Alcohol and Liver Disease

“I have drunk since I was fifteen and few things have given me more pleasure. Modern life is often a mechanical oppression and liquor is the only mechanical relief…”

Ernest Hemingway

When Hemmingway took his own life in 1961, his long and abusive relationship with alcohol was most certainly a contributing factor. Hemmingway’s life and his quote above do encapsulate many of contributory risk factors that those who eventually develop alcoholic liver disease exhibit; they tend to be older lives, they are predominantly male, and have drunk alcohol in high volumes over many years. In conjunction with these factors, the disease often is strongly associated with depression, with individuals ‘self-medicating’ with alcohol to cope with loneliness and mental ill-health.

Humanity’s relationship with alcohol goes back millennia; indeed some archaeologists suggest beer was a happy side effect of bread making. The medicinal or health related reasons for drinking alcohol go back just as far, and for much of history imbibing distilled or brewed liquid was much safer than drinking water. Today, it has been scientifically demonstrated that moderate drinking, particularly of red wine, the source of the so called ‘French Paradox’, has a number of health benefits. For example the reduction of risk from cardiovascular disease, is beneficial to joint health and reducing risk of gall stone formation.

That said, alcohol is toxic, and causes acute and chronic damage - of all drugs used by humans, scientists rank alcohol first in causing most harm physically, psychologically and socially.

Health and mortality impact

If we examine the top causes of death in the UK for adults aged 15-64, five reasons dominate: cancer (42%), cerebrovascular/ischaemic heart disease (20%), respiratory disease (7%), accidents (11%) and liver disease (6%) .

3 Artero, A. The impact of moderate wine consumption on health, *Maturias* Vol 80, issue 1 Jan 2015, pp3-13
4 Nutt, D. et al Drug harms in the UK: a multicriteria decision analysis
5 ONS, Mortality Statistics 2012
Excess alcohol contributes directly to the latter two and indirectly to the others. For example, continuous misuse of alcohol increases blood pressure in the long term and this is one of the contributory factors in coronary and cerebrovascular disease. In terms of carcinogenesis alcohol damages cells, it increases the permeability of cells to toxins like tobacco, affects hormones (particularly those associated with breast cancer) and when metabolised it produces cancer inducing chemicals. As a result alcohol contributes significantly to the incidence of at least seven types of cancer.

The liver and alcoholic liver disease
The liver plays an essential role in ridding the body of poisons and toxins, both highly present in alcohol, and it is therefore unsurprising that this organ is one most directly affected by abuse. Around 4,500 people annually die of alcoholic liver disease, an increase of 400% since 1970 and 40% in only the last 12 years. When compared to other causes of death these increases are stark (see figure 1).

The liver is damaged by alcohol in two ways as it metabolises: toxins are produced which damage the hepatocytes (liver cells), and simultaneously make them inflamed. Over time individuals can develop chronic inflammation (hepatitis) and/or fatty liver - at this point damage is reversible. However, once cirrhosis (or end stage liver failure) is established the liver is irreversibly damaged and scarred, with half of all those reaching this stage dying within 5 years.

While cirrhosis can be caused by a number of things, such as auto-immune disease, it is estimated 60-70% of cases are triggered by alcohol.

Studies show that of all heavy drinkers (those consuming 10 units or more per day) 10% develop cirrhosis after 10 years and further 20% after 20. Quite why some develop cirrhosis and others do not is unknown, but clearly the volume and regularity of alcohol consumption is key. On average the consumption of 600kg of pure alcohol over 20 years will result in cirrhosis in most individuals.

How can we assess and identify alcohol misuse?
Clearly with these emerging trends and risks it is essential insurers have tools in place to screen and pick out applicants who have an increased risk of developing alcoholic liver disease. But, identifying those individuals is one of the most problematic issues in underwriting. We know from the UK Governments annual General Lifestyle survey that 5-9% of the population drink heavily (8-12 units in any one day) and a further 6-9% drink very heavily (>12 units in any one day), yet, less than 1% of applicants for life insurance would admit to it.

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6 Cancer Research UK


8 Ibid.
This ‘risk gap’ is already rightly causing concern and has resulted in many providers looking at better ways to elicit more truthful responses to their application questions. For example, moving away from asking about ‘units’ and using ‘how many drinks’ people consume, asking when people drink and how often, or whether they have been told to cut back or have been referred for counselling about their drinking. However, given the stigma associated with heavy drinking and the lack of insight people have about their habits, asking questions on consumption can only get underwriters so far. As Professor Mark Jayne of Cardiff University stated:

“It is virtually impossible to assess accurately how much alcohol someone really drinks. Questionnaires tend to be useless in this respect as the information people give on their alcohol intake is unreliable...”

However underwriters do have an arsenal of tests to draw upon, notably the liver function tests (LFTs - see box1). These are either used over certain sum assured limits or if alcohol misuse is suspected. These are not strictly tests of liver function but instead are markers for acute or chronic disease or damage to the liver (ALT, AST, and GGT) and bile ducts (GGT, AP). GGT is perhaps the most important indicator of risk particularly if serially elevated. For example individuals with GGT 2-5x higher than normal have an increased risk of death some 2-3x higher than those with normal LFTs.

However, the main problem with these blood tests is that they lack sensitivity and specificity particularly in relation to alcohol as the cause of the elevations are numerous and as such cannot be relied on to make diagnoses let alone prognoses. Other tests, such as EtG and blood alcohol (used in drink driving test), have limited application in the insurance sphere, as they only demonstrate if alcohol has been consumed recently, they cannot determine however if that consumption is a one off or part of a more chronic lifestyle problem.

Box 1
Biochemical/Haematological makers of Liver Disease
- Aspartate aminotransferase (AST, once known as SGOT)
- Alanine aminotransferase (ALT, once known as SGPT)
- Gamma-glutamyl transpeptidase (Gamma GT, GGT)
- Alkaline phosphatase (AP)
- Carbohydrate deficient transferrin – CDT
- EtG – ethyl glucuronide – 5 day half
- Blood alcohol short half life – sign of acute alcohol intake

In the USA, the use of Carbohydrate deficient transferrin in combination with other LFTs to get a more accurate alcohol screening has become common place. As box 2 illustrates CDT along with GGT provides much more precision in detecting alcohol abuse with sensitivity and specificity of >90%.

Box 2

<table>
<thead>
<tr>
<th>Biomaker</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>AST</td>
<td>15-69</td>
<td>47-68</td>
</tr>
<tr>
<td>ALT</td>
<td>18-58</td>
<td>50-57</td>
</tr>
<tr>
<td>GGT</td>
<td>34-38</td>
<td>11-95</td>
</tr>
<tr>
<td>MCV</td>
<td>34-89</td>
<td>26-95</td>
</tr>
<tr>
<td>CDT</td>
<td>39-94</td>
<td>82-100</td>
</tr>
<tr>
<td>CDT + GGT</td>
<td>90+</td>
<td>98</td>
</tr>
<tr>
<td>Alcohol</td>
<td>0-100</td>
<td>0-100</td>
</tr>
<tr>
<td>EtG</td>
<td>76-91</td>
<td>77-92</td>
</tr>
</tbody>
</table>

* Values vary considerably according to gender, age, drinking pattern, prevalence of alcohol abuse/ dependence, and prevalence of comorbidity, among other factors [39,6,40,42,43]

Sensitive - ability to test to identify those who have a trait - those who test positive really are positive

Specificity - ability to test to identify those who do not have a trait - those who test negative are really negative
Future approach
So what of the future? Trends indicate that people’s drinking problems, and the life insurance industry’s consequent challenges will continue to be an issue for some time to come. While we must always ask good clear questions of individuals about their habits and lifestyle, given the common generalised ‘amnesia’ associated with alcohol it seems we need to do better to screen or profile individuals who are more likely to have an issue with alcohol. For example, in a study of the Longitudinal survey of ageing, a number of key markers of problem drinking were identified:

- Older age (over 50s)
- Smoking, and particularly the number of cigarettes
- Single, divorced or widowed men
- Higher educational attainment
- Higher income was associated with a higher probability of problem drinking in women but not men
- Self-reported feelings of loneliness
- Poor diet

In order to achieve this we do not necessarily need to reach for ‘big data’ solutions, but rather use the data we already have and add or adjust the questions we utilise in our existing underwriting engines.

At Hannover Re UK Life Branch we are already working on these ideas, looking to harmoniously implement and combine our evidence based research with risk based product and customer journey design.

Paul Edwards
Manager, Medical Risk Research
paul.edwards@hannover-re.com

How to contact us:
We hope you enjoy infocus and we welcome your feedback, please forward comments to Alessandra Pierandrei at uk.marketing@hannover-re.com.

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