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Evidence based practical management of type 2 diabetes

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SOCIETY OF RURAL PHYSICIANS OF CANADA

EVIDENCE-BASED PRACTICAL MANAGEMENT OF TYPE 2 DIABETES

Type 2 Diabetes Flow Chart- 2001

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Since rural physicians deal with a broad scope of primary and secondary health care, type 2 diabetes is one of the commonest diseases encountered. Rural areas serving First Nations communities have a prevalence of 30%,[1] while it sits at 4% for all Canadians.[2] Management of diabetes has recently been the focus of both new Canadian guidelines[3] and the first large scale RCT specifically studying type 2 diabetes.[4]

Given the multitude of tests available, the encouragement toward better monitoring and management, how should one proceed? To this end, the Society of Rura1 Physicians of Canada has undertaken this review of the literature and designed a Type 2 Diabetic Flow Chart. The flow chart is designed for a patient's chart with easy access of clinical and laboratory values. It also contains information relevant to diabetes and co-morbid conditions, and is meant to assist the practitioner in developing an informed up to date, straight forward approach to the office management of type 2 diabetes. The chart is designed to be photocopied, or it can be downloaded from the SRPC website (www.srpc.ca).

A French version is available on request.

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INTRODUCTION

Type 2 diabetes is a common primary care disease with serious repercussions, both for affected patients and our health care system. The 1998 publication of a 20 year prospective study of over 5,000 patients refocused our attention on the morbidity and mortality of type 2 diabetes. The United Kingdom Prospective Diabetes Study (UKPDS)[4] reinforced the importance of both monitoring diabetes control and the patient's co-morbid conditions. Rural physicians who work in First Nations regions are inundated with a heavy caseload of diabetics, who may or may not be comparable to those studied overseas, but who certainly suffer similar consequences and at alarming rates.

In 1998, the CMAJ published Clinical Practice Guidelines For The Management of Diabetes in Canada[5] The document included a set of 93 recommendations for screening and management of all types of diabetes (type 1, gestational and type 2). The Canadian guidelines[3] supply caregivers with an excellent summary of the literature as it existed before UKPDS[4] arrived. It clearly sets forth recommendations which are coded in terms of the level of supporting evidence, and it is thoroughly referenced. Despite revision[5] of the recommendations since the publication of UKPDS, an examination of the level of support for their recommendations indicates that about one half are of level D evidence (no evidence beyond expert opinion). Clearly, we have a lot more to learn. The present challenge is to understand and develop a practical approach to type 2 diabetes management.

The UKPDS[4,6,7,8,] studies have been widely seen to prove that tight glycemic control is associated with better outcomes in type 2 diabetics, similar to the findings of the earlier DCCT[9] study regarding type 1 diabetes. This is only partially true. [10]

Specifically the UKPDS demonstrates:

- 10 years of treatment with sulphonylureas or insulin had no effect on macrovascular end points (CVA, MI).[4]
- Metformin alone demonstrated clinically significant macrovascular benefits (a 10% decrease in CVA, MI and a 7% decrease in overall mortality). This effect may be irrespective of its hypoglycemic properties.[6]
- Microvascular complications (nephropathy, neuropathy and retinopathy) were reduced by 3% with tight glycemic control. These effects were seen in ‘surrogate end points’: decreased retinal photocoagulation and albuminuria. There was no effect on visual acuity, blindness or renal failure[4]
- Tight control of blood pressure reduces all macrovascular events and microvascular events,[7] irrespective of the agent used.[8]

In order to incorporate this and other relevant information into our practices, the Society of Rural Physicians of Canada has divided the topic of type 2 diabetes into 14 categories of practical interest to clinicians and of import to diabetic patients: rural population; aboriginal patients; definition; screening; level of glycemic control; home glucose monitoring; risk stratification; hypertension; lipids; nephropathy; eye care; foot care; exercise; therapeutics.

We anticipate that this information will be applied in various ways in different rural settings. It is clear that in many cases the evidence is incomplete and monitoring and treatment decisions will still have to be individualized.

RURAL POPULATIONS

Rural healthcare is provided by 10%[11] of Canada's family physicians and 4% of its specialists[11] for 30% of Canada's population.[12] Rural physicians have a broad scope of practice and may spend much of their time on provision of secondary level medical services (emergency, obstetrics, in-patient care, surgery/anesthesia). One rural community's physician resource plan identified 32% of a physician's workload involving primary care.[13] The reality of the shortage of rural physicians is illustrated by a recent Ontario survey which noted 26% of rural patients[14] had trouble accessing family doctors.

Twenty two percent of Canadians live in small communities (<10,000).[15] In general, people in rural areas are older and have lower economic and educational status than urban Canadians.[12] Rural Aboriginals are younger[12] (by an average of 10 years) and have a shorter life expectancy[12] (by 8 years) than the general population.

With the exception of Aboriginal patients, there is no evidence that the burden of type 2 diabetes in the rural population is greater than in the rest of Canada, but certainly the resources for managing it are fewer. Ancillary services, common in urban areas are scarce in rural areas: dieticians, nurse educators, diabetes education programs, primary care physicians and specialist backup are all at a premium.

ABORIGINAL PATIENTS

Aboriginal communities (First Nations, Metis, Inuit) have prevalence rates for type 2 diabetes approaching 30%.[1] Since 56% of Canada's Aboriginal people live in rural areas[12] (35% reserves; 20% in non reserve rural areas), type 2 diabetes is of special interest to rural physicians.

The common theories advanced for this prevalence include lifestyle and dietary changes.[16,17,18] Over the past decades the hunting economy with cycles of feast and famine has eroded. 'Store-bought' foods which are high in carbohydrates and low in fibre have replaced a more traditional diet. This, combined with a more sedentary lifestyle, explains some of the picture.

Community-based genetic research[19] is beginning to outline the genetic predisposition some First Nations have for type 2 diabetes. This includes a genetic association for developing the disease:[19] associated dyslipidemias[20] and susceptibility to renal complications.[21] Several distinct genetic loci on chromosomes 6,8,16 and 22 may be involved.[19]

Not only is the Aboriginal population of Canada much younger than the general population,[22] but rural physicians working in First Nations areas encounter patients with diabetes at an increasingly early age and age-adjusted epidemiology studies bear this out.[16,23] Physicians move quickly through treatment regimes to maximal therapy. In populations of such high prevalence, diabetes presents in diverse ways, and almost any clinical situation is an opportunity for screening.

Since many Aboriginal type 2 diabetics develop truncal obesity, the UKPDS made an important contribution, by identifying the clear benefits of starting obese patients on Metformin, as drug of first choice.[6]

DEFINITION

Type 2 diabetes is a disease of carbohydrate metabolism involving insulin resistance and eventual pancreatic beta cell exhaustion. The disease is a process along a continuum and the defining line has recently been re-drawn . We have a new definition of type 2 diabetes[3], which does not require an oral glucose tolerance test (OGTT) (see Flow Chart)

Fasting levels of ≥7.0 mmol/L are now diagnostic of type 2 diabetes, as are random levels ≥11.1 mmo1/L. A confirmatory test should be undertaken, and it may be a repeat fasting test, a 2 hour 75 gram OGTT(≥11.1 mmo1/L), or even a high random glucose (≥11.1 mmol/L). Severe hyperglycemia does not require repeat testing.

The change in definition allows diagnosis with fasting testing alone. This reflects evidence that microvascular disease (albuminuria, retinopathy) is associated with lower levels of fasting glucose than previously assumed.[24] A fasting level of ≥7.0 mmol/L also correlates well with an abnormal 2 hour OGTT level.[25] These new diagnostic criteria are more likely to capture middle aged and obese patients than older diagnostic levels.[25]

Since type 2 diabetes is sometimes managed with insulin, the older terms of NIDDM (type 2) and IDDM (type 1) have fallen into disrepute. Type 1 diabetes refers to islet cell destruction and a tendency toward ketoacidosis.

SCREENING

Canadian consensus guidelines[3] recommend initial screening at age 45. Further screening is recommended every three years.[3] While there is little evidence to support this,[26] the recommendation recognizes population-based prevalence data suggesting that in the age range of 40-49, prevalence rates increase dramatically.[3] Additionally, 50% of patients have some diabetic tissue damage at the time of diagnosis[27,28] suggesting prolonged pre-existing disease. In areas of high prevalence, or in high risk vasculopathic patients, earlier screening appears to make good sense.

LEVEL OF GLYCEMIC CONTROL

The UKPDS[4] looked at intensive blood glucose control with sulphonylureas or insulin compared with conventional treatment(diet, exercise) and risk of complication in patients with type 2 diabetes. After ten years, of intensive treatment (average HbA1c = 0.07) vs. conventional treatment (average HbA1c = 0.079) there were no statistically significant differences in endpoints in: fatal or non-fatal MI, sudden death, CHF, angina, fatal or non-fatal stroke, death or amputation from peripheral vascular disease, renal failure or death from diabetes or other causes. Myocardial infarctions (classed aggregately as fatal MI, plus non-fatal MI, plus sudden death) came close to statistical significance (p= .052). There were important microvascular disease endpoints: statistically significant (p= .O03) differences in the rate of retinal photocoagulation favouring the intensive treatment groups.

Side effect rates in UKPDS[4] were described as events of major hypoglycemia per year; the rates were: 0.7% with conventional treatment, 1.0% with chlorpropramide, 1.4% with glibenclamide, and 1.8% with insulin. Thus the 10 year rates would be: 7% with conventional, 10%, 14% with sulphonylureas, and 18% with insulin. For chlorpropramide, the Absolute Risk would be 0.03 (3%), the Relative Risk increase would be 30% and the Number Needed to Harm would be 33.3. For insulin, the Absolute Risk increase would be 0.11 (11%), the Relative Risk increase would be 60% and the Number Needed to Harm would be 9.

In summary, UKPDS[4] data demonstrates that 10 years of intensive treatment with sulphonylureas and or insulin would:

- result in HbA1c reduction from. 0.079 to 0.070
- result in reduced microvascular complications (mostly retinal photocoagulation, 2.7%) with an absolute risk reduction of 2% and a relative risk reduction of 22%. To prevent one case of microvascular disease, 42 patients will require intensive treatment for 10 years.
- result in major episodes of hypoglycemia with the number needed to harm ranging from 9-33, depending on the medication used.
- presents no definite evidence that intensive treatment will result in reduced macrovascular endpoints.

How do we balance the benefits and the harm? This is a clinical decision. For each patient saved from retinal photocoagulation, 1-3 patients will suffer major hypoglycemic reactions. In contrast, we shall see that intensive blood pressure control has greater benefits with reduced risks.[7] It is reasonable to aim for HbA1c's < 0.080, and excellent blood pressure control. Tighter glycemic control may benefit patients where hypoglycemic reactions are less of a concern (i.e. diet controlled, Metformin).

HOME GLUCOSE MONITORING

Despite the widespread use of home-glucometers there is scant evidence that their use contributes to metabolic control or clinical outcome. One study[29] showed no difference between home urine versus fingertip monitoring in body weight, HbAlc, or fasting plasma glucose; another[30] reviewed 20 years of RCT's and found questionable evidence. A third study[31] found better glycemic control was associated with home glucometer use, only if accompanied by an intensive 5 day teaching program.

Canadian guidelines recommend use of home monitoring, particularly if insulin or oral hypoglycemics are used. If surveillance for hypoglycemic events is not an issue, a HcAlc every 3 months may suffice. A table correlating the 2 sets of values is enclosed. (see Flow Chart)

RISK STRATIFICATION

Risk stratification may be useful in clinical prediction, management and patient education. As evidence develops, we may use risk stratification to target interventions for high risk diabetic sub-groups and avoid them in low risk groups where harm might outweigh benefit.

Risk stratification[32,33] in type 2 diabetes demonstrates that:

- there is evidence that the more risk factors a diabetic has the greater the mortality risk and the greater the potential benefit from risk reduction.[33]
- microalbuminuria is a strong predictor for mortality in type 2 diabetes, with an odds ratio of approximately 2.4 for all causes of death.[34]
- there is evidence that smoking cessation and hypertension control in diabetics have the greatest potential for impact on mortality.[7, 35, 36, 37]
- as UKPDS[4] showed little mortality benefit from glycemic control, other risk factor modification became far more important.[4]

HYPERTENSION

For most type 2 diabetics adverse outcomes are macrovascular events (CVA, MI)[4] and control of blood pressure has the greatest impact[7] on these events. Fifty percent of newly diagnosed type 2 diabetics[7] will be hypertensive at diagnosis and suffer[38] an increased risk of cardiovascular morbidity and mortality: twofold increase for men and a fourfold increase for women.

One of the UKPDS studies[7] followed over a 1000 patients for 8 years and assigned them to ‘tight' blood pressure (mean 144/82) or ‘less tight' control (mean 154/87). The tight control group had a reduction in risk of diabetic related deaths of 32%, stroke 44% and microvascular complication of 37%. The patients who did the best had systolic pressures under 125 mmHg, and tight blood pressure control often (29%) required 3 or more antihypertensive medications.

In the UKPDS, diuretics, ACE inhibitors, alpha blockers, beta blockers and calcium channel blockers were used.[7, 39]

Other studies[40, 41] have shown that systolic pressures as low as 120 mmHg decrease cardiovascular risk, as do diastolic pressures below 80mm Hg.[42,43] While 140/90 mm Hg is the treatment goal for hypertension, the evidence would suggest we should be aiming for 130/80 mmHg in our type 2 diabetic patients.

LIPIDS

The science of lipid management and screening has changed a great deal over the past 10 years. When Framingham data was presented a decade ago, total cholesterol was the sole indicator of cardiac risk. Subsequent studies have focused on low-density lipoprotein cholesterol (LDL-C, previously called LDL) rather than total cholesterol as harbouring atherosclerotic risk. This laboratory-calculated value (HDL plus TG/2.2 are subtracted from Total Cholesterol), has been identified as the strongest predictor of heart disease by one of the UKPDS studies.[44]

The dyslipidemia profile in type 2 diabetes is often characterized[45, 46] by higher triglicerides, and lower HDL levels. Elevated trigliceride levels seem to affect the structure of LDL-C, by rendering it smaller, more dense and perhaps more atherogenic,[46] although elevated TGs are not

yet identified as an independent risk factor. Both elevated TG and HDL levels are independent of glycemic control, whereas total cholesterol and LDL-C levels may normalize with adequate glycemic control.[47]

Five major trials[44,48, 49, 50, 51] studied dyslipidemia, cardiac risk and type 2 diabetes. The results showed statistically significant primary prevention[51] (decreased onset of CAD) and secondary prevention[49,50] (decreased cardiac events in known CAD patients) when cholesterol levels were normalized, particularly LDL-C in type 2 diabetic patients.

The recently revised Canadian guidelines[52] consider diabetics over age 30 as very high risk for CAD and recommend an LDL-C level of less than 2.5 mmo1/L. The recommended class of drug for typical type 2 diabetic dyslipidemia (high LDL-C) are the statins.[52] Fibrates are useful for increased TG and low HDL-C dyslipidemias.[52]

NEPHROPATHY

The development of diabetic nephropathy is most effectively reduced by treatment of hypertension[7, 40, 53] and less so by glycemic control.[4] Surveillance for microalbuminuria is the initial step in following diabetic nephropathy, the leading cause of end stage renal failure. Microalbuminuria (30 - 300mg/day) refers to a quantity of proteinuria too minute to be picked up by traditional protein dipsticks (proteinuria, > 300mg/day).

If a type 2 diabetic patient screens positive on a simple protein office dipstick (>1 plus), the patient already has proteinuria (referred to as overt nephropathy) and the confirming and quantifying test would be an initial 24 hour urine collection for protein.

A patient with a negative protein dipstick needs assessment for microalbuminuria, by mau dipstick, or a single lab urine measurement. A spot urine for albumin/creatinine ratio is an accurate confirmatory test for microalbuminuria, which corrects for urine concentration.[3, 54, 55] The level of microalbuminuria is generally provided with the ratio. Once the level of protein is greater than 300mg/day, a 24 hour protein collection replaces albumin/creatinine ratio as the annual quantifying test.

Increasing spilling of protein indicates disease progression and the need for more aggressive blood pressure and glycemic control where possible. Microalbuminuria is followed annually by spot urine for albumin/creatinine ratio; while gross proteinuria is monitored by annual 24 hour urine protein collections (bear in mind patient compliance is poor and there may be a normal 20% daily variation in 24 hour urine protein excretion).

Microalbuminuria correlates highly as a predictor of cardiac disease in type 2 diabetics.[34, 56] It is thought to be a marker of endothelial damage, which affects both micro and macro sized vessels. ACE inhibition has been documented[55, 57, 58,59, 60] to slow the progression from microalbuminuria to proteinuria, to end stage renal failure.

The initial ACE inhibitor studied after Captopril[58] was Enalapril[60], at a nephro-protective dose of 10 mg/day. Subsequent studies have found positive effects with Fosinopril,[61] Lisinopril[56] at the same dose, and Ramipril at 2.5 mg/day.[62] Angiotensin II receptor blockade also seem to be nephro-protective[56, 63, 64] as does beta blockade.[7]

Current Canadian guidelines suggest a nephrology referral based on serum creatinine levels of >300 μmol/L.[65] Serum creatinine level should be routinely checked upon initiation and two weeks following ACE inhibitor therapy, and annual checks seem prudent.

EYE CARE

Vision loss occurs by two mechanisms[3] in type 2 diabetes: proliferative retinopathy (3- 14%) and macular edema (4-15%). UKPDS[4] showed that tight glycemic control had no effect on visual acuity or vision loss. It did decrease the need for photocoagulation, as did diastolic blood pressure <90 mm Hg.[7] Diabetic retinopathy is often present at the time of diagnosis, hence screening should begin then and at least every 2 years thereafter.[3] General practitioners are often uncomfortable with such retinal exams[66] but in rural areas they may have to take on that task,[67] or more commonly rely on local optometry expertise, or regional ophthalmology services.

FOOT CARE

Over a 15 year period, up to 60% type 2 diabetics will develop a peripheral neuropathy.[4] Poor glycemic control is associated with progression[68] and simple office screening methods may be predictive of complications[69, 70] and may be protective.[70, 71, 72, 73] Lower limb amputation might be reduced by as much as 85%[71] with these methods.

As peripheral neuropathy develops and sensation declines, so too will self-reported symptoms. Current recommendations are for annual sensory extremity exams[3] and insensate patients will require more frequent assessment. Such high risk patients should be flagged for increased surveillance. (see Flow Chart)

Three simple methods of sensory testing have correlated well[74] with more complex nerve conduction studies:

Neuropathy is assumed to be present if 50% of responses are incorrect:

1) 10 gram monofilament (similar to nylon fishing line) applied 4 times to dorsum of great toe, just proximal to toe nail, or
2) on-off 128 Hz vibration, applied 2 times to same area of great toes, or
3) pin prick to same areas 4 times.

Complications of such neuropathies requires aggressive management of diabetic ulcers and peripheral vascular disease, including debridement and antibiotic therapy as required.

EXERCISE

Exercise is a key component of therapy for type 2 diabetes. It proffers glycemic control though improved insulin sensitivity and carbohydrate metabolism; facilitates weight loss; decreases hypertriglyceridemia and improves hypertension and hyperinsulimenia.[75]

Type 2 patients on insulin or sulphonylureas may experience hypoglycemia and those who exercise intensely on Metformin can experience severe lactic acidosis, making this drug undesirable in more elite athletes.[76]

A typical exercise prescription should include: a 5 to 10 minute period of warm up and cool down after exercise; a period of stretching after warming up and then exercise in the range of 50-70% VO2 max for 20-45 minutes at least 3, but preferably 5 to 6 times per week.[77] This level of exercise is one in which the participant is able to carry on a conversation, but will be puffing between sentences. Physicians should be cautious against being too prescriptive, as no studies yet define the ideal exercise program, and gains are seen in type 2 diabetes even with modest activity.[76,78, 79, 80]

Risks for exercising diabetics include worsening of retinopathy with overly vigorous activity, cardiac complications such as ischemia, silent ischemia, hypotension, increased albumin excretion (of unknown significance), foot damage from nephropathy and dehydration and thermoregulatory problems from autonomic neuropathy.[81]

THERAPEUTICS

The cornerstone of clinical management of type 2 diabetes is an emphasis on and frequent return to the basics of self-management:

- healthy eating
- daily exercise
- smoking cessation

The prospects for successful subsequent medical management are compromised if not built on a firm foundation of patient self-management. When this fails and medical management is required, clinicians are faced with some choices. The choices begin with the use of a single oral hypoglycemic. If control remains unacceptable with a single agent, one may:

- add a second and possibly a third or fourth oral agent
- add an evening NPH insulin dose to the sole or multiple oral agent
- switch to insulin, 2 to 4 times daily

In the UKPDS[4] study, insulin replaced oral agents when it was introduced, so decisions about combined therapies lead us onto new ground, without long term evidence. UKPDS[4] did identify the independent safety of glyburide or Metformin or insulin over extended periods of time.

Newer agents will initially be judged by their cost and their ability to achieve glycemic control,[82, 83] but their long term safety and efficacy will be judged on morbidity and mortality rates - information we do not presently have. What follows is a discussion on best evidence to date regarding therapeutic options, combining proven older medications, and promising newer ones. Clinical experience, patient care issues and side effect profiles will all play a role in arriving at the best possible care for a given patient with type 2 diabetes.

Metformin (Glucophage) is clearly[6] the first-line drug in the obese type 2 diabetic patient, when self-management fails to achieve adequate glycemic control. The UKPDS demonstrated it to be superior to insulin or sulphonylureas in mortality reduction and diabetic-related end points. Interestingly, it did this despite often equivalent reduction in HbAlc to the other medications. Metformin rarely causes hypoglycemia, a significant advantage over other standard agents, and causes less weight gain. GI side effects (nausea and bloating) are lessened by beginning with 250 mg/day and gradually increasing to a daily maximum of 2.5 grams. It is contraindicated in mild renal failure (creatinine >140 mol/L) and hepatic dysfunction due to risk of lactic acidosis.

Glyburide (Diabeta) a second generation sulphonylurea is a first line agent of choice in some patients (i.e. lean type 2 diabetics). Weight gain (average 3.1 kg) and hypoglycemic episodes complicates the use of these agents. The starting dose is 2.5 mg. up to 10 mg. bid. As these agents promote insulin secretion by a direct action on pancreatic beta cells, they are prone to secondary failure, thought to be due to beta cell exhaustion. Recent studies have demonstrated the safety and efficacy of glyburide[84] in second and third trimester pregnancies for control of gestational diabetes.

Repaglinide (Gluconorm) is similar to glyburide, but shorter acting and taken only before meals and is less likely to cause hypoglycemic events. It is ineffective if the traditional 'insulin-secretalogue', glyburide, has not worked. Dose is 0.5 - 4 mg, 15-30 minutes AC meals, to a daily maximum of 16 mg.

Thialzolidinediones, the 'glitazones' (Pioglitazone/Actos and Rosiglitazone/Avandia) function by decreasing insulin resistance through a different mechanism than Metformin and can be safely added to it.[82, 83] These agents may have a role in patients with renal failure (a contraindication to Metformin) or those suffering hypoglycemic episodes from the 'insulin-secretalogues' (Glyburide and Repaglinide), an unlikely side effect with this class of drug. Their use is complicated by cost, the need for regular (q 2 monthly) hepatic monitoring and their slow onset of action - up to 12 weeks before considering dosing change. Pioglitazone dose range is 15-45 mg OD. Rosiglitazone is 4-8 mg OD.

Alpha-glucosidase inhibitors (Acarbose) are less potent than the above agents and generally are not indicated in patients with severely compromised glycemic control. These agents do not cause hypoglycemia and are not systemically absorbed. Since they block sucrose absorption, self treatment of hypoglycemic episodes secondary to concurrent use of other agents, requires oral glucose tablets, as oral sucrose intake will be ineffective. Starting dose is 25mg/day to a maximum of 100 mg tid. Titration must be very slow as significant numbers of patients experience GI side effects, and they are contraindicated in chronic gastrointestinal disorders.

Initial first-line medication choices might include Metformin for the obese patient and Glyburide for the lean type 2 diabetic. Some authors include the 'glitazones' at this stage. All 3 use different mechanisms, are additive and can be safely used together.

Insulin therapy may be indicated when self-management and the use of oral agents fail to achieve adequate control. Initially, it may be added to oral agents and may replace them.

Insulin increases appetite with concomitant weight gain and can cause hypoglycemia. It is presently the recommended agent for fertile type 2 diabetic women wishing to become pregnant.

If insulin is added to oral agents, care should be taken for the additive hypoglycemic effects of insulin on glyburide, and this might be the first oral agent to stop once insulin therapy has begun. A nighttime dose of 10-25 units of NPH is reasonable (0.1-0.3 units/kg) with weekly increases of 5 units until control is achieved or 40 units is reached. Higher doses of insulin might best be administered in divided doses, BID - QID, with or without the continuation of Metformin and a 'glitazone'.

Since the natural history of type 2 diabetes is progressive beta-cell impairment, it would not be surprising to encounter a treatment pattern which moves from diet, to oral agents and eventually to insulin use.

One last therapeutic note concerns the routine use of daily ASA. The Canadian and American Diabetes Associations recommend daily ASA for high risk diabetics, now defined as those over 30 years old. This comprises most adult type 2 diabetics, yet few are on aspirin[85] despite a cardiovascular risk equal to that of a non-diabetic patient with a previous myocardial infarction.[45]

CONCLUSION

There is a plethora of data and divergent interpretations concerning type 2 diabetes management since the 1998 release of the UKPDS. Busy clinicians need a simple accurate method for documenting and managing type 2 diabetes and its complications. We need to focus our investigations and intervention efforts where they can be most effective. The numerous laboratory investigations available often arise from their use in a particular study, and are often not designed for primary care patient management.[86] This leaves the clinician with choices, which may vary according to geographic region, laboratory availability and cost. Rural physicians whose practices include a high prevalence of type 2 diabetes require a set of easily monitored laboratory investigations, as this disease will constitute a large part of their workload.

The first change in practice ushered in by UKPDS is tight blood pressure control for type 2 diabetic patients - it may be more important than glycemic control. Obese patients will benefit from Metformin use. Those spilling protein (microalbuminuria or frank proteinuria) in their urine benefit from ACE inhibitors and blood pressure control. Reasonable glycemic control lessens microvascular complications (retinopathy, nephropathy, neuropathy) with little downside; tight glycemic control improves these parameters, with some risk of hypoglycemic episodes.

The enclosed chart incorporates many of the recent developments in consensus and evidence in type 2 diabetes. The field will be changing as further primary and secondary prevention studies are underway.

Presently, a type 2 diabetic would do well to leave the office with:

- lifestyle management encouragement
- optimal blood pressure control
- HbA1C q 3/12
- Yearly lipid profile, urine microalbuminuria screen and sensory exam of the foot.

Their treatment regime will likely include, Metformin, a statin, an ACE inhibitor and ASA. Patients may well progress to second oral agents and/or nighttime NPH insulin or multiple insulin doses- all of which fall under the purvey of rural family physicians.

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Name _____

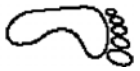
D.O.B. _____ Allergy _____

HC# _____ Date of Dx _____

Phone# (H) _____ (W) _____

Society of Rural Physicians of Canada Type 2 Diabetic Flow Chart *2004*

- ☐ Retinopathy
- ☐ Proteinuria-Microalbuminuria
- ☐ CAD
- ☐ B.P.↑
- ☐ Smokes
- ☐ Impotence
- ☐ Fam Hx CAD
- ☐ Chol ↑



Colour foot if high risk

Eye Exam

Date												
Weight/BMI												
B.P. (130 / 80)												
* A1C (< 0.07)												
LDL (< 2.5)												
Chol/HDL (< 4.0)												
TG (< 2.0)												
Creatinine (1-133)												
or Alb/cr (M<2.8 F <2.0) 24 hr protein (1x yr)												
Exercise x / wk.												
Foot exam ? done												
Medications												
Metformin												
Glyburide												
…glitazone												
Repaglinide												
Insulin												
Statin/Fibrate												
ACE/ARB												
ASA (> 30y.o.)												
.....												

Society of Rural Physicians of Canada Type 2 Diabetic Flow Chart *2004*

DIAGNOSTIC CRITERIA FOR DIABETES

A confirmation test must be done on another day unless severe hyperglycemia

Type 1 and Type 2 DM
Random ≥ 11.1mmol/L
OR
Fasting ≥ 7.0mmol/L
OR
75gm 2h ≥ 11.1mmol/L

Getstational DM screening (24-28/52)
non-fasting 1 hr 50 gm: ≥ 10.3 is diagnostic;
7.8 - 10.3 do confirmatory fasting 75 gm 2 hr:
two out of three: fasting ≥ 5.3 *or* 1 hour ≥ 10.6 *or* 2 hour ≥ 8.9

A1C - AVERAGE GLUCOSE

Glycosylated Hemoglobin	Average blood glucose in last 3 mo.
0.06	6
0.065	7
0.07	8
0.075	9
0.08	10
0.085	11
0.09	12
0.095	13
0.1	14
0.105	15
0.110	16
0.115	17
0.120	18
0.125	19
0.130	20
0.135	21
0.140	22

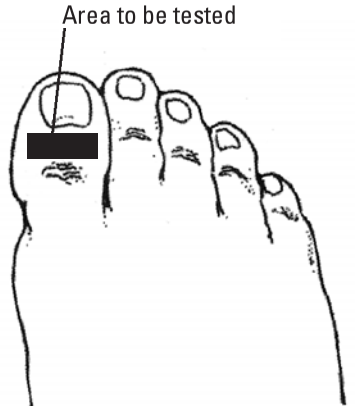
SCREENING SENSORY FOOT EXAM

Choice of Method

1] 10 gram monofilament x 4
2] Pin prick x 4
3] ON-OFF 128 Hz x 2

Score 2 out of 4 or less = neuropathy
Colour foot on top of flow chart solid colour to indicate high risk

Once neuropathy established, discontinue this testing and do regular foot exams on this high risk patient to screen for ulcers and infections.



Available at www.srpc.ca

LIPID VALUES *Targets based on 10 year risk of CVD event*

	Target Values LDL-C (mmol/L)	TC/HDL Ratio	TG (mmol/L)
Very high risk 10 year risk> 10% on history of CVD or Diabetes Mellitus	<2.5	<4	<2.0

For complete risk assessment, see CMAJ 2000; 162 (10): 1441-7

MANAGEMENT APPROACH

Step 1. Diet and excercise
Step 2. Oral agent: Metformin if obese; or Glyburide; or …glitazone
Step 3. Combine two or three oral agents
Step 4. Add NPH Insulin, QHS, 10 - 20 units
Step 5. BID-QID Insulin alone, or with metaformin / …glitazone

* GLYCEMIC CONTROL

CDA recommends A1c <0.07; UKPDS notes ↑ hypoglycemia (up to 18%) with such tight control. Consider A1C level of 0.07-0.08 in the frail elderly, or if using meds causing hypoglycemia (insulin, glyburide).

Notes

The chart is designed for a 3 year period, but can be used flexibly for any time frame. Not every clinic appointment need be recorded, nor all of the patients medications. Once peripheral neuropathy is documented, please fill in the top foot in a solid colour, so it acts as a reminder that this patient is at high risk for peripheral complications·

Updated from: Kelly L, Roedde S, Harris S, Kapasi H, Bozek N, Baechler M, Wilms L, Kaiser J, Sehgal Y, Hyder B. *Society of Rural Physicians of Canada Evidenced-based Practical Management of Type 2 Diabetes. CJRM 2001; 8 (1) insert*

Nom _____

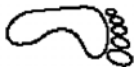
Naissance. / / Allergies _____

dossier _____ Date du Dx _____

Téléphone (R) _____ (T) _____

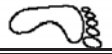
La Société de la médecine rurale du Canada-Feuille sommaire diabète Type 2, 2004

- ❑ Rétinopathie
- ❑ Protéinurie-Microalbuminurie
- ❑ MCAS
- ❑ Hypertension ↑
- ❑ Tabagisme
- ❑ Dysfonction érectile
- ❑ Hx Fam MCAS
- ❑ Chol ↑



Colorier si haut risque

Examamen de la vue

Date												
Poids (IMC)												
T.A. (130 / 80)												
* Hb A1C (< 0.07)												
LDL (< 2.5)												
Chol/HDL (< 4.0)												
TG (< 2.0)												
Créatinine(1-133)												
ou Alb/cr (M<2.8 F <2.0) 24h protéine (1 x/an)												
Exercice x / sem.												
Examen du pied- ? fait												
Médicaments												
Metformin												
Gluburide												
… glitazone												
Repaglinide												
Insuline												
Statine/Fibrate												
ieca (ARA)												
ASA (> 30 ans)												
.....												

La Société de la médecine rurale du Canada - Feuille sommaire diabète Type 2 2004

CRITÈRES DE DIAGNOSTIC DU DIABÈTE

Un test de confirmation doit être fait un autre jour à moins d'une élévation marquée

Diabète Type1+2
randomisé ≥ 11.1mmol/L
OU
A jeûn ≥ 7.0mmol/L
OU
75gm 2hs ≥ 11.1mmol/L

Diabète gestationnel: dépistage (24-28/52)
1 heure p.c 50 gm ≥ 10.3 est diagnostique;
7.8 - 10.3 confirmer À jeûn 75 gm 2 heures:
2 de 3: A jeûn ≥ 5.3 ou 1 heure ≥ 10.6
ou 2 heures ≥ 8.9

A1C - GLUCOSE MOYEN

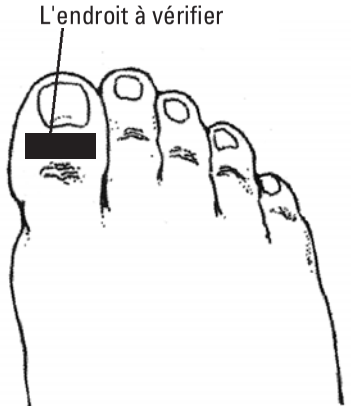
	Hémoglobine Glycosylée	Glucose moyen pour les derniers 3 mois
*	0.06	6
	0.065	7
*	0.07	8
	0.075	9
	0.08	10
	0.085	11
	0.09	12
	0.095	13
	0.1	14
	0.105	15
	0.110	16
	0.115	17
	0.120	18
	0.125	19
	0.130	20
	0.135	21
	0.140	22

DÉPISTAGE: EXAMEN SENSORIEL DES PIEDS

Choix de méthode
1] monofilament 10gm x 4
2] Aiguille x 4
3] 128 Hz intermittent x 2

pointage 2 / 4 ou moins = neuropathie
Colorier pied d'une couleur solide
pour indiquer un haut risque

Une fois la neuropathie établie discontinuer cette méthode et faire un examen régulier des pieds chez ce patient à haut risque pour dépister les ulcères et les infections.



Disponible à www.srpc.ca

VALEURS CIBLES DES LIPIDES sur le risque à 10 ans d'évé cardiovasculaires

	Valeurs cibles LDL-C (mmol/L)	TC/HDL Ratio	TG (mmol/L)
Très haut risque 10 ans > 10% histoire de MCV ou diabète	<2.5	<4	<2.0

Pour une évaluation du risque, consultez CMAJ 2000; 162 (10): 1441-7

APPROCHE / GESTION

Étape 1. diète et exercice
Étape 2. médicament orale: Metphormin si obèse; ou Gluburide; ou...glitazone
Étape 3. combinaison de 2 ou 3 médicaments oraux
Étape 4. ajouter Insulin NPH, QHS, 10 - 20 unités
Step 5. BID-QID Insulin seule, ou avec metaformin / …glitazone

CONTRÔLE GLYCÉMIQUE

ADC recommende A1C <0.07; UKPDS note ↑ hypoglycémie de (18%) avec un contrôle serré. Viser un A1C à 0.07 - 0.08 chez la personne âgée fragilisée, ou si les médicaments pouvant causer l'hypoglycémie sont utilisés (insuline, glyburide).

NOTES

La tableau permet un suivi de 3 ans, mais peut être utilisé pour une période de temps variable. Il n'est pas nécessaire d'y inscrire tous les Il n'est pas nécessaire d'y inscrire tous les compte-rendus des visites ni tous les médicaments du patient. Une fois le diagnostic de neuropathie périphérique établi, colorier le pied en une couleur, témoignant que le patient est à haut risque de complications périphériques.

Mise à jour de - Kelly L, Roedde S, Harris S, Kapasi H, Bozek N, Baechler M, Wilms L, Kaiser J, Sehgal Y, Hyder B. *Society of Rural Physicians of Canada Evidenced-based Practical Management of Type 2 Diabetes. CJRM 2001; 8 (1) insert*