

Диабетическая ретинопатия: современные проблемы (взгляд диабетолога)

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Диабетическая ретинопатия (ДР) является хорошо известным и легко диагностируемым хроническим осложнением со стороны органов зрения, которое может быть выявлено практически у всех пациентов с сахарным диабетом (СД). В настоящее время в мире насчитывается 45 миллионов слепых людей и 180 миллионов со сниженным зрением. За последующие 50 лет, предположительно, количество слепых возрастет на 27% и лиц со сниженным зрением на 45% соответственно (ВОЗ 2002 г.). ДР является наиболее частой причиной слепоты среди лиц трудоспособного возраста в большинстве стран мира. ДР была описана впервые более 100 лет назад MacKessie (1879), однако и сегодня данное осложнение представляет огромную проблему для здравоохранения. Несмотря на широкое внедрение для диагностики и лечения новых эффективных инструментальных методов, ДР остается по-прежнему главной причиной утраты зрения. В течение ряда лет изучается как естественное развитие данного осложнения СД, так и его взаимосвязь с некоторыми хорошо изученными зрительными патогенезами.

Стоимость хирургического лечения (вытравки) 1 больного с ДР составляет до 12 000 долл. в год, что соответствует стоимости лечения 65 больных с помощью лазерной фотокоагуляции или скрининга и раннего лечения на обратной стадии 126 больных.

Приводятся разные цифры распространенности ДР при СД 1 и 2 в разных странах. В большинстве работ указывается, что обычно ДР не выявляется в течение первых пяти лет заболевания СД 1 и может быть отмечена впервые в период препубертата при плохом контроле СД. Тем не менее, начало диабета в препубертатном периоде может быть самостоятельным фактором риска для более позднего прогрессирования пролиферативной ДР [1]. В противоположность этому, у больных с позднодиагностированным СД 2, признаки ДР выявляются в момент диагностики заболевания у 7–30% пациентов. Причем, пролиферативная ДР не составляет у них большой проблемы, в противоположность СД 1, тогда как диабетическая макулопатия становится главной причиной ухудшения остроты зрения [2]. Необходимо подчеркнуть, что при длительности течения СД более 10 лет случаи отека макулярной области встречаются с одинаковой частотой независимо от раннего или позднего дебюта диабета.

Существует мнение, что имеется ряд локальных факторов, которые могут снизить риск прогрессирования диабетической ретинопатии. Среди них наиболее более 2 дигетерич (Rand et al. 1985) [3], особенно при наличии HLA-DR фенотипов 4/0, 3/0 и X/X; глаукома или атрофия зрительного нерва, ретикулярная деструкция вследствие инфекции, воспаления или врожденной патологии сетчатки (Selander, 1968) [4]. Среди факторов, вызывающих прогрессирование диабетической ретинопатии (ДР) [3] можно назвать следующие:

- степень компенсации углеводного обмена;
- продолжительность диабета;
- возраст;

- гипертония;
- курение;
- заболевания почек;
- беременность;
- генетическая предрасположенность.

Диабетическая ретинопатия характеризуется наличием специфических аномалий в сосудах и тканях сетчатки. Для нее характерно изменение калибра и интимальности ретинальных сосудов, появление микроаневризм, кровоизлияний, отека, твердых и мягких экссудатов, новообразованных сосудов, глияльной пролиферации, интравитреальных тракций.

Изучение морфологических изменений при ДР выявило утолщение базальной мембраны (Friedenwald, 1950), потерю капиллярных перипортов (Kuwahara and Cogan, 1963), вследствие этого – развитие аскалярности капилляров, что ведет к тому, что микрокапилляры представлены трубками, состоящими из базальной мембраны. Перфузия кислорода через их стенку ухудшается и развиваются ишемия и гипоксия сетчатки. Все это, в конечном итоге, ведет к развитию неоваскуляризации. Новообразованные сосуды, в свою очередь, функционально бесполезны и становятся новым источником кровоизлияний [5, 6].

По мнению E.R. Westman (1991), сетчатка может быть особенно чувствительна к повреждению, поскольку имеет самую высокую скорость утилизации глюкозы и кислорода на единицу веса, чем какая-либо другая ткань, и имеет высокую активность гликолитического и аэробного патологического пути метаболизма глюкозы. Неоспоримым является факт, что главную роль в развитии ДР играет хроническая гипергликемия. Имеются данные, полученные в результате многоцентровых исследований, выполненных при сахарном диабете 1 и 2 типа, подтверждающие первостепенное значение нормогликемии. Многолетние наблюдения за пациентами с сахарным диабетом 1 типа, у которых современными методами интенсифицированной инсулинотерапии, самоконтролем, обучением удалось в течение длительного времени поддерживать состояние нормогликемии, подтвердили значительное снижение риска прогрессирования микрососудистых осложнений. Результаты многоцентрового международного исследования – Diabetes Control and Complication Trial (DCCT 1993) [7], – показали, что поддержание удовлетворительного гликемического контроля в группе лиц без сосудистых осложнений способствовало снижению риска развития:

- диабетической ретинопатии – на 76%;
- диабетической нейропатии – на 60%;
- микроальбуминурии – на 39%;
- альбуминурии – на 54%.

Многоцентровое исследование, выполненное у больных с СД 2 в Великобритании, и его результаты были представлены в 1998 г. Оказалось, что и при СД 2 нормализация показателей углеводного обмена также играет существенную роль, как важнейший фактор снижения риска прогрессирования сосудистой патологии – United Kingdom Prospective Diabetes

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