

Влияние автономного электростимулятора желудочно-кишечного тракта и слизистых оболочек на состояние углеводного и липидного обмена у больных сахарным диабетом*

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По данным A.S.Krolewski и соавт. [1], у больных сахарным диабетом (СД) 1 типа летальность вследствие инфаркта миокарда в возрасте 55 лет составляет 35% по сравнению с 6% у лиц того же возраста, но не страдающих диабетом. Повышение частоты инфаркта миокарда у больных СД является следствием нескольких факторов: наличие автономной нейропатии, микроангиопатии и диабетической кардиомиопатии. СД — частая причина слепоты, смерти от уремии. Более 40% всех не обусловленных травмой ампутаций нижних конечностей проводится в связи с синдромом диабетической стопы и гангреной нижних конечностей. Дистальная полинейропатия и автономная нейропатия являются причиной низкого качества жизни, нарушения трудоспособности и инвалидизации большого количества больных СД.

Несмотря на общность патогенеза макро- и микроангиопатий, клинические и патоморфологические характеристики этих поражений различны. Атеросклероз, который значительно чаще встречается при СД, развивается как результат взаимодействия нескольких факторов риска: гиперлипидемия и дислипидемия, инсулиновая резистентность и гиперинсулинемия, гипертензия, повышенная агрегация тромбоцитов, повышение свертываемости крови, снижение фибринолиза, нарушение функции эндотелия сосудов.

Потенциальные атерогенные факторы при диабете [2]:

1) Изменение артериальной сосудистой стенки — изменения в клеточной биологии, эндотелиальных клеток, клеток гладкой мускулатуры; изменения метаболизма, липогенез, стимулированный инсулином, накопление сорбитола, гипоксия, на-

копление конечных продуктов гликозилирования.

2) Гипертензия.

3) Нефропатия — с гипертензией или без нее, недостаточность функции почек, микро- и макроальбуминурия.

4) Нарушение тромбогенеза — патология тромбоцитов, повышение уровня фибриногена, снижение активности PAI-1 (ингибитор-1 активатора плазминогена).

5) Гипергликемия.

6) Ожирение, (самостоятельное влияние или через другие атерогенные факторы).

7) Гиперинсулинемия и/или резистентность к инсулину.

8) Дислипидемия.

Макроангиопатия при СД практически не отличается от атеросклеротического поражения сосудов, которое имеет место и у больных без диабета, но поражение сосудов у больных диабетом проявляется на 8-10 лет раньше, чем у их сверстников, не страдающих диабетом. Одним из факторов развития атеросклероза при СД наряду с нарушением углеводного обмена является дислипидемия, которая в большей степени и более часто имеет место при СД 2 типа. Нарушение липидного обмена, как правило, не сопровождается достоверными изменениями концентрации холестерина липопротеидов низкой плотности (ЛПНП) в плазме крови [3]. Изучению патогенеза нарушений липидного обмена при СД и методам его коррекции посвящено большое количество работ [4, 5, 6]. Тем не менее вопросы эффективной терапии нарушения обмена липидов при СД остаются актуальными.

Материалы и методы

Целью исследования было изучение влияния автономного электростимулятора желудочно-кишечного тракта и слизистых оболочек (АЭС ЖКТ и СО - ЭН), производимого НПО «Эко-

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