Diabetic neuropathies include both focal neuropathies and diffuse polyneuropathy. Polyneuropathy, has yet to be fully explained by a single disease mechanism despite intensive investigation and the pathogenesis is considered to be multifactorial. Increasing evidence exists to link abnormalities in the polyol pathway to the onset of diabetic neuropathy.28,29 In addition, nerve regeneration is less effective in patients with type 1 IDDM.25

Almost half of the women in this study had evidence of pudendal nerve conduction abnormality although this was not manifested clinically as altered faecal continence nor did it appear to progress following vaginal delivery. However, a high incidence of faecal incontinence in the general diabetic population has been described and this has been associated in a number of studies with the presence of peripheral neuropathy.26

It is particularly common when diabetes is complicated by peripheral neuropathy27 although autonomic neuropathy has also been suggested as a contributing cause.28,29 Alternatively, a combination of somatic pudendal neuropathy and autonomic neuropathy has been proposed.30,31,32

In conclusion, this pregnant diabetic cohort showed abnormalities consistent with subtle axonal and demyelinating changes in both motor and sensory nerves including the pudendal nerve and these changes were more pronounced in longstanding disease. Apart from some decline in median squeeze increment there was no evidence of significant anal sphincter decompensation following vaginal delivery. These data would suggest that vaginal delivery is a safe option in women with type 1 IDDM.

References