Jaundice as a presentation of heart failure

R van Lingen MRCP² U Warshaw MRCP¹ H R Dalton DPhil FRCP¹ S H Hussaini MD FRCP¹

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SUMMARY

On rare occasions the first manifestation of heart disease is jaundice, caused by passive congestion of the liver or acute ischaemic hepatitis. We looked for this presentation retrospectively in 661 patients referred over fifty-six months to a 'jaundice hotline' (rapid access) service. The protocol included a full clinical history, examination and abdominal ultrasound. Those with no evidence of biliary obstruction had a non-invasive liver screen for parenchymal liver disease and those with suspected heart disease had an electrocardiogram, chest X-ray and echocardiogram.

8 patients (1.2%), bilirubin 31–79 $\mu\text{mol/L}$, mean 46 $\mu\text{mol/L}$, had a primary cardiac cause for their jaundice. All had dyspnoea, an increased cardiothoracic ratio on chest X-ray and an abnormal electrocardiogram. The jugular venous pressure was raised in the 3 in whom it was recorded. In 6 patients the jaundice was attributed to hepatic congestion and in 2 to ischaemic hepatitis. All patients had severe cardiac dysfunction.

Jaundice due to heart disease tends to be mild, and a key feature is breathlessness. The most common mechanism is hepatic venous congestion; ischaemic hepatitis is suggested by a high aminotransferase.

INTRODUCTION

Jaundice is an uncommon presentation of cardiac disease.^{1–3} The two major causes are chronic congestion due to heart failure and ischaemic hepatitis from acute circulatory impairment. We conducted a retrospective review of patients seen at a jaundice hotline service to determine the proportion of such cases and their clinical characteristics.

METHODS

The Royal Cornwall Hospital is a district general hospital serving a population of about 400 000. A hotline service was started in November 1998 to facilitate rapid diagnosis and treatment of patients with jaundice in the community, and the initial results have been reported.⁴ All patients had a full history taken for alcohol use, medications and risk factors for viral hepatitis. An abdominal ultrasound was performed to identify biliary obstruction. In patients without biliary obstruction, blood was tested for evidence of virus infections (hepatitis A, B and C, Epstein–Barr, cytomegalovirus), for autoantibodies and for alpha-1-antitrypsin concentration, together with iron and copper studies. In patients without evidence of biliary obstruction or parenchymal liver disease, cardiac evaluation included an

electrocardiogram (ECG), an echocardiogram and a chest X-ray.

RESULTS

Of 661 patients seen by the jaundice hotline service in fifty-six months 8 (1.2%) had a primary cardiac disorder. All reported dyspnoea. Details are in Table 1. Their jaundice was mild (bilirubin 31–79 $\mu\text{mol/L}$, mean 46 $\mu\text{mol/L}$) and only 2 had an alkaline phosphatase above normal. 2 patients with severe cardiac failure and an alanine aminotransferase exceeding 1000 iu/L were judged to have ischaemic hepatitis. Both had a raised troponin, so the probable cause of their cardiac decompensation was myocardial infarction within the last 10 days; their liver function tests became normal with treatment of their heart disease. All patients had abnormal electrocardiograms, and echocardiograms showed severe global or left ventricular impairment, valvular abnormalities and in one case a left atrial myxoma. The clinical assessment of jugular venous pressure was recorded in only 3 of the 8 patients.

DISCUSSION

Among patients presenting via the hotline, heart disease was a rare cause for jaundice. Moreover, the jaundice was always mild. In all 8, the history of dyspnoea together with cardiac enlargement on X-ray and ECG abnormalities pointed to the underlying disorder. The jugular venous pressure, a bedside assessment with diagnostic, therapeutic and prognostic value,⁴ was not well recorded in this series.

¹Cornwall Gastrointestinal Unit, Royal Cornwall Hospital Trust, Truro TR1 3LJ;

²Department of Cardiology, Derriford Hospital, Plymouth PL6 8DH, UK

Correspondence to: Dr Hyder Hussaini

E-mail: hyder.hussaini@rcht.cornwall.nhs.uk

Table 1 Clinical details

Patient (age)	Dyspnoea duration	Bilirubin $\mu\text{mol/L}$ (ref. range 8–23)		ALT IU/L (10–44)	Alk phos IU/L (45–122)	Liver U/S	ECG	CXR	Echo
		JVP							
M (54)	5 months	↑	33	41	99	Enlarged; engorged hepatic veins	AF; partial RBBB	Bilateral pleural effusions. ↑C-th ratio	Left atrial myxoma; dilated RV
M (69)	1 month	NR	51	50	82	Normal	AF; LBBB	Bilateral pleural effusions. ↑C-th ratio	Dilated LV, EF 15%
M (79)	1 month	↑	56	38	57	Dilated IVC and hepatic veins	LBBB	↑C-th ratio	Dilated LV, EF 18%
M (61)	3 months	NR	33	25	111	Mild parenchymal irregularity	AF; LBBB	↑C-th ratio; bibasal shadowing	Dilated LV, EF < 20%
M (72)	> 2 years (known cardiomyopathy)	NR	31	11	272	Normal	AF; LBBB	↑C-th ratio	Dilated LV, EF 23%
F (69)	2 weeks	NR	79	1213	887	Normal	LBBB	↑C-th ratio	Severe LV impairment; severe AS
M (80)	3–4 weeks	NR	32	1085	141	Dilated hepatic veins	AF; partial LBBB	↑C-th ratio	Severe MR; normal LV
M (75)	1 month	↑	55	41	68	Incidental single gallstone	1st degree heart block; LBBB	↑C-th ratio	Severe MR; moderate AS; mild LV impairment

JVP=jugular venous pressure; ALT=alanine aminotransferase; alk phos=alkaline phosphatase; U/S=ultrasound; ECG=electrocardiogram; CXR=chest X-ray; IVC=inferior vena cava; R/L BBB=right/left bundle branch block; AF=atrial fibrillation; c-th ratio=cardiothoracic ratio; RV, LV=right, left ventricle; EF=ejection fraction; AS=aortic stenosis; MR=mitral regurgitation; NR=not recorded

In 6 of the 8 patients the jaundice was probably due to the passive liver congestion of low-output cardiac failure. Other groups have described a raised alkaline phosphatase in these circumstances^{5–7} but this was seen in only 2 of the 6. The phenomenon has been linked to the severity of tricuspid regurgitation.^{8,9} Suggested mechanisms for the jaundice of low-output heart failure are decreased hepatic blood flow, increased hepatic venous pressure and decreased arterial oxygen saturation. In addition, work in animals raises the possibility of endotoxin mediated damage.¹⁰

In the 2 patients with ischaemic hepatitis the probable cause was myocardial infarction in the setting of severe valvular disease. Such patients tend to have a massive rise in aminotransferases with associated derangement in prothrombin time.¹¹ Ischaemic hepatitis, which results from hepatic circulatory failure, predominantly affects the

perivenular zone of the hepatic acinus.² Hepatic blood flow declines by about 10% for every 10 mmHg drop in arterial pressure.¹² Rapid resolution of the hypotension usually leads to full recovery of the hepatitis.^{12,13} It is noteworthy that healthy individuals with acute hypotension from events such as trauma do not seem to develop ischaemic hepatitis. A retrospective analysis of patients with ischaemic hepatitis indicated that all had underlying heart disease, predominantly right-sided.¹⁴ Thus a baseline of hepatic congestion may be required as a 'primer' before the development of ischaemic hepatitis.

We conclude that the combination of jaundice and breathlessness should prompt a careful cardiological examination, including assessment of the jugular venous pressure, electrocardiogram, chest radiograph and echocardiogram to exclude a cardiac cause. Ischaemic hepatitis is suggested by a high alanine aminotransferase.

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